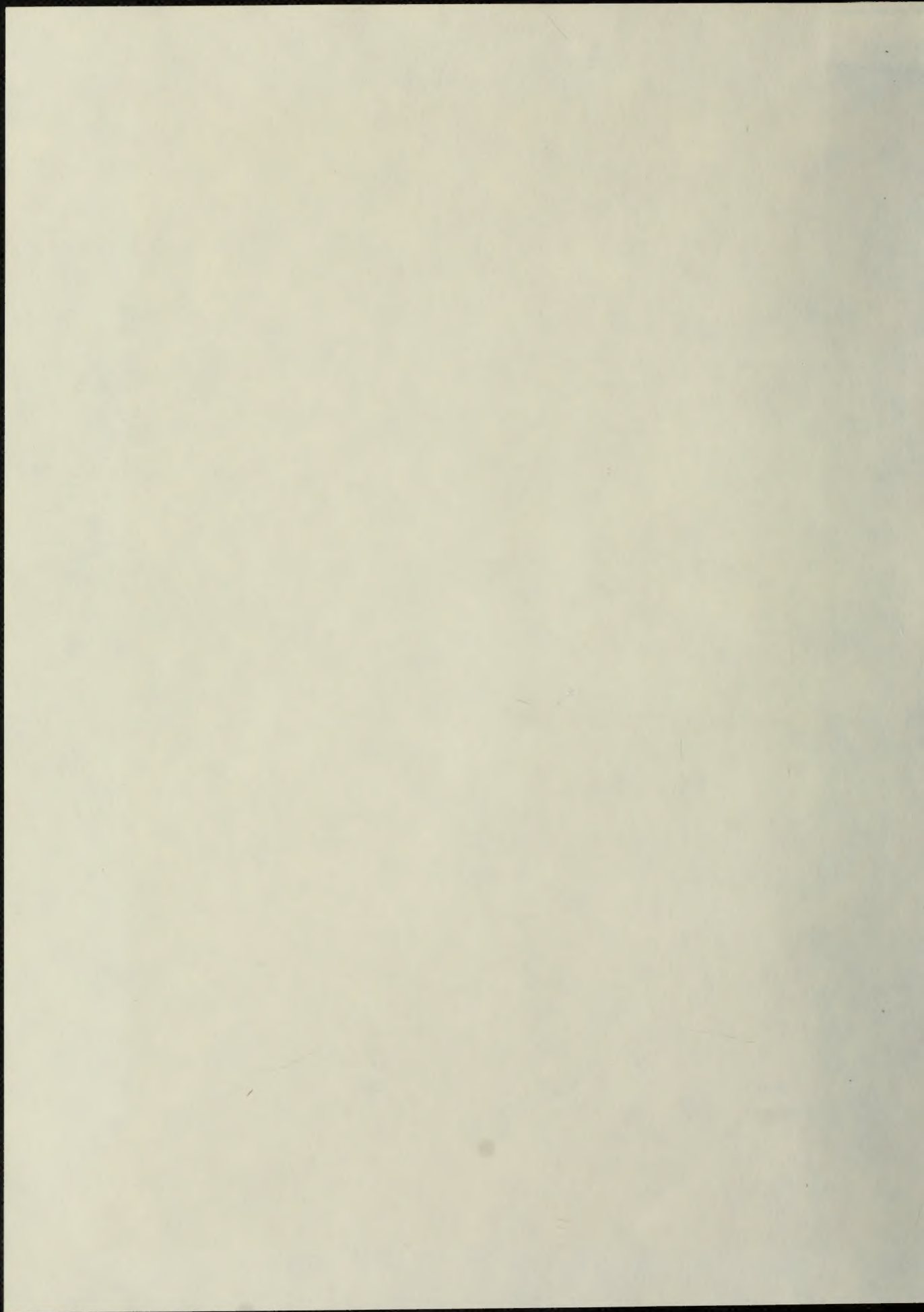


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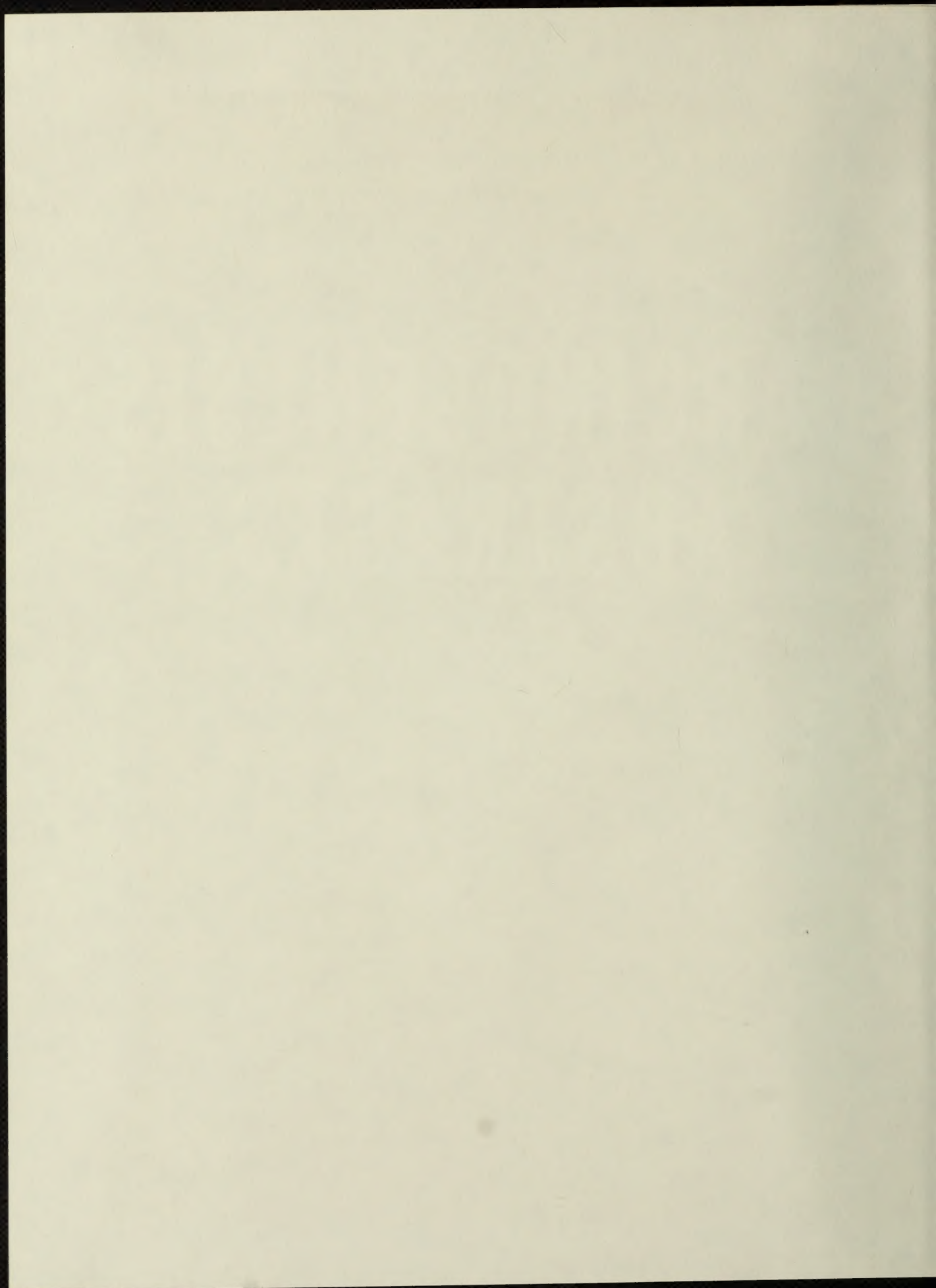
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CARCINOGENESIS ABSTRACTS

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CARCINOGENESIS ABSTRACTS makes available abstracts, annotations or citations of significant carcinogenesis articles collected from the current major biomedical sources of world literature. This service is provided by the National Cancer Institute through a contract with the Franklin Research Center for preparation of the publication, under Contract No. NOI-CP-75885 with the National Cancer Institute, U.S. Department of Health, Education and Welfare. Published and distributed by the Franklin Institute PressSM.

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ABBREVIATIONS

JOURNAL names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations*.

LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED₅₀	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO₂	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD₅₀	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD₅₀	median lethal dose		
M	molar		
μM	micromolar		

REVIEW

- 79-2401 **Somatic Mosaicism in Plants with Special Reference to Somatic Crossing Over.** (Eng) Vig, B. K. (Dept. Biology, Univ. Nevada at Reno, Reno, NV 89557) *Environ Health Perspect* 27: 27-36; 1978.

The use of somatic mosaicism induction in plants as a tool in the study of environmental mutagenesis was evaluated. Higher plant systems that are promising for the study of mutagen-induced mosaicism include *Tradescantia hirsuticaulis*, *Glycine max*, *Antirrhinum majus*, *Pisum sativum*, *Nicotiana tabacum*, *Zea mays*, and *Arabidopsis thaliana*. As an example of the nature of mosaicism, the soybean system is described in detail. The gene combination of $Y_{11}y_{11}$ has been used as a marker system in this species. Various mutagens may result in mosaicism, which may consist of dark green, yellow, or twin (double) spots on the two simple leaves. The twin spots are inferred to originate by somatic crossing over. Some of the single spots may occur as a result of the failure of one component of twin spots; others may be caused by chromosomal segmental losses or numerical inequalities in chromosome distribution during mitosis. Twin spots occur in other species, and they can be induced by caffeine administration. Studies of mosaicism in plants offer inexpensive, rapid, and reliable tests of mutagenicity, at least as preliminary eukaryotic test systems. (47 refs)

- 79-2402 **Literature Survey of Bacterial, Fungal, and *Drosophila* Assay Systems Used in the Evaluation of Selected Chemical Compounds for Mutagenic Activity.** (Eng) Brown, M. M. (Environmental Mutagen Information Center, Information Center Complex Information Div., Oak Ridge Natl. Lab., P.O. Box Y, Building 9221, Oak Ridge, TN 37830); Wassom, J. S.; Malling, H. V.; Shelby, M. D.; Von Halle, E. S. *J Natl Cancer Inst* 62(4): 841-871; 1979.

Literature data on the use of seven nonmammalian assay systems for the detection of mutagenicity or other related genetic effects are reviewed. The activities of 54 selected noncarcinogens, procarcinogens, and ultimate carcinogens as revealed by these test systems were compared. Forty-nine of the compounds tested were active in one or more of the assays, and 42 were positive in at least one system without metabolic activation. In one or more test systems, 17/17 ultimate carcinogens, 27/28 procarcinogens, and 6/9 noncarcinogens were positive. The Ames Salmonella-microsome assay responded with increased mutation frequency to 37/44 carcinogens but to only 2/8 noncarcinogens tested. The *Drosophila* system responded to 19/21 carcinogens and to 3/6 noncarcinogens tested. Pro-

phages were induced when lysogenic bacteria were exposed to 12/21 carcinogens, but not enough tests were done with the noncarcinogens (1/3) for a comparison. The other systems reviewed, such as the killing of repair-deficient bacteria, mutations in *Escherichia coli* and *Neurospora crassa*, and the host-mediated assay, were not challenged by enough of the compounds for valid comparisons. (439 refs)

- 79-2403 **A Strategy Using Short-Term Assays to Identify Carcinogens That Act in Man.** (Eng) Bruce, W. R. (Ontario Cancer Inst., 500 Sherbourne St., Toronto M4X 1K9, Ontario, Canada); Dion, P. D.; Kakizoe, T.; Land, P. C. *Nutr and Cancer* 1(2): 4-7; 1979.

A scheme is presented for the use of short-term tests to identify carcinogens that act in humans, and it is illustrated by applications to colon and bladder cancer. Short-term tests can be used to determine if there is a measurable level of presumed carcinogen in individuals at risk for the disease. If the presumed carcinogen(s) appears to associate with specific cancers, then it is possible that the chemical detected by the assay is responsible for the cancer. The shorter-term tests can also be used to determine the nature and structure of the presumed carcinogen. This procedure involves chemically purifying the agent detected by the assay and identifying its structure. These steps are followed by basic studies of the origin of the presumed carcinogen and confirmation of its carcinogenicity in animal tests. The scheme also involves inhibition of or reduction of exposure to the presumed carcinogen in vivo, intervention in high-risk patients, and intervention in normal populations. Because the short-term assays are cheap, they should be used in the initial investigations to search for possible problems and to define areas of concern. Because they are not always accurate, they must be supported by systematic animal studies. (19 refs)

- 79-2404 **A Review of the Metabolism of Xenobiotics by Microorganisms with Relation to Short-Term Test Systems for Environmental Carcinogens.** (Eng) Callen, D. F. (Flinders Univ. South Australia, Bedford Park, S.A. 5042, Australia) *Mutat Res* 55(3/4): 153-163; 1978.

Aspects of the metabolism of xenobiotics by microorganisms that are relevant to short-term test systems for environmental carcinogens are reviewed. The presence of a cytochrome P-450-dependent monooxygenase system

has been established in a variety of fungi and bacteria. The cytochrome P-450-dependent monooxygenase system of fungi is similar to that of mammals, but some differences are apparent. The bacterial enzyme system consists of a soluble cytochrome P-450 with a requirement for NADH. A microbial test system in which activation is possible within the microbial cells may be a more adequate model of the in vivo situation than the activation systems now in use. A disadvantage of these systems is that a carcinogen may be metabolized in microorganisms by pathways that differ from those of mammals. Mammalian intestinal microbial flora have a role in the etiology of colon and rectal cancer, which should be recognized in microbial test systems for environmental carcinogens. A number of hydroxy-anthraquinones that were nonmutagenic in the Ames *Salmonella* assay were mutagenic when the liver homogenate used in the assay was replaced by a cell-free extract of intestinal microflora. It is suggested that gut microflora should be incorporated in short-term screening tests. (83 refs)

- 79-2405 Data on Environmental Carcinogenic Polycyclic Aromatic Hydrocarbons. (Hun) Shabad, L. M. (Oncology Res. Center, USSR Acad. Medical Sciences, Moscow, USSR) *Magy Onkol* 23(1): 3-11; 1979.

Studies of the general environmental problems of carcinogenic polycyclic aromatic hydrocarbons (PAH's) are reviewed. In view of the good correlation between concentrations of benzo(a)pyrene and other PAH's, the former can be used as an indicator of overall PAH levels. PAH's are generated during combustion processes and also by plants (eg, during germination). They accumulate in soil and bodies of water, and they are partially decomposed by certain microorganisms; but a background level persists. Similar tumor induction rates were observed in mice following topical application of pure benzo(a)pyrene and of soot extracted from automotive exhaust gases. (no refs)

- 79-2406 Clinical Practice and Epidemiology: Two Worlds or One? (Eng) Acheson, E. D. (Univ. Dept. Community Medicine, General Hosp., Southampton SO9 4XY, England) *Br Med J* 1(6165): 723-726; 1979.

The relationship between epidemiology and clinical medicine is discussed, particularly with regard to the study of various suspected carcinogens. (13 refs)

- 79-2407 Environmental Carcinogenesis--Part II. (Eng) Hadden, J. W. (Dept. Medicine, Sloan-Kettering Inst. Cancer Res., New York, NY) *Clin Bull* 8(4): 164-167; 1978.

Several carcinogenesis research projects of the Sloan-Kettering Institute (New York, NY) are reviewed. The induction of rat tumors by methylazoxymethanol (MAM) acetate is being used as a model for human colon cancer. These studies suggest that the effects of the carcinogen are due to a metabolite resulting from a dehydrogenase reaction. In humans and rodents with a genetic predisposition to colon, stomach, and cervical cancer, abnormal proliferative activity can be detected in the cells even before the subjects develop neoplastic lesions. A study is in progress to quantitate the development of abnormally proliferative gastrointestinal cells in several high-risk population groups as an early indication of susceptibility to neoplasia. Cloned lines of epithelial liver cells from rats sacrificed after po administration of diethylnitrosamine contained juxtanuclear filamentous aggregates resembling the Mallory bodies (alcoholic hyaline) frequently seen in cirrhotic livers of chronic alcoholics. These heritably maintained cytoplasmic structures are the result of an impairment of microtubule formation and function, an abnormal accumulation of intermediate filaments, and modulation of cell membrane receptors. Other studies are focusing on the theory that diol epoxides are the ultimate carcinogens of polycyclic hydrocarbons, on the interaction of adriamycin with DNA, on changes produced in transfer RNA's of target organs by carcinogens, and on whether in vitro screening tests for transformation and mutagenesis have predictive value for detection of carcinogenic activity. (no refs)

- 79-2408 Saccharin: From Carcinogen to Promoter. (Eng) Boyland, E. (London Sch. Hygiene and Tropical Medicine, London, England) *Nature* 278(5700): 123-124; 1979.

Evidence that saccharin may be a promoting agent rather than a complete carcinogen is reviewed. In eight studies in which saccharin was fed to rats, the bladder tumor incidence was never more than 44% and was usually lower, even when the diets contained 5% saccharin. When small initiating doses of methyl nitrosourea were instilled into the urinary bladder of Wistar rats, no bladder tumors were detected. Of the rats that were subsequently given saccharin in drinking water, 57% developed tumors. Similar results were obtained when rats were fed 2% N-(4-(5-nitro-2-furyl)-2-thiazolyl)formamide (FANF) followed by 5% saccharin. Cultures of C3H/10T1/2 cells treated with non-transforming initiating doses of methylcholanthrene + saccharin had a statistically significant number of transformations. These experiments indicate that saccharin is a promoter rather than a direct carcinogen. Although saccharin may not be harmless, it is unlikely to be a serious hazard because the dose required to produce the promoting effect is so large (1 kg/kg body wt). (18 refs)

- 79-2409 Azide. (Eng) Kleinhofs, A. (Program in Genetics, Washington State Univ., Pullman,

WA 99164); Owais, W. M.; Nilan, R. A. *Mutat Res* 55(3/4): 165-195; 1978.

The mutagenic and toxicological properties of sodium and potassium azides are reviewed. Most of the physiological and toxicological properties of azide can be traced to its inhibition of enzymes containing a coordinated divalent metal ion. The potent mutagenicity of azide has been recognized only recently; this mutagenicity is highly dependent on a defective excision-repair system and a functional recombination system. Azide mutagenesis is very specific in that it is unable to revert any of the frameshift, *ochre*, or *amber Salmonella typhimurium* mutants so far tested. There is little or no relationship between the mutagenic properties of azide and its properties as a respiration and catalase/peroxidase inhibitor. Sodium azide was not mutagenic in *Drosophila*, but it induced mutations in Chinese hamster cell lines. The mono- and di-azido derivatives of ethidium bromide were highly mutagenic in yeast and in *S. typhimurium*. Azide analogs of proflavine and acriflavine induced high frequencies of frameshift mutations in excision-repair *Salmonella* strains. Sodium azide was not carcinogenic in male or female rats given the max tolerated dose or half that level in the diet 2x/wk. The mutagenic action of sodium azide in barley is of concern because of the danger of introducing stable mutagens into the food chain. (226 refs)

79-2410 Chloroprene (2-Chloro-1,3-butadiene)--What Is the Evidence for Its Carcinogenicity? (Eng)

Haley, T. J. (Dept. Health, Education, and Welfare, Food and Drug Admin., Natl. Center Toxicological Res., Jefferson, AR 72079) *Clin Toxicol* 13(2): 153-170; 1978.

The biochemistry, metabolism, and toxicology of chloroprene (CP: 2-chloro-1,3-butadiene) were reevaluated to establish whether it is a potential carcinogen. The LD_{50} 's of CP in mice and rats are 260 and 251 mg/kg, respectively. The pathologic changes in these animals included hemorrhages and dystrophic changes in the CNS, lungs, kidneys, and spleen. Chronic inhalation of CP by dogs resulted in changes in higher nervous activity, the nerve cells of the cerebral cortex, and the brain vasculature. Blood histamine increased and histaminase activity decreased in 103 Soviet workers exposed to CP, and the changes were related to duration of exposure. During chronic CP intoxication, there was dysfunction of both the CNS and peripheral nervous system, particularly the cholinergic branch. Cytogenetic analysis of somatic cells from exposed workers aged 23-59 yr revealed both chromosome and chromatid aberrations. Immunization of 208 CP workers with typhoid vaccine produced low immunologic reactivity and no increase in phagocytic activity. In mice, the growth of transplanted Crocker's sarcoma was accelerated by sc CP injection. During 1956-1970, 137 cases of skin cancer were diagnosed in 24,989 Soviet patients; CP workers had the highest skin cancer incidence (21/684),

followed by persons working with CP derivatives (38/2,250). In the same period, 87 lung cancers were found in 19,979 workers; 18 of the patients had direct and prolonged exposure to CP and 16 had a history of exposure to CP latexes. Worldwide epidemiology studies should be undertaken to validate the Soviet reports of CP carcinogenicity in humans. Additional metabolic studies are also necessary to define the neurohumoral mechanism of CP action. (149 refs)

79-2411 Carcinogenicity of Phenacetin (2 Letters to Editor). (Eng) Tomatis, L. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, World Health Organization, 69372, Lyon Cedex 2, France); Johansson, S.; Angervall, L. *Science* 204(4389): 129-130; 1979.

Some recently published results of clinical, epidemiologic, and experimental studies of the possible carcinogenicity of phenacetin (PA) and PA-containing compounds are cited in two letters. These studies were not mentioned in a previous letter in which a study demonstrating that PA was not carcinogenic in rats was quoted. However, other studies have shown that PA and N-hydroxyphenacetin cause urinary tract and nasal cavity tumors in rats. In addition, epidemiologic studies have shown an association between PA abuse and the development of renal pelvic, bladder, and ureteral tumors. (17 refs)

79-2412 The Carcinogenicity of Kepone. (Eng) Reuber, M. D. (NCI Frederick Cancer Res. Center, Frederick, MD 21701) *J Environ Pathol Toxicol* 2(3): 671-686; 1979.

All known animal studies of the carcinogenicity of the chlorinated pesticide decachlorooctahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalen-2-one (Kepone) are reviewed. Kepone induced malignant liver tumors in rats and mice fed the chemical in the diet. Malignant tumors were also found in organs other than the liver in rats, and these were induced even by the lowest dose administered. Female rats were more susceptible to the development of malignant tumors than male rats. Kepone also induced toxic changes, particularly in male rats, and they included renal interstitial fibrosis, testicular atrophy, and polyarteritis of the mesenteric, pancreatic, and other arteries. (7 refs)

79-2413 Environmental Pollutants and the Epidemiology of Cancer. (Eng) Health, C. W. (Chronic Diseases Div., Bureau Epidemiology, Center Disease Control, Atlanta, GA 30333) *Environ Health Perspect* 27: 7-10; 1978.

Approaches used in epidemiologic studies of environmental carcinogenesis are reviewed briefly. Some approaches, as with kepone, a chlorinated hydrocarbon insecticide, and with polybrominated biphenyls, start with exposed populations and measure disease frequency, the so-called prospective or cohort approach. Others, illustrated by vinyl chloride monomer studies, start with cases of cancer (hepatic angiosarcoma) and measure the extent of toxic exposure; ie, the retrospective or case history (case-control) approach. Other studies describe patterns of cancer occurrence and then seek correlations with potentially related population characteristics. All of these approaches are part of epidemiologic methodology and all are predicated on the existence of prior human exposure or prior disease occurrence. In this regard, major limiting factors involve concepts of latency and exposure dose. Although in vitro and animal test systems will never fully supplant human studies, they represent the only means for detecting potential carcinogenicity before human exposure has become widespread or long-established. (17 refs)

- 79-2414 **Hazards Associated with Exposure to Vinyl Chloride and Polyvinyl Chloride Materials.** (Rus) Diubankova, E. N. (F. F. Erisman Res. Inst. Hygiene, Moscow, USSR); Bykhovskii, A. V. *Gig Sanit* (1): 69-74; 1979.

Current data on the hazards of vinyl chloride (VC), in general, and of the use of polyvinyl chloride packages, in particular, are reviewed. Seventeen cases of liver angiosarcoma have been reported in workers in the VC industry (compared with the total 45 cases reported in the world literature). In addition to liver angiosarcomas, the workers had an increased incidence of cancer of the respiratory, CNS, lymph, and hematopoietic systems. The long-term storage of alcohol in PVC bottles showed distinct organoleptic changes in the alcohol that prompted a ban on the use of PVC containers for food products containing alcohol. (26 refs)

- 79-2415 **Vinyl Halides: Carcinogenicity. Vinyl Bromide, Vinyl Chloride, and Vinylidene Chloride.** (Eng) Bahlman, L. J. (Natl. Inst. Occupational Safety and Health, 5600 Fishers Lane, Rockville, MD 20857); Alexander, V.; Infante, P. F.; Wagoner, J. K.; Lane, J. M.; Bingham, E. *Am Ind Hyg Assoc J* 40(4): A-30-A-40; 1979.

Laboratory studies demonstrating the carcinogenicity and mutagenicity of vinyl chloride (VC), vinylidene chloride (VDC), and vinyl bromide (VB) are reviewed, together with studies demonstrating the carcinogenicity and mutagenicity of VC in humans. Liver angiosarcomas have been induced in rats or mice by vinyl halide concentrations of 25-55 ppm. It is recommended that VB and VDC be considered in the

workplace as potential carcinogens to humans and controlled with the same degree of prudence as VC. (35 refs)

- 79-2416 **Some Comments on the History and Experimental Explorations of Metal Carcinogens and Cancers.** (Eng) Hueper, W. C. (Journal of the Natl. Cancer Inst., Room 2A01, Blair Building, Bethesda, MD 20014) *J Natl Cancer Inst* 62(4): 723-725; 1979.

The historical background and studies of metal carcinogens and metal-induced cancers are reviewed. Around 1935, cancers of the lung were discovered in chromate workers and cancers of the lung and nasal sinuses were found in nickel refiners. Since then, beryllium, cadmium, cobalt, lead, selenium, mercury, gold, and zinc have been identified as carcinogens, and iron has been suggested as a possible carcinogen. Because of their extensive use in industry and consumer goods, the importance of chromates and Ni as environmental carcinogens has increased. Animal experiments have firmly established the carcinogenicity of chromium when it is introduced in a biologically available form that permits interaction between the carcinogen and tissue. Both epidemiologic evidence and animal experiments have also demonstrated the carcinogenicity of Ni compounds and metallic Ni. Large granulomas, but not cancers, resulted when powdered Fe was injected into the pleura or lungs of rats, and osteogenic sarcomas resulted when uranium was injected into the marrow cavities of rat femurs. Whether the latter cancers were caused by radiation or the metal itself is not certain. (no refs)

- 79-2417 **Pesticide Use in Agriculture.** (Eng) Ridgway, R. L. (Science and Education Admin., U.S. Dept. Agriculture, Beltsville, MD 20705); Tinney, J. C.; MacGregor, J. T.; Starler, N. J. *Environ Health Perspect* 27: 103-112; 1978.

Quantitation of mutagenic and carcinogenic risks from pesticide use in agriculture is important because of recent reports that representatives of the major classes of pesticides are suspect mutagens and/or carcinogens. These classes include triazines, organochlorines, carbamates, dithiocarbamates, organophosphates, phthalimides, amides, phenyl ureas, phenoxyacetic acids, and benzimidazoles, >80% of all synthetic organic pesticides now used in the US. Regulatory decisions will be a major determinant of future pesticide technology. Evaluation of laboratory findings in terms of risk is often impossible if the mechanisms by which genetic damage arises are unknown. For example, interpretation of the finding that certain herbicides cause structural abnormalities in plant chromosomes would be quite different if the effect resulted from direct alkylation of DNA rather than, as has been suggested, as a secondary effect of severe physiological

disturbances in the plant. Higher plants have potential use as monitors of mutagens in the environment. For example, monitoring the frequency of atrazine-induced mutations in field plants revealed a mutagenic effect due to environmental conversion products that might not have been appreciated from conventional laboratory tests of the pesticide per se or from chemical determinations of atrazine residues in the field. Hopefully, the integration of monitoring data with laboratory and epidemiological data will result in a quantitative measure of health risk. The use of benefit-cost (risk) analyses in regulatory decision-making processes is also surveyed. (46 refs)

- 79-2418 **Activation of Chemicals into Mutagens by Green Plants: A Preliminary Discussion.** (Eng) Plewa, M. J. (Inst. Environmental Studies, Univ. Illinois, Urbana, IL 61801) *Environ Health Perspect* 27: 45-50; 1978.

Recent studies that demonstrate the activation of chemicals, particularly pesticides, into mutagens by green plants are reviewed. Three techniques are involved in research on plant activation. In the in vitro method, growing plants are exposed to a suspect chemical, the plant tissues are homogenized, and microbial indicator organisms are exposed to an extract made from the treated plants. The in vitro method involves incubating a test chemical with an untreated plant homogenate supplemented with cofactors and assaying the homogenate by microbial indicator organisms. The genetic endpoint of the in situ method is reversion at the *waxy* (*wx*) locus of *Zea mays* maize microgametophytes (pollen grains). The history and results of several studies with this method are described. Although it does not have the controlled laboratory conditions of the in vivo and in vitro methods, it does detect the presence of a mutagen within the ecological dynamics of the agricultural field. Results of screening some pesticides and combinations of pesticides with the maize *wx* locus assay are tabulated. The s-triazine herbicides have been chosen for studies in plant activation because of their wide-spread use in agriculture and the contradictory results concerning their mutagenicity. The results of various assays indicate that the s-triazine herbicides atrazine, simazine, and cyanazine induce both mitotic and meiotic chromosome aberrations and that they are activated biologically into agents that induce point mutations. (54 refs)

- 79-2419 **Role of pH in Chemical Mutagenesis.** (Eng) Singh, C. (Regional Res. Lab., Jammu Tawi, India); Kaul, B. L. *J Sci Ind Res* 37(8): 426-432; 1978.

Studies of the effect of pH on chemical mutagenicity are reviewed. Although there are contradictory reports, in general, ethylmethane sulfonate- and diethyl sulfate-

induced mutagenic activities increase at low pH and are almost similar. They may differ quantitatively, however, depending on the concentration of the mutagen used and its hydrolysis rate. Isopropylmethane sulfonate, β -propiolactone, and 1,3-propane sultone are largely unaffected by pH changes. Nitroso compounds also have increased activity at acidic pH, and they show a correlation between mutagenicity and decomposition rate. Mutagenicity is increased if the nitroso compounds, eg, N-methyl-N-nitrosourea, N-ethyl-N-nitrosourea, and N,N-dimethyl-N-nitrosourea, are administered at a pH range in which the half-life of the mutagen exceeds the duration of treatment. Aliphatic nitrogen mustards [N-ethylbis-(β -chloroethyl)amine hydrochloride and N-methylbis-(β -chloroethyl)amine hydrochloride] and ethylenimine have increased mutagenic activity under alkaline conditions. (122 refs)

- 79-2420 **An Evaluation of Systemic Tumorigenesis Following Topical Application of Chemicals (Meeting Abstract).** (Eng) Tobin, P. (Div. Toxicology, Food and Drug Admin., Washington, DC); Kornhauser, A.; Scheuplein, R. *Clin Res* 27(2): 537A; 1979 (no refs)

- 79-2421 **Glycolipids and Malignancy: A Review of Some Current Ideas (Meeting Abstract).** (Eng) Critchley, D. R. (Dept. Biochemistry, Univ. Leicester, Leicester, England) *Br J Cancer* 39(4): 460-462; 1979 (29 refs)

- 79-2422 **Prostaglandins and Drug Induced Agranulocytosis, Aplastic Anaemia and Leukemia.** (Eng) Das, U. N. (Dept. Genetics, Osmania Univ., Hyderabad-500007, India) *Prostaglandins Med* 2(3): 235-238; 1979.

The relationship between drug-induced agranulocytosis (AGC), aplastic anemia (AA), and leukemia and the effects of the drugs on the prostaglandin system are considered. Drugs such as aspirin, indomethacin, phenylbutazone, chloroquine, and quinine have been reported to induce, or at least are associated with, AA, AGC, and, occasionally, leukemia. Cyclic AMP and cyclic guanosine monophosphate (GMP) may regulate the proliferation and differentiation of granulocyte-macrophage progenitor cells. Since prostaglandins influence intracellular cyclic AMP and cyclic GMP levels, drugs that affect the synthesis or action of prostaglandins could indirectly influence the levels of these two nucleotides and thus cause AGC and AA. Some cases of leukemia have been reported in chloroquine-induced AA and AGC. Leukemic cells have a low phagocytic index, low chemotacticity, and low levels of cyclic AMP, parameters influenced by prostaglandins. A

chloroquine-induced decrease in prostaglandin activity may be responsible for the malignant transformation of WBC. This hypothesis is supported by the fact that prostaglandin E can block tumor development in animals and in vitro. In addition, treatment of malaria by chloroquine may be a factor in the pathogenesis of Burkitt's lymphoma. It is proposed, therefore, that drugs that interfere with the prostaglandin system can induce AGC, AA, and leukemia. (22 refs)

- 79-2423 Toxicity of Chloramphenicol and Thiamphenicol (CAP and TAP). (Ger) Keiser, G. (Medizinische Abteilung, Burgerspital, 6300 Zug, Switzerland) *Haematol Bluttransfus* 21: 179-195; 1978.

The bacteriostatic action of chloramphenicol (CAP) and thiamphenicol (TAP) and their early and late toxic effects on mammalian cells, including the occurrence of aplastic anemia and leukemia in humans, are reviewed. CAP has an -NO₂ group in the para- position, TAP a -CH₃SO₂ group. Under the same conditions, the covalent binding of CAP to liver and bone marrow mitochondria is 12 times greater than that of TAP. At therapeutic doses, CAP inhibits DNA synthesis in animal erythroblast cultures, but TAP does not. Thus, CAP has two effects that are characteristic of toxic or mutagenic substances. CAP, but not TAP, leads to irreversible bone marrow damage and thus to aplastic anemia. CAP is the drug most commonly implicated in aplastic anemia cases associated with chemicals (312 cases vs 35 for phenylbutazone, the second most commonly implicated drug). Thirty cases of leukemia after CAP use are described in the literature; 15 are described in detail, and an effect of CAP seems probable in these cases. The therapeutic use of CAP is not justified in view of these findings. In contrast, TAP appears to be a useful antibiotic and cytostatic agent, particularly for polycythemia. (39 refs)

- 79-2424 Vitamin A and Cancer of Epithelial Origin. (Eng) Basu, T. K. (Div. Nutrition, Dept. Biochemistry, Univ. Surrey, Guildford, Surrey, England) *J Hum Nutr* 33(1): 24-31; 1979.

The role of vitamin A in protecting against and treating cancers of epithelial origin is reviewed. Vitamin A has been shown to protect against squamous metaplasia in experimental animals exposed to carcinogenic polycyclic aromatic hydrocarbons (PAH). In humans, low plasma vitamin A and β -carotene levels have been associated with squamous cell carcinomas of the bronchi, oral cavity, and oropharynx. A wealth of experimental evidence from animal studies suggests that supplementary feeding of vitamin A and retinoic acid can induce cellular repair of pathologic changes by PAH. The incidence of carcinoma in hamsters treated with benzo(a)pyrene was drastically in-

hibited when the animals were placed on lifetime po treatment with 13-cis-retinoic acid following completion of carcinogen dosing. No deleterious side effects were noted with retinoid doses as high as 9 mg/wk, although even one-half as much retinyl acetate or all-trans-retinoic acid would have been severely toxic. 13-cis-Retinoic acid has also been reported to have a marked inhibitory effect on bladder cancers induced in rats by N-methyl-N-nitrosourea. Thus, vitamin A appears to have prophylactic as well as therapeutic activity against certain cancers, and the use of vitamin A analogs may reduce toxicity. (39 refs)

- 79-2425 The Epidemiology of Cancer of the Oesophagus. (Eng) Cook-Mozaffari, P. (Medical Res. Council External Staff, Dept. Regius Professor Medicine, Univ. Oxford, Oxford OX1 3QG, England) *Nutr and Cancer* 1(2): 51-60; 1979.

The epidemiology of esophageal cancer (EC) is reviewed. The range of incidence between different regions varies 500-fold. Sharp gradients of incidence occur between regions only a few hundred miles apart. High-incidence areas (HIA's) have been found in Iran and in southern and eastern Africa. Sometimes the rates for women show the same high frequency as those for men, but often they occur at a much lower level. In the HIA's of Normandy and Brittany, the male:female ratio is >25:1. Clear but small-scale increases in incidence have been recorded for both sexes among US Blacks and for men in northern France. During the last 30-40 yr, EC has become one of the commonest types of cancer diagnosed in southern Africa; previously, it was nonexistent. In the HIA of Iran, however, the disease was described as long ago as the 12th century. In several HIA's vitamin A and riboflavin deficiencies appear to be associated with EC. In Kenya and other parts of Africa, there is some evidence to implicate home-brewed beer made from maize. A case-control study in Normandy and Brittany indicated that most of the excess risk for EC could be ascribed to a multiplicative effect of drinking and smoking. However, the sum of these various factors is not sufficient to account for the exceptionally high incidences and extraordinary pattern of variation of EC. (59 refs)

- 79-2426 Effects of Hair Sprays Need Investigation (Letter to Editor). (Eng) McInnes, B. (140 Dubois Ave., Valley Stream, NY 11581) *N Engl J Med* 300(15): 863; 1979.

The importance of investigating the possible relationship between the use of hair sprays and lung cancer is pointed out. One study, which did not control for cigarette smoking, demonstrated that the risk of lung cancer among female beauticians was 6.92 times greater than that among nonbeauticians; however, the study focused on contact with hair dyes, and sprays were not mentioned. (1 ref)

79-2427 Cigarette Smoke-induced Laryngeal Cancer: A Model for Bronchogenic Carcinoma in Humans (Letter to Editor). (Eng) Homburger, F. (Boston Univ. Sch. Medicine, Boston, MA 02118); Bernfeld, P. *N Engl J Med* 300(15): 862; 1979.

In a critique of the Surgeon General's report on smoking and health, it is pointed out that carcinogenesis using tar fractions or smoke inhalation has not been successful in the respiratory tract of small animals, except for the induction of laryngeal cancer in certain susceptible inbred Syrian hamster strains. In addition, in these hamsters, it is presently possible to determine at least whether low-tar cigarettes are more or less carcinogenic than high-tar cigarettes. (9 refs)

79-2428 Oral Contraceptives and Liver Tumours (Letter to Editor). (Eng) Gray, R. H. (World Health Organization, 1211 Geneva 27, Switzerland) *Med J Aust* 2(11): 531; 1978.

Conclusions of a World Health Organization study of the risk of liver neoplasia associated with the use of oral contraceptives (OC), especially those containing high doses of steroids, are summarized. There is a marked increase in the relative risk of hepatocellular adenoma (HCA) among women who have used OC for >3 yr. The magnitude of risk increases with dose of steroid, duration of use, and age of user. However, preliminary calculations suggest that the attributable risk of HCA among women <30 yr old is no more than 3/100,000 users per year, regardless of length of use. This risk is higher for women >30 yr old. (4 refs)

79-2429 Features of Liver Damage due to 17 α -Alkyl-substituted Anabolic Steroids. (Ita) Lovisetto, P. (Istituto di Medicina Interna, Università di Torino, Turin, Italy); Mairano, D.; Actis, G. C.; Marchi, L. *Minerva Med* 70(11): 769-790; 1979.

Studies of liver damage (intrahepatic cholestasis, hepatic peliosis, and tumors) caused by 17 α -alkyl-substituted steroids are reviewed. Liver tumors induced by these steroids are very similar to those induced by estrogens and progestins. The 17 α -alkylation is believed to lead to proliferation of the hepatic parenchyma. The anatomical picture resembles that of benign adenoma, hepatocarcinoma, and widespread multinodular hyperplasia. There is a marked discrepancy between histological type and clinical course: some tumors rated as carcinomas regress completely on suspension of the steroid treatment. The term tumor is thus inappropriate: these lesions can best be described as hormone-dependent, histopathologically unusual proliferative alterations. Liver tumors reported in the literature occurred following treatment with testosterone (50 mg/day for 4 mo), oxymetholone (30-300 mg/day for

3-50 mo), methyltestosterone (20-50 mg/day for 40 mo-30 yr), and methandrostenolone (15 mg/day for 89 mo). (57 refs)

79-2430 Essential Hormones as Carcinogenic Hazards. (Eng) Hickey, R. J. (Wharton Sch., Univ. Pennsylvania, Philadelphia, PA 19104); Clelland, R. C.; Bowers, E. J. *J Occup Med* 21(4): 265-268; 1979.

The inclusion of some endocrine biochemicals on a list [published by the Occupational Safety and Health Administration (OSHA)] of environmental substances that pose a potential occupational carcinogenic risk is critically evaluated. At appropriate levels, these substances are essential for proper physiological functioning both in humans and other mammals. These substances include estradiol, estrone, estriol, progesterone, and testosterone. Review of some of the evidence implicating steroidal hormones as carcinogens indicates that estradiol and progesterone may promote the growth of mammary tumors previously initiated by true carcinogens, but that they themselves are not carcinogens. Estrogens and androgens are used in the treatment of some mammary carcinomas. Other substances on the OSHA list include tannic acid and tannins (which are present in tea), maltose, and lactose, a normal ingredient at appreciable concentration in human and other mammalian milks. It is concluded that a major reappraisal of the OSHA list is essential. (22 refs)

79-2431 Hormones and Mammary Carcinogenesis. (Eng) Segaloff, A. (Section Endocrine Res., Richard W. Freeman Res. Inst., Alton Ochsner Medical Foundation, 1516 Jefferson Highway, New Orleans, LA 70121) *Breast Cancer 2. Additives in Research and Treatment.* McGuire, W. L., ed. (New York: Plenum Medical Book Company): 407 pp.; 1-22; 1978.

Studies of hormones and their association with mammary cancer are reviewed, with emphasis on studies in the rat. Mammary carcinogenesis can be induced in intact A x C female rats by the continuous administration of estrogen. This strain is probably the one most consistently susceptible to estrogen induction of mammary gland cancers. Oophorectomized rats are not as sensitive to the effects of estrogen as intact animals, even when progesterone is replaced by pellets or injections. Studies of the various other substances from the ovary that might explain these results led to the hypothesis that relaxin is the ovarian nonsteroidal substance required for the greatest carcinogenesis. Estrogen seems to provide the substrate for carcinogenesis in animals that already have a genetic or other means of initiation. Thyroid substance does not seem to be related to breast cancer, except as its level in the host has an effect on the biotransformation of estrogens. Continuous estrogen action on the mammary gland seems to be

required for a prolonged period of time and in an appropriate amount for each animal species for mammary carcinogenesis. Insulin is probably required for mammary carcinogenesis, at least for promotion to identifiable clinical size. Studies of the effects of progesterone and pituitary hormones have produced conflicting results. In rats, pituitary hormones are apparently able to synergize with a dose of radiation or chemical carcinogen that is insufficient to cause tumors by itself, such that a substantial number of animals will develop mammary tumors. Nutrition has an effect on mammary carcinogenesis because of its effect on the endocrine system. (58 refs)

- 79-2432 Potential Carcinogenic Effect of Sex Hormones During the Perinatal Period of Ontogenesis. (Rus) Ird, E. A. (Moscow, USSR); Smirnov, I. O. *Akush Ginekol (Mosk)* (12): 4-7; 1978.

Literature on the potential carcinogenic effect of transplacental exposure to various sex hormones is reviewed. Data on an increased risk of vaginal cancer in persons who had been exposed to synthetic estrogens in utero prompted the ban on the use of diethylstilbestrol during pregnancy. Experimental administration of estrogens to pregnant rats (0.04-1 mg, 2-4 doses) resulted in masculinization of the female progeny. Numerous cases of female hermaphroditism were reported in the offspring of women who, during 1950-1960, received synthetic progestins. Prolonged (up to 10 yr) use of androgenic progestins as oral contraceptives increases the incidence of benign and malignant tumors of the liver. (50 refs)

- 79-2433 Estrogens and Endometrial Carcinoma. (Ger) Zander, J. (I. Frauenklinik der Universitat, Maistrasse 11, D-8200 Munich 2, W. Germany) *Munch Med Wochenschr* 121(13): 443-444; 1979.

Two epidemiological studies from the US concerning the possible relationship between the use of estrogens in menopause and endometrial carcinoma are reviewed. A case-control study of 451 endometrial carcinoma patients and 888 controls demonstrated a sixfold risk of endometrial carcinoma among women using estrogens compared with controls. The risk was increased for both conjugated estrogens and diethylstilbestrol, and there was no distinct difference between cyclic and continuous treatment in terms of risk. The relative risk was 2.2 in women who had been treated with estrogens for <1 yr and 15 in women who had been treated for >5 yr. Another study demonstrated a risk factor of >10 for women who had been treated with estrogens for >5 yr and a recent decline in the incidence of endometrial carcinoma, possibly as a consequence of the less liberal use of estrogens. (2 refs)

- 79-2434 Carcinogenic Risks from Food—Real or Imaginary? Methodology in Assessing Carcinogenic Risk. (Eng) Grasso, P. (Occupational Health Unit, BP Res. Centre, Sunbury on Thames, Middx TW16 7LN, England) *Chem Ind* (February 3, 1979): 73-76; 1979.

The methodology involved in the administration of carcinogens to laboratory animals to assess carcinogenic risk is reviewed. The administration of carcinogens by the po route is emphasized because it is the most frequently used route in carcinogenicity testing and because it is thought to be the route most relevant to the assessment of carcinogenic risk from food. Rats and mice are the most commonly used test animals, followed by hamsters, dogs, and monkeys. Longevity and cost are major factors against use of the latter two species. With regard to the smaller rodents, choice of a hardy and preferably outbred strain with a known and stable background incidence of tumors is recommended. The use of two or three dose levels is recommended to reduce the effects of random variation, to demonstrate dose-response relationships, and to study tumor histogenesis. The use of max tolerated doses may invalidate the experimental results, in large part as a consequence of nonspecific injury to or irritation of the target tissue. Experiments should be terminated when the total incidence in the controls is approx 20%-25%. (10 refs)

- 79-2435 Naturally Occurring Carcinogens in Food. (Eng) Austwick, P. (Dept. Clinical Immunology, Cardiothoracic Inst., Univ. London, Fulham Road, London SW3, England); Mattocks, R. *Chem Ind* (February 3, 1979): 76-83; 1979.

Naturally occurring carcinogens in green plants and the production of carcinogens by fungi and actinomycetes are discussed. Plant carcinogens and other possible carcinogens that may reach human food include pyrrolizidine alkaloids, safrole, cycasin, bracken, nitrosamines, tannins, and sanguinarine. Among microorganisms, only the fungi and filamentous actinomycetes have been shown to produce carcinogenic toxins. The evidence for associations between actinomycetes and environmental carcinogens is small, but the potential role of these organisms in this problem requires more attention. In contrast to the actinomycete metabolites, most carcinogens from fungi have been detected in foodstuffs. These carcinogens include aflatoxins, sterigmatocystin, ochratoxin A, patulin, penicillic acid, luteoskyrin, cyclochlorotine, and 12',13'-epoxytrichothecenes. Only certain fungi produce the characteristic moldy flavor. Therefore, detection of fungal growth in food is a task for direct microscopy, mycology, and biochemistry, and the estimation of carcinogen content is a task for chromatography and bioassay. Where and when carcinogens are produced by fungi, and the amounts of carcinogens produced, are variable. It is likely that epidemiologic investigations will

show correlations between particular forms of cancer and mycotoxin occurrence. (63 refs)

- 79-2436 **Naturally Occurring Toxicants and Food Additives: Our Perception and Management of Risks.** (Eng) Hall, R. L. (Science and Technology, McCormick and Co., 11350 McCormick Road, Hunt Valley, MD 21031) *Nutr and Cancer* 1(2): 27-36; 1979.

The risks of food contaminants and additives are placed in perspective in a discussion of the various sources of food hazards, problems in interpreting dose-response data, and the wide variety of naturally occurring food toxins. The principal food hazards are those from microbiological and nutritional causes. Far more rare are hazards from environmental contaminants and natural food toxicants, followed by the extremely remote risks from pesticide residues and food additives. Probably the most debated issue in safety evaluation is whether, for any specific carcinogen or class of carcinogens, the dose-response curve is linear or curved. In the first case, every dose would produce some toxic response, and in the second case, an unfavorable response would not be produced until a threshold dose was reached. Steps that can be taken to reduce the hazards of naturally occurring food toxins include: exposure reduction, particularly by dietary choice; sanitation; and choice or limitation of geographic or taxonomic source. The chemical properties and means of controlling several natural food toxins are described. These include solanine, an intrinsic neurotoxin found in potatoes; islanditoxin, a hepatotoxic and carcinogenic metabolite of rice molds; cyanogenetic glycosides, which produce cyanide upon bruising in bamboo shoots, cherry seeds, cassava, and lima beans; carototoxin, a potent neurotoxin found in carrots and celery; aethusin, neurotoxin occurring in fool's parsley; coniine, a neurotoxin found in hemlock berries; saxitoxin, produced by dinoflagellates that may be consumed by edible shellfish; and the honey contaminants, tutin and hyenanchin. It is suggested that virtually every food contains natural toxicants and that if the present safety standards for food additives were applied consistently to all foods, we would have nothing left to eat. (no refs)

- 79-2437 **Public Health Problems Posed by the Use of Pesticides in Agriculture.** (Fre) Zamfir, G. (Institut d'Hygiene et de Sante Publique, Jassy, Rumania) *Sante Publique (Bucur)* 21(1/2): 63-70; 1978.

The long-term mutagenic, teratogenic, and carcinogenic effects of organochloride pesticides are discussed. This type of pesticide accumulates in the food chain (phyto- and zooplankton, fish, and birds), leading to ingestion of large amounts by man. (48 refs)

- 79-2438 **Nutritional Factors That May Be Involved in Cancer of the Bladder.** (Eng) Friedell, G. H. (St. Vincent Hosp., 25 Winthrop St., Worcester, MA 01604); Greenfield, R. E.; Cohen, S.M. *Nutr Cancer* 1(2): 82-88; 1979.

Studies of the relationship between nutrition and bladder cancer (BIC) are reviewed. Elevated amounts of kynurenine, hydroxykynurenine, acetylkynurenine, and kynurenic acid were excreted by 50% of a group of BIC patients who had no history of occupational exposure to aromatic amines. However, BIC patients with known exposure to industrial amines did not exhibit abnormal tryptophan (TP) metabolite excretion patterns. In another study, 17% of BIC patients from an urban area had abnormal TP metabolite excretion patterns, compared with 47% of a group of patients from an agricultural area. These results suggest that TP metabolites may have a role in the etiology of so-called spontaneous BIC, but probably have little to do with the development of industrial or environmental BIC. In Fischer rats fed the carcinogens N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) or N-methyl-N-nitrosourea (MNU), both saccharin and sodium cyclamate acted as BIC promoters. In similar studies, TP also acted as a promoting agent. Pyridoxine (vitamin B6: 25 mg/day) restores an abnormal pattern of excretion of urinary TP metabolites to a normal pattern and also apparently diminishes the frequency of recurring BIC. Vitamin A and vitamin C also appear to have some beneficial effects in inhibiting the induction of BIC in experimental animals. (29 refs)

- 79-2439 **Diet and Old Age.** (Eng) Norden, A. (Dept. Medicine, Univ. Hosp., Lund, Sweden) *Scand J Gastroenterol Suppl* 14(52): 22-27; 1979.

Experimental, clinical, and epidemiological studies of the relationship between diet and old age are reviewed. Studies with rats indicate that conditions in early life govern the life-span and affect the development of age-related diseases, including cancer. Longevity correlates with the rate of wt gain during the early phase of life. Energy restriction during growth delays the development of a full immunological response in mice and prolongs the maintenance of immunological competence. Chronic energy restriction inhibits the formation of many types of tumors. Hepatic tumors, however, are stimulated by energy restriction. Epidemiological studies indicate that obesity is related to an increased tumor risk, particularly cancers of the intestinal tract, liver, gallbladder, breast, uterus, and genitourinary tract. A correlation has been reported in humans between fat consumption and breast cancer. It has been hypothesized that industrialization has affected cancer incidence by the mass refining of carbohydrates rather than by the addition of specific chemicals. The relation between trace elements and cancer has also been investigated. Iodine deficiency was related to

an increased incidence of thyroid cancer and magnesium deficiency was associated with thymoma, but copper appears to inhibit tumor induction. Conflicting findings have been reported for some of the other trace elements in relation to cancer. (40 refs)

- 79-2440 Dietary Fibre and Experimental Colon Cancer** (Letter to Editor). (Eng) Cruse, P. (Surgical Unit, Rayne Inst., Univ. Coll. Hosp. Medical Sch., London WC1E 6JJ, England); Lewin, M.; Clark, C. *Lancet* 1(8112): 376; 1979.

In response to criticisms of previous study of the possible modifying role of dietary fiber in dimethylhydrazine (DMH) carcinogenesis, it is stated that the dose of DMH was not excessive. In a parallel experiment in which rats received an identical DMH dose, 30% of those fed a fiber and cholesterol-free diet failed to develop colon carcinomas, and there was a highly significant prolongation of time to tumor presentation and of survival. A cholesterol-free diet was concluded to be a more powerful dietary modifier of DMN carcinogenesis than added dietary fiber (bran). It is agreed that human colon cancer is likely to have a multifactorial etiology. (8 refs)

- 79-2441 A Prudent Diet for the Nation.** (Eng) Mann, J. I. (Dept. Social and Community Medicine, Univ. Oxford and Radcliffe Infirmary, 8 Keble Road, Oxford OX1 3QN, England) *J Hum Nutr* 33(1): 57-63; 1979.

Nutritional factors in the etiology of coronary heart disease, maturity-onset diabetes, diverticular disease, and dental caries are discussed. The possibility that nutrition plays a role in the etiology of malignant large bowel tumors and other diseases is also noted. Recommendations for a prudent diet are outlined. Complex carbohydrates, especially whole grain cereals and unprocessed fruits and vegetables, should be increased at the expense of fats, simple sugars, and refined carbohydrates. In addition, as much as possible of the daily intake of fat should be in the polyunsaturated form. (24 refs)

- 79-2442 Discussion: Introductory Remarks.** (Eng) James, W. P. (Dunn Nutrition Unit, Old Addenbrookes Hosp., Trumpington St., Cambridge, CB2 1QE, England) *Nutr Cancer* 1(2): 89-103; 1979.

Papers presented at a symposium on nutrition and cancer are summarized following introductory remarks that emphasize the need for better techniques for assessing and validating the dietary habits and intake of individuals in cancer studies. The consensus seems to be that total caloric intake rather than a specific nutrient, such as fat, correlates

with a higher risk for breast cancer in women. In animal studies, however, fat can be considered as distinctive from total calories and the two sets of data seem to follow the same trend. Evidence presented in some papers points to an association between dietary fat and colon cancer or between animal protein, particularly beef consumption, and colon cancer. However, this issue is not clear because most of the dietary information is extremely crude and various studies have resulted in contradictory findings. The decline of gastric cancer seen in many parts of the Western world seems to be caused by the introduction of refrigeration and freezing rather than by the use of antioxidants. It is concluded that too little is known about the relationship between nutrition and cancer to make any recommendations about specific dietary changes to decrease cancer risk. There is a need for more scientific and sophisticated data collection and evaluation techniques regarding food intake and for more prospective studies. (5 refs)

- 79-2443 Mechanism of Action of Diet as a Carcinogen.** (Eng) Weisburger, J. H. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Dana Road, Valhalla, NY 10595) *Nutr Cancer* 1(2): 74-81; 1979.

The mechanism of the dietary induction of cancer of the stomach, breast, prostate, and colon is discussed. A typical population at high risk for gastric cancer consumes a diet high in carbohydrates with limited protein and limited micronutrients on an annual basis, and, of greater importance, low levels of select micronutrients, especially vitamin C, on a seasonal basis. Certain groups also have a predilection for pickled, highly salted, and smoked foods. A working hypothesis states that gastric cancer stems from the formation in the stomach or in pickled foods of alkylnitrosourea compounds. Vitamin C antagonizes the effect of nitrite with respect to the formation of carcinogenic nitrosamines. o-Methylarylamines, which cause colon and breast cancer in male and female rats, have been detected in broiled and fried protein-containing foods. It is possible that these compounds may be the actual carcinogens responsible for the colon and breast cancers. In one study, mutagens were detected in the stools of about half the healthy individuals screened. Whether these mutagens are derived from the metabolism of a mutagen in fried meats or whether they are derived from other precursors is not known. The amount of fecal mutagen can be reduced by increasing dietary fiber intake or by vitamin C. Selenium derivatives have been implicated as protective elements in cancer of the colon, and vitamin A and retinoids have been noted to reduce cancer development in the breast and bladder. (61 refs)

- 79-2444 Nutrition and Breast Cancer.** (Eng) Dickerson, J. W. (Div. Nutrition and Food Science, Dept. Biochemistry, Univ. Surrey, Guildford, Surrey, England) *J Hum Nutr* 33(1): 17-23; 1979.

Present evidence indicates that a high fat intake may be one factor in the etiology of breast cancer (BC). Overnutrition and early menarche, which is influenced by nutritional status, are associated with a high incidence of BC. In experimental animals and humans, there appears to be a correlation between body wt and tumor development, and animals on high-fat diets are more susceptible to the induction of BC by chemical carcinogens. Mortality from BC in humans is strongly positively correlated with animal protein and fat intake. A chronic high fat intake elevates serum prolactin levels and thus raises the prolactin:estrogen ratio, promoting tumor cell growth. The plasma of postmenopausal women with BC contains higher concentrations of total lipids, phospholipids, cholesterol, and lipase activity than that of age-matched women with cancers of other sites. Thiamine deficiency, low WBC ascorbic acid concentrations, hypercalcemia, hypercalciuria, and disturbed tryptophan metabolism have also been reported in BC patients. It is possible that the progress of the disease is associated with an increasing requirement for thiamine and that large amounts of ascorbic acid could be of therapeutic value for BC patients. (41 refs)

- 79-2445 **Preneoplasia in Breast Cancer.** (Eng) Medina, D. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 77030) In: *Breast Cancer 2: Advances in Research and Treatment*. McGuire, W. L., ed. (New York: Plenum Medical Book Company): 407 pp.; 47-102; 1978.

Studies of preneoplastic (PNP) mammary lesions are reviewed with emphasis on the cellular and cell-population characteristics of precursor mammary populations. The principal PNP lesion in mouse mammary glands is the hyperplastic alveolar nodule (HAN), which can be induced by viruses, chemical carcinogens, and prolonged hormone stimulation. In addition to HAN's, two other types of lesions have been identified, ductal hyperplasias and foci of keratinizing alveoli (keratinized nodules: KN). The in vivo behavioral properties of PNP lesions are often intermediate between those of normal and neoplastic tissues; some properties are completely opposite those of neoplastic populations. Their response to hormonal agents and cytostatic drugs suggests that a qualitative change occurs in the transformed cells as they progress from a PNP to a neoplastic state. There appears to be no simple correlation between carcinogen-induced immunosuppression and tumorigenesis in mammary tumors arising in PNP outgrowths. Limited scanning electron microscopy information on normal, PNP, and neoplastic mammary cells in vitro suggests that there are no major differences in surface attachment patterns, microvilli, or number and degree of surface distortions. Neither alterations in cytoplasmic microtubules and microfilaments nor lectin agglutination appear to be of value in distinguishing between normal and PNP mammary cells. It is not clear whether nuclear

magnetic resonance techniques are able to distinguish between normal and PNP states in the breast. (173 refs)

- 79-2446 **Gene Expression in Normal and Neoplastic Breast Tissue.** (Eng) Rosen, J. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 77030) In: *Breast Cancer 2: Advances in Research and Treatment*. McGuire, W. L., ed. (New York: Plenum Medical Book Company): 407 pp.; 337-393; 1978.

This review covers the isolation and characterization of individual rat and mouse milk protein messenger RNA's (mRNA's) and mouse mammary tumor virus (MMTV) RNA, the synthesis of their respective complementary DNA's (cDNA's), and their use in studies of the hormone regulation of gene expression. Casein mRNA was demonstrated in 70% of >30 7,12-dimethylbenz(a)anthracene (DMBA)-induced tumors assayed by molecular hybridization. However, casein mRNA levels were usually $\leq 10\%$ of those observed in an 8-day lactating mammary gland. Casein mRNA was not detected by molecular hybridization using a mouse 15S casein mRNA-cDNA probe in a number of hormone-independent mouse mammary tumor lines. Very low levels of casein mRNA were detected in D2 hyperplastic alveolar nodule tissue, and no detectable sequences were found in autonomous D2 mouse tumors. Therefore, the expression of the casein gene appears to be repressed during mouse mammary tumorigenesis. Very low levels of MMTV RNA were detected in normal mammary tissues. However, high levels of endogenous virus expression were found in the D2 nodules, and a significant number of MMTV RNA sequences were present in the transplantable D2 tumors. Radioimmunoassay of casein in the serum of normal and breast cancer patients indicated the presence of casein in patients with both primary and metastatic breast cancer, but not in normal nonpregnant women. However, negative results for breast cancer patients were obtained in a different laboratory. Thus, the usefulness of casein determination as a diagnostic assay for human breast cancer is unresolved. (149 refs)

- 79-2447 **Non-Trophoblastic Tumours of the Placenta.** (Eng) Fox, H. (Dept. Pathology, Univ. Manchester, Manchester, England) *Major Probl Pathol* 7: 343-367; 1978.

The incidence, origin, pathology, and clinical features of primary and secondary nontrophoblastic tumors of the placenta are reviewed. The only primary types known to occur are hemangiomas and teratomas. Hemangiomas, which probably arise as malformations of the primitive angioblastic tissue of the early placenta, are found in approx 1% of placentas. Hemangiomas are mainly considered to be hamartomatous malformations rather than

true neoplasms. Most are of no clinical importance, but they may cause complications affecting the mother, fetus, or neonate. Teratomas are benign tumors arising from germ cells. They are quite rare and are distinguished from a fetus acardius amorphus by their lack of an umbilical cord and lack of axial organization. Placental neoplasms may also occur as metastases from maternal or fetal neoplasms. Maternal metastases to the placenta occur only in women with widely disseminated malignant disease, and they are most common in patients with malignant melanomas or carcinomas of the breast or bronchus. In the case of malignant melanoma, malignant cells may pass over to the fetus. The dissemination of fetal malignant disease to involve the placenta is extremely uncommon. Neuroblastoma is the only solid fetal tumor reported to have spread to the placenta. There are also examples of fetal leukemia with placental villous involvement. (132 refs)

- 79-2448 **Leukemia in Small Ruminants.** (Rus) Kunakov, A. A. (No affiliation given) *Veterinariia* (12): 76-78; 1978.

Current data on the incidence of leukemia in sheep and goats are reviewed. Clinical manifestations of the disease include wt loss and loss of appetite, paresis of the hind extremities, and tumor formations in the extremities or along the vertebral column. The duration of survival is 2-3 wk. The incidence of leukemia has been found to range from 12/24,080 to 46/1,000,000. (no refs)

- 79-2449 **Infectious and Chronic Disease Epidemiology: Separate and Unequal?** (Eng) Barrett-Connor, E. (Div. Epidemiology, Dept. Community Medicine M-007, Sch. Medicine, Univ. California at San Diego, La Jolla, CA 92093) *Am J Epidemiol* 109(3): 245-249; 1979.

The division of epidemiology into the subspecialties of infectious disease and chronic disease is discussed. Epidemiologically, acute diseases differ from chronic diseases in two major aspects: immediacy of response and uniqueness of observation. Acute and chronic disease epidemiologists have important lessons to offer each other, and the division between epidemiology and epidemiologists into the two subspecialties is detrimental to the study of disease. (13 refs)

- 79-2450 **Pancreas Cancer (Non-endocrine): A Review--Part II.** (Eng) Cubilla, A. L. (Dept. Pathology, Sloan-Kettering Memorial Cancer Center, New York, NY); Fitzgerald, P. J. *Clin Bull* 8(4): 143-155; 1978.

Findings from a retrospective and a prospective study of nonendocrine pancreatic cancer (PC) are reviewed. The increased risk of PC in diabetics is greater than that for any other cancer, especially in women. In the retrospective

study, smoking significantly increased the risk in men. In neither study was there evidence that alcohol increased the risk, but tobacco and alcohol may be cocarcinogens in a small number of patients. There was no consistent combination of factors that might identify asymptomatic patients with early PC. Approx two-thirds of the patients in both studies had Stage III disease at diagnosis. The presence of marked atypia and carcinoma in situ in the duct epithelium adjacent to the cancers suggests that the duct cell is the origin of mucin-producing adenocarcinomas. The high incidence of perineural invasion probably contributes to the retroperitoneal spread and very poor prognosis. The incidence of carcinoma in situ in the duct epithelium of resected specimens suggests that there may be a sufficiently long in situ cancer phase, during which new techniques could be used to detect the disease. These techniques may include detection of pancreatic oncofetal antigen, determination of serum pancreatic ribonuclease levels, the leukocyte adherence inhibition assay, and ultrasonography. (85 refs)

- 79-2451 **Role of Genetic Factors in the Etiology of Esophageal Cancer.** (Eng) Kasenov, K. U. (Dept. Pathophysiology, Medical Inst., Aktiubinsk, USSR) *Vopr Onkol* 25(2): 68-72; 1979.

Data pertaining to the potential role of genetic factors in the etiology and pathogenesis of esophageal cancer are reviewed. Significant differences in the incidence of this cancer among ethnic groups might be indicative of a genetic predisposition. The incidence of esophageal cancer was almost two times higher among relatives of the patients than in a control sample. However, it is strongly emphasized that the results of population and pedigree analyses can also be interpreted as being indicative of similar environmental factors. (37 refs)

- 79-2452 **The Costenbader Memorial Lecture. Genesis and Genetics of Retinoblastoma.** (Eng) Francois, J. (De Pintelaan 135, B-9000 Ghent, Belgium); DeBie, S.; Matton-Van Leuven, M. T. *J Pediatr Ophthalmol Strab* 16(2): 85-100; 1979.

Studies of the development and genetics of retinoblastoma (RB) are reviewed. The incidence of RB is 1/20,000 live births, and 16%-19% of affected children die. About 94% of all RB cases are sporadic and 6% are familial. Hereditary RB's, whether sporadic or familial, represent 40% of all cases. The delayed mutation and multistage mutation hypotheses can explain the clinical characteristics of sporadic and familial RB. The multistage mutation theory, which postulates that at least two mutational events are necessary to convert a normal retinal cell into a RB cell, best fits the observed behavior of RB's. This model, however, does not exclude delayed mutation in the process

leading to the first mutation. RB patients and affected families have a relatively high susceptibility to the development of other malignant tumors, particularly osteogenic sarcomas. The pathological gene for RB may be acquired by a somatic mutation, a germinal mutation, or a chromosome aberration. A new dominant germinal mutation is responsible for 100% of the bilateral sporadic cases and for 10%-15% of the unilateral sporadic cases. A deletion of the long arm of a chromosome (13q14) may be the cause of some, if not all, RB's. (125 refs)

- 79-2453 The Surface Properties of Invasive and Metastatic Tumour-Cell Populations (Meeting Abstract).** (Eng) Poste, G. (Dept. Experimental Pathology, Roswell Park Memorial Inst., Buffalo, NY 14263) *Br J Cancer* 39(4): 467-469; 1979 (19 refs)

- 79-2454 Morphology and Histogenesis of Precancerous Lesions of Cervix Uteri.** (Rus) Iakovleva, I. A. (Dept. Pathomorphology, Moldavian Res. Inst. Oncology, Kishinev, USSR) *Akush Ginekol (Mosk)* (12): 12-15; 1978.

The histological features of various precancerous lesions in the cervix uteri are reviewed. The most frequent precancerous lesion is dysplasia; dysplasia is transformed into carcinoma in situ in 40%-64% of the cases. In 89% of the cases, the dysplasia developed from endocervicitis (pseudoerosion) or during epidermization. In young women, dysplasias are usually diagnosed in the squamous epithelium of the vaginal region of the cervix, but in women >40 yr old, dysplasias are located in the cervical canal. Precancerous changes in the cervix also include leukoplakia, erythroplakia, metaplasia, and adenomatosis. (14 refs)

- 79-2455 The Aetiology of Thyroid Tumours.** (Eng) Williams, E. D. (Welsh Natl. Sch. Medicine, Glamorganshire, Wales) *Clin Endocrinol Metab* 8(1): 193-207; 1979.

The etiology of the major types of human thyroid cancer (papillary, follicular, anaplastic, and medullary carcinoma, and lymphoma) is reviewed. Papillary carcinoma, the most common type of thyroid cancer, is derived from thyroid follicular cells. Thyroid-stimulating hormone (TSH) plays a permissive but not initiating role. The tumor is more common in areas with an iodide-rich diet and more common in some races than others. It can be induced by radiation, at very low doses. Follicular carcinoma occurs more commonly in iodide-deficient areas and is regarded as a TSH-induced tumor. It may also be induced by radiation. Anaplastic carcinoma of the spindle or giant cell type arises from a preexisting differentiated carcinoma. Radiation

and, possibly, viruses are implicated in this transformation. This tumor has an extremely rapid growth rate compared with papillary and follicular carcinomas. There are no known racial or genetic factors involved in the etiology of anaplastic carcinoma. Medullary carcinoma is the only thyroid tumor with a genetic etiology. In the 80% of tumors that are sporadic, the roles of dietary calcium, dietary vitamin D, and radiation remain speculative. Malignant lymphoma is a disease of the elderly, with a marked female preponderance. The great majority of primary lymphomas of the thyroid arise in glands showing severe thyroiditis. (59 refs)

- 79-2456 The Approach to the Irradiated Thyroid.** (Eng) Witt, T. R. (Dept. General Surgery, Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 60612); Meng, R. L.; Economou, S. G.; Southwick, H. W. *Surg Clin North Am* 59(1): 45-63; 1979.

The occurrence and management of pathology resulting from irradiation of the thyroid are reviewed. The mechanism of radiation-induced thyroid carcinogenesis probably involves both injury to the thyroid and effects on the hypothalamic-pituitary axis that lead to elevated thyroid-stimulating hormone levels. The risk of thyroid cancer appears to increase linearly with radiation doses between 20 and 1,125 rads. The incidence of both thyroid cancer and benign thyroid disease are increased by irradiation of the thyroid. The malignancies tend to be well-differentiated, with papillary, follicular, and mixed papillary-follicular types predominating. The clinical recurrence rate in irradiated patients is also increased. Detection of irradiation-associated thyroid disease has been greatly improved by mass screening and patient recall programs. Physical examination, thyroid scan, and examination of biopsied tissue are the most reliable indicators of thyroid malignancies. (69 refs)

- 79-2457 Reemergence of Thyroidectomy as Treatment for Graves' Disease.** (Eng) Klementschtch, P. (Dept. Surgery, Univ. Chicago Hosp., Box 402, 950 E. 59th St., Chicago, IL 60637); Shen, K.; Kaplan, E. L. *Surg Clin North Am* 59(1): 35-44; 1979.

Because of the potential long-term risks of radioiodine therapy, the popularity of thyroidectomy as the definitive treatment for Graves' disease is growing. These potential risks include progressive hypothyroidism, leukemia, genetic abnormalities, and thyroid carcinoma. (26 refs)

- 79-2458 Well-differentiated Carcinomas of the Thyroid.** (Eng) Block, M. A. (Dept. Surgery, Henry Ford Hosp., Detroit, MI) *Curr Probl Cancer* 3(8): 1-60; 1979.

The essential features of well-differentiated thyroid carcinomas (WDTC) are presented. WDTC include the papillary (Pap) and follicular (Fol) varieties and their subsets. In the US, the Pap variety is the most common type of TC. In areas of Europe with high rates of endemic goiter, Fol carcinomas predominate, although the incidence of Pap carcinoma is increasing. The histologic features of WDTC approximate those of normal tissue to varying degrees. Except for the high-grade angioinvasive Fol carcinomas and the Pap or Fol carcinomas with anaplastic elements, WDTC are generally indolent and very slow-growing. WDTC remain occult with a significant frequency and never have an impact on the health of numerous individuals. They present commonly as a nodule in the neck, usually in the thyroid, but occasionally in a cervical lymph node. The presence of microscopic foci of calcification, psammoma bodies, is a specific identifying factor for Pap carcinoma. Little dedifferentiation may be present. There is a predisposition for dissemination via the lymphatic system and for a multifocal origin. Fol carcinoma is composed microscopically of follicles of relatively uniform size but smaller than normal. It is divided into the low-grade, well-encapsulated and the high-grade, angioinvasive varieties. The incidence of multicentricity and bilateral occurrence is increased in WDTC associated with external radiation therapy. A history of previous external radiation to the head and neck; a recently detected, firm, single nodule in the thyroid, especially in a man; ipsilateral cervical lymphadenopathy; a recent appearance of a single thyroid nodule in a patient >40 yr old; or rapid growth of a firm nodule point to a high degree of likelihood of carcinoma for a few thyroid nodules. A variety of tests to facilitate the identification of nodules likely to represent carcinoma are described. (36 refs)

- 79-2459 Clinical Features of Thyroid Tumours.** (Eng) Taylor, S. (Royal Postgraduate Medical Sch., London, England) *Clin Endocrinol Metab* 8(1): 209-221; 1979.

Clinical features of thyroid tumors in 355 patients treated from 1950 to 1975 are discussed. All thyroid tumors, with the exception of medullary carcinoma, are at least twice as common in women as in men. Papillary tumors present as a hard mass in the gland and involved lymph nodes are usually mobile but firm. X-rays may reveal fine stippling due to calcification. Radioisotope scanning will show cold areas where the gland is replaced by tumor. Hoarseness due to paralysis of the vocal cord makes malignancy almost certain. Follicular carcinoma occurs as a solitary, well-encapsulated nodule in the thyroid; it is typical of areas where goiter is endemic. Follicular nodules are usually fairly hard on palpation, occasionally show calcification, and are almost invariably cold on scanning. Anaplastic carcinoma affects old people, mostly women >65. The mass is often multinodular and fixed to surrounding tissues. Anaplastic thyroid tumors spread by direct extension into

the tissues and also by lymphatics and the blood stream. Medullary thyroid carcinoma is a tumor of the calcitonin-secreting parafollicular C-cells. Initial spread is typically by the lymphatics; later, it spreads to the liver and skeleton. Diagnosis rests on the detection of raised plasma calcitonin levels either basally or after stimulation. Lymphoma of the thyroid is associated with autoimmune thyroiditis. Histologically, it consists of small round cells and central, deeply staining nuclei. Rare tumors of the thyroid include teratoma, sarcoma, and squamous cell carcinomas. (23 refs)

- 79-2460 Thyroid Nodules.** (Eng) Beckers, C. (Univ. Louvain, Medical Sch., Brussels, Belgium) *Clin Endocrinol Metab* 8(1): 181-192; 1979.

The clinical status of patients with thyroid nodules, the laboratory evaluation of the nodules and, the pathophysiology of thyroid nodule formation, are reviewed. When nodules are present, the functional status and the morphology of the thyroid gland must be evaluated. The nodular transformation of thyroid tissue results from the interplay of several mechanisms, the most important of which is probably alternate periods of iodine deficiency and iodine repletion. (60 refs)

- 79-2461 Pathology of the Membranes.** (Eng) Fox, H. (Dept. Pathology, Univ. Manchester, Manchester, England) *Major Probl Pathol* 7: 458-472; 1978.

Pathology of extraplacental membranes is reviewed. Abnormalities of the amniotic epithelium include pseudostratification, epithelial disorganization, epithelial cell necrosis, "basket" deformities, and giant goblet cells. Squamous metaplasia is seen on the surface of the amnion and umbilical cord in foci consisting of layers of stratified squamous epithelial cells. It is of no clinical or pathological significance. Amnion nodosum, in which the fetal surface of the amnion is studied with multiple small nodules consisting of deposits of vernix caseosa, should serve as a warning of the possibility of a congenital abnormality in the neonate. Amniotic polypi are derived from the amniotic epithelium; their significance is unknown. Premature rupture of the membranes during the later months of pregnancy, extramembranous pregnancy, extraamniotic pregnancy, and amniotic band formation, a complication of early amniotic rupture, are also covered topically. (77 refs)

- 79-2462 Endocrine Secretions by Lung Carcinomas.** (Fre) Huchon, G. (Centre de Pneumologie, Hopital Tenon, 4, rue de la Chine, 75970 Paris Cedex 20, France); Akoun, G. *Sem Hop Paris* 55(3/4): 180-188; 1979.

Studies of ectopic hormone secretion by lung carcinomas

are reviewed. The overall frequency of endocrine hormone-secreting lung carcinomas is estimated at 10%. Parathyroid hormone is secreted by epidermoid carcinomas, calcitonin by small cell anaplastic carcinomas and carcinoid adenomas, antidiuretic hormone by small cell anaplastic carcinomas, ACTH and melanocyte-stimulating hormone by small cell anaplastic carcinomas and carcinoid adenomas, and gonadotropins (human chorionic gonadotropin, luteinizing hormone, and follicle-stimulating hormone) by all histologic types of lung carcinoma. Hypotheses concerning these ectopic secretions are given. (92 refs)

- 79-2463 Cell Surface Glycoproteins and Malignant Transformation.** (Eng) Yamada, K. M. (Lab. Molecular Biology, NCI, Bethesda, MD 20014); Pouyssegur, J. *Biochimie* 60(11/12): 1221-1223; 1978.

The role of cell-surface glycoproteins in cell adhesion, cell-cell recognition, uptake of nutrients, and growth control during malignant transformation is reviewed. Alterations in cell behavior that occur after malignant transformation are summarized briefly, and the many accompanying changes in individual cell-surface proteins are described in detail. Two major approaches have provided insight into the causal relationships of glycoprotein changes to altered properties of transformed cells: (1) isolation, characterization, and reconstitution of each cell-surface component, and examination of the ability of the reconstituted protein to restore functions altered in transformed cells; and (2) isolation and characterization of cell mutants in each specific function, and examination of which aspects of transformation result from alterations in each function. The apparent relationships between altered cell-surface proteins and biological properties found in many transformed cells are outlined schematically. In the fibroblast tissue culture model, many characteristics of transformed cells, such as morphology and cell alignment, appear to depend on cell adhesion, which is often defective due to alterations in fibronectin, cyclic AMP, and cell-surface carbohydrates. Many of these defects can be reproduced by altering adhesion, whether by inhibiting glycosylation, inactivating fibronectin, or by growing cells on poorly adhesive substrates. (188 refs)

- 79-2464 Trophoblastic Tumours of the Placenta.** (Eng) Fox, H. (Dept. Pathology, Univ. Manchester, Manchester, England) *Major Probl Pathol* 7: 368-425; 1978.

The pathology of trophoblastic disease is reviewed, with particular emphasis on the clinico-pathological aspects of hydatidiform mole and gestational choriocarcinoma. Histologically, the following types of 'choriomas' were recognized originally; hydatid mole, chorioadenoma

destruens, choriocarcinoma, syncytial endometritis, and syncytioma. The precise cause of gestational trophoblastic tumors remains obscure, but the tumors show a marked geographical variation in distribution, and genetic factors appear to play a part in their etiology. A hydatidiform mole may be regarded as an abnormal product of gestation, and it is considered to be invasive when molar villi have penetrated into the myometrium or its blood vessels. Its histopathology, ultrastructure, pathogenesis, and prognosis are reviewed. Choriocarcinoma is composed of both cytotrophoblasts and syncytiotrophoblasts, but it is without chorionic villi. The most striking clinical feature of this tumor is the remarkable reversal in mortality brought about by the introduction of cytotoxic therapy. Its histopathology, macroscopic and microscopic appearance, metastatic spread, diagnosis and immunological aspects are surveyed. (174 refs)

- 79-2465 Asbestos and Mesothelioma.** (Ger) Ferlinz, R. (Abteilung für Pneumologie, Klinikum der Universität Mainz, Langenbeckstrasse 1, 6500 Mainz, W. Germany); Endres, P. *Dtsch Med Wochenschr* 103(6): 1055-1056; 1978.

The relationships between asbestos exposure and lung cancer and pleural and peritoneal mesothelioma are reviewed. A dose-effect relationship is probable in the case of mesothelioma. Recent studies suggest an increased incidence of malignant gastrointestinal tumors in patients with asbestosis. (3 refs)

- 79-2466 Talc in Normal and Malignant Ovarian Tissue (Letter to Editor).** (Eng) Henderson, W. J. (Tenovus Inst. Cancer Res., Welsh Natl. Sch. Medicine, Cardiff CF44XX, Wales); Hamilton, T. C.; Griffiths, K. *Lancet* 1(8114): 499; 1979.

Confirmatory evidence from a 1971 report showing that talc particles were present in certain ovarian and uterine cervix tumor tissue and normal tissue is cited to refute an editorial statement that the original report was probably erroneous. (13 refs)

- 79-2467 Risks and Benefits of the Treatment of Psoriasis.** (Eng) Epstein, J. H. (Univ. California Sch. Medicine, San Francisco, CA 94108) *N Engl J Med* 300(15): 852-853; 1979.

Problems related to the treatment of psoriasis are reviewed briefly. Treatment with coal tar and short-range UV radiation has not resulted in noteworthy skin cancer formation, despite their usage for >50 yr. In contrast, the use of combined psoralens and long-range UV radiation has been

associated with an epidermal cancer incidence 2.63 times that expected for a control population. This may be due to the fact that most patients require maintenance psoralen photochemotherapy at relatively short intervals. This type of therapy must still be considered experimental. (13 refs)

- 79-2468 Immunologic Surveillance Revisited.** (Eng) Moller, G. (Dept. Immunobiology, Karolinska Int., Wallenberglab., Lilla Frescati, S-104 05, Stockholm 50, Sweden); Moller, E. *Transplant Proc* 11(1): 1041-1046; 1979.

Evidence for the monoclonality of tumors is discussed, and the relationship between genetic changes, immunodeficiency, and cancer is considered. A variety of lymphatic tumors are of monoclonal origin, including Waldenstrom's macroglobulinemia, chronic lymphatic leukemia, and chronic lymphosarcomatous leukemia. Other tumors from a variety of anatomical locations, including carcinomas, sarcomas, and various lymphoid tumors are also monoclonal. It is suggested that carcinogens may only accelerate the appearance of rare genetic changes leading to neoplasia. The concept that carcinogens cause tumors by a genetic mechanism is supported by the evidence that mutagenicity and carcinogenicity occur in parallel, provided the carcinogens have been metabolized in the body. If the concept of immune surveillance were true, tumors in immunosuppressed individuals would not be monoclonal. It appears, however, that such tumors are monoclonal. It is concluded that a major mechanism of carcinogenesis by chemical and physical carcinogens is somatic mutations caused by errors during the repair of DNA damaged by carcinogens. (23 refs)

- 79-2469 Acute Leukemia and Immunosuppressive Drug Use. A Review of Patients Undergoing Immunosuppressive Therapy for Non-neoplastic Diseases.** (Eng) Grunwald, H. W. (Div. Hematology, Queens Hosp. Center Affiliation, Long Island Jewish-Hillside Medical Center, 82-68 164th St., Jamaica, NY 11432); Rosner, F. *Arch Intern Med* 139(4): 461-466; 1979.

Sixty-one reported cases of acute leukemia developing in patients who had previously received immunosuppressive agents for nonneoplastic disorders are reviewed. In three patients, the diagnosis of acute leukemia was made <6 mo after the first exposure to immunosuppressive drugs and was, therefore, considered coincidental. Among the remaining 58 patients, most diagnoses were of myeloblastic or myelomonocytic leukemia. The interval from immunosuppressive therapy to the development of acute leukemia ranged from 0.75-18 yr, with a mean of 4.8 yr. The underlying diagnoses in most of the 58 patients were rheumatoid arthritis or renal disease, or the patients were renal transplant recipients. Thirty patients had received

alkylating agents exclusively, 10 had received antimetabolites only, and the remaining 18 patients had received multiple therapeutic modalities including antimetabolites, alkylating agents, and/or radiation. Most patients had also received large amounts of corticosteroids. Although the overall denominator of population at risk is not known, the relatively high proportion of patients who had received alkylating agents before developing acute leukemia does suggest a causal relationship. (71 refs)

- 79-2470 Surface Antigens on Human Herpesvirus-infected and -transformed Cells.** (Eng) Lausch, R. N. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033); Rapp, F. In: *Virus Transformed Cell Membranes* 373-396; 1978.

The expression of virus-associated antigens on the surface of cells infected or transformed by the human herpesviruses, herpes simplex virus (HSV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV), is reviewed. Virus-associated antigens can be demonstrated on the surface of cells transformed by HSV, CMV, and EBV by a variety of serological assays. Antigens are also demonstrable in cellular immunity tests. Serum from virus-immunized hosts can specifically block the cytotoxicity of sensitized lymphocytes for HSV- or CMV-transformed cells, indicating that the humoral and cellular immune responses are probably directed against similar antigenic determinants. There is evidence that the membrane antigens (MA) found on transformed cells are also expressed on infected cells and on the virion envelope, which indicates that the antigens are coded for by the virus genome. There is no evidence of cross-reacting virus-associated membrane antigens on cells transformed by HSV, CMV, or EBV. The occasional spontaneous regressions observed in Burkitt's lymphoma patients may be the result of a cellular immune response to EBV surface markers. Immunization of syngeneic hosts with HSV or CMV has not resulted in protection against challenge with homologous tumor cells. Thus, MA may not function as a transplantation rejection antigen; alternatively, antibody may interfere with cell-mediated immunity via masking of antigen or induction of antigen modulation. (149 refs)

- 79-2471 Viruses and Mammary Carcinoma.** (Eng) Schlom, J. (NCI, NIH, Bethesda, MD 20014); Colcher, D.; Drohan, W.; Kufe, D.; Teramoto, Y. A. In: *Breast Cancer 2: Advances in Research and Treatment*. McGuire, W. L., ed. (New York: Plenum Medical Book Company): 407 pp.; 23-46; 1978.

Cell culture, nucleic acid, and immunological studies of murine mammary tumor viruses (MMTV's) are reviewed. Growth of mouse mammary tumors in densely seeded

primary mammary tumor cultures containing insulin and hydrocortisone allows the production of pure MMTV from all mouse strains examined. Molecular hybridization studies indicate that the viral genomes of the 60S-70S RNA's of MMTV's released from early mammary tumors of several mouse strains are at least 95% related in their nucleic acid sequences. There is a substantial difference, however, between the MMTV released from early C3H mammary tumors and that released from late C3H mammary tumors. Molecular hybridization and recycling experiments demonstrate that there are proviral sequences in early mammary tumors that are non-germ line-transmitted. The only mouse strain examined in which these recycled MMTV(C3H) sequences are found in apparently normal livers is the GR mouse, a strain in which there is genetic evidence for a one-gene dominant characteristic for MMTV. Qualitative differences between MMTV's of different mouse strains were also determined by the thermal analysis of hybrids. MMTV's derived from different mouse strains contain both type-specific and group-specific reactivity, both of which are associated with the 52,000-dalton major external glycoprotein (gp52) of the MMTV virion. The type and group specificities of MMTV's grown in feline cells are indistinguishable from those observed in murine-grown MMTV's, providing strong evidence that the MMTV gp52 antigens are virus-coded. (37 refs)

- 79-2472 Genetic Regulation of Expression of Endogenous Viruses in Mice.** (Pol) Dus, D. (Zaklad Immunologii Nowotworow, Instytut Immunologii i Terapii, Doswiadczalnej PAN, Czerska 12, 53-114 Wroclaw, Poland); Szkudlarek, J.; Radzikowski, Cz. *Postepy Hig Med Dosw* 32(6): 683-702; 1978.

Studies of the genetic regulation of endogenous oncornavirus expression in mice and on the transmission, activation, and biological role of these viruses are reviewed. C-type endogenous viruses in mice can be activated by halogenated derivatives of pyrimidine, protein synthesis inhibitors, amino acid analogs, herpesvirus, and by the graft-vs-host reaction. The endogenous virus MTV-X, which induces mammary cancer in mice, is activated in vivo by ionizing radiation. (93 refs)

- 79-2473 Virus-associated Cell Surface Products: Relevance to Malignant Transformation** (Meeting Abstract). (Eng) Wyke, J. (ICRF, London, England). *BR J Cancer* 39(4): 458-459; 1979. (no refs)

- 79-2474 RNA Tumor Virus Genes and the Transforming Genes: Genetic Transmission, Infectious Spread, and Modes of Expression.** (Eng) Todaro, G. J.

(Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014) *Natl Cancer Inst Monogr* (48): 199-213; 1978.

The genetic conservation and evolution of virogenes in mammalian species are described in relation to the horizontal, vertical, and congenital transmission of C-type RNA oncogenic viruses within members of a given species or among members of near and/or distantly related species. Examples of oncogenic virus infection between ancestors of the mouse, cat, pig, and primates as well as integration of the virus into the host's genome are documented. There are several possible normal functions of the virogenes as a part of the cellular genome of many species. They may play a role during development, they may provide protection against diseases caused by related but more virulent viruses, and they may provide protection on an immunological basis against cancer. Recent evidence of genetic recombination (genetic mixing) among distinct C-type viruses is also reviewed, thus completing an overview of the evolutionary past, present, and complex future relationships of oncogenic RNA viruses and mammalian species. (130 refs)

- 79-2475 Glycolipids in Virus-transformed Cells.** (Eng) Steiner, S. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Steiner, M. R. In: *Virus-transformed Cell Membranes*. Nicolau, C., ed. (London: Academic Press): 393 pp.; 91-110; 1978.

Studies of glycolipid composition and metabolism in tumor cells are reviewed. Alterations in glycolipid metabolism have been found in human solid tumors and in leukemic lymphocytes. Cells transformed by DNA and RNA tumor viruses show a decrease in the more complex glycolipids. The specific glycolipid alteration is a function of both the particular cell line and the transforming virus. Glycolipid metabolism varies not only as a function of oncogenic transformation but also, in untransformed cells, as a function of cell population density. Gangliosides may serve as receptors for molecules such as interferon and the plant lectin ricin. The possible function of gangliosides as surface membrane receptors implies that the lipids are located on the surface membrane and are external and accessible. An alteration in glycolipid antigenicity, which may be related to a change in accessibility, has been observed in virus-transformed cells. There are also immunological differences between normal and transformed cells that appear to be related directly to differences in their glycolipid composition. The composition and distribution of amino acyl fucosides, neoproteolipids, and phospholipids also differs between normal cells and their transformed counterparts. The particular changes may be a function of both the transforming agent and the specific affected cell, which would account for the pleiotropic alterations encountered in neoplastic cells. (114 refs)

- 79-2476 Surface Proteins of Virus-transformed Cells.** (Eng) Vaheri, A. (Dept. Virology, Univ.

Helsinki, Helsinki, Finland) In: *Virus Transformed Cell Membranes*. Nicolau, C., ed. (New York: Academic Press): 408 pp.; 1-89; 1978.

The mechanism and significance of surface protein changes that occur during viral transformation are reviewed. For most in vitro work on viral oncogenesis and transformed cells, diploid fibroblasts or untransformed established fibroblastic cell lines are used to represent normal cells. Hematopoietic cells and human glial cells are also used. Fractionation, specific labeling of cell-surface proteins, polyacrylamide gel electrophoresis, and immunochemical

methods are among the techniques used to study cell-surface proteins. Particular emphasis is placed on the interactions, during transformation, between surface proteins and other cell-surface components, ie, membrane proteins, lipids, submembranous structures, and external glycosaminoglycans, and extracellular components such as plasma proteins, enzymes, and their inhibitors. The functional aspects of altered surface proteins in normal cell physiology and in the mechanism of viral transformation are stressed. Although working hypotheses and the prospects arising from the data are discussed, there is no attempt to construct unifying theories. (618 refs)

CHEMICAL CARCINOGENESIS

79-2477 Study of Phospholipid Absorption by Chrysotile Fibers: Comparison of Chemical and Photoelectron Spectrometric (XPS Method) Data. (Fre) Jaurand, M. C. (Laboratoire de Biopathologie pulmonaire, C.H.U. Henri-Mondor 51, avenue du Marechal-de-Lattre-de-Tassigny, 94000 Creteil, France); Thomassin, J. H.; Magne, L.; Baillif, P. *C R Acad Sci [D] (Paris)* 288(2): 279-282; 1979.

The adsorption of synthetic dipalmitoyl phosphatidylcholine (DPPC) by chrysotile fibers was studied following a 15-min incubation at 37 C. The max adsorption was approx 130 µg/mg chrysotile. The results are in good agreement with the bilayer adsorption hypothesis. (13 refs)

79-2478 Thymectomy and Asbestos-induced Mesotheliomas in Rats. (Eng) Wagner, M. M. (Pneumoconiosis Unit, Medical Res. Council, Penarth, Wales) *Br J Cancer* 39(3): 337-341; 1979.

The effects of thymectomy on the induction of mesotheliomas by crocidolite asbestos were studied in Wistar rats. The rats were thymectomized or sham-operated before 4 days of age and then treated with the asbestos (20 mg in physiological saline inoculated into the pleural cavity). Some animals were given three intrapleural injections of carrageenan after asbestos treatment. The carcinogenicity factor (a measure of tumor growth rate) was significantly increased in the thymectomized rats relative to the sham-thymectomized rats, but it was not significantly different from that in the unoperated controls. The carcinogenicity factor was significantly reduced ($p < 0.01$) in the sham-operated rats compared with the intact rats. Intrapleural carrageenan was associated with an 11-fold increase in the carcinogenicity factor relative to the sham-operated rats. There was no significant difference in age at tumor occurrence in any of the groups. The data indicate that thymectomy before the fourth day of age does not alter the carcinogenicity factor of crocidolite, whereas surgical intervention alone at the same age markedly reduces it. (27 refs)

79-2479 Gallium-67 Citrate (⁶⁷GA) Uptake in Benign and Malignant Pleural Disease (Meeting Abstract). (Eng) Sorek, M. (Dept. Oncology, Mount Sinai Medical Center, New York, NY); Teirstein, A. S.; Goldsmith, S. J.; Chahinian, P. *Clin Res* 27(2): 491A; 1979 (no refs)

79-2480 Natural Carcinogenic Products. (Eng) Weisburger, E. K. (NCI, Bethesda, MD 20014) *Environ Sci Technol* 13(3): 278-281; 1979.

Naturally occurring carcinogens produced by plants or by microbes are enumerated along with the species and organs known to be affected by these agents. (7 refs)

79-2481 Carcinogenicity Examination of Betel Nuts and Piper Betel Leaves. (Eng) Mori, H. (Dept. Pathology, Gifu Univ. Sch. Medicine, 40 Tsukasa-machi Gifu 500, Japan); Matsubara, N.; Ushimaru, Y.; Hirono, I. *Experientia* 35(3): 384-385; 1979.

ACI rats were divided into four groups (containing 8-11 males, 8-9 females each) and fed diets containing a dry powder of (1) betel nuts (20% of diet) for 480 days, (2) betel nuts (20%) + lime (1%) for 480 days, or (3) piper betel leaves (20%) for 300-327 days or (4) a normal diet. The betel nuts, betel leaves, and lime are the main ingredients of the betel quid that is chewed by Southeast Asians. Epidermal thickening was frequently observed in the upper digestive tracts of Groups 2 and 3 rats. A forestomach papilloma was seen in one rat in Group 3. The epidermal changes were rarely seen in rats of Groups 1 or 4. One transitional cell carcinoma of the urinary bladder and one myeloid leukemia were detected in Group 1, and one myeloid leukemia was detected in Group 2. These results suggest, but do not demonstrate, the carcinogenicity of chewing betel quid. (14 refs)

79-2482 Inhibition of Chemical Carcinogen-induced Neoplasia by Coumarins and α -Angelicalactone. (Eng) Wattenberg, L. W. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455); Lam, L. K.; Fladmoe, A. V. *Cancer Res* 39(8): 1651-1654; 1979.

Four naturally occurring plant constituents, coumarin (CM), umbelliferone (7-hydroxycoumarin), scopoletin (7-hydroxy-6-methoxycoumarin), and limettin (5,7-dimethoxycoumarin), were studied for their effects on 7,12-dimethylbenz(a)anthracene-induced neoplasia of the Sprague-Dawley rat mammary gland. CM was a moderately potent inhibitor, limettin was less effective, and scopoletin showed only marginal inhibitory activity. Umbelliferone did not inhibit. CM and its three derivatives were also investigated for their effects on benzo(a)pyrene (BP)-induced neoplasia of the ICR/Ha mouse forestomach.

CM inhibited, but the three derivatives did not. Coumaranone and phthalide, two related compounds, were inactive, as were three substituted pyrones included in the study. Four five-membered ring lactones were also investigated. One of these, α -angelicalactone, inhibited BP-induced neoplasia of the mouse forestomach and was more potent in this regard than CM. The other three, γ -valerolactone, L-ascorbic acid, and isocitric lactone, were inactive. Three structure-activity relationships are evident from this study. With the CM's, increased polarity of substituents results in decreasing activity as inhibitors. For both the CM's and the five-membered ring lactones, protic groups, such as hydroxy and carboxy groups, abolish the capacity to inhibit. Although unsaturation in the lactone ring does not always lead to inhibitory activity, the presence of at least one double bond is essential. Thus, the property of inhibition of BP and 7,12-dimethylbenz(a)anthracene is not a general characteristic of all CM's and alicyclic lactones but is restricted to those with specific structural features. (19 refs)

- 79-2483 Testing the Environment for Dispersed Mutagens: Use of Plant Bioconcentrators Coupled with Microbial Mutagen Assays.** (Eng) Barnes, W. S. (Dept. Botany, Univ. Massachusetts, Amherst, MA 01003); Klekowski, E. J. *Environ Health Perspect* 27: 61-67; 1978.

Data on an assay that utilizes certain indigenous or introduced components of an ecosystem to concentrate dispersed mutagens from the environment are presented. Extracts made from these bioconcentrators are fractionated and tested for mutagenic activity in microbial systems. The assay is termed a coupled assay, since it couples an accumulator of environmental mutagens with a microbial system. There are numerous plant and animal species that concentrate toxic substances from the environment; eg, mussels, algae, and aquatic angiosperms such as the water hyacinth. Organisms most widely used to screen for genetic activity are strains of *Escherichia coli* or *Salmonella typhimurium* containing frameshift or base substitution mutations that revert in the presence of mutagens specific for these effects. However, fractions can be tested with a broad range of microbial assays covering numerous genetic end points both with and without mammalian microsomal activation. Compared with physicochemical techniques, the sampling techniques in the coupled assays are simpler and a wider chemical spectrum can be screened. However, there are certain inherent aspects of these procedures that may generate false positives (either the presence of the necessary metabolite in the extract when the microbial test depends on a back mutation system from auxotrophy to prototrophy, or the extraction of an endogenous mutagen) or false negatives (presence of microbial toxins in the extract, inactivation of the mutagen by the bioconcentrator, or selection of a mutagen assay with the wrong genetic end point). In spite

of these obstacles, assays based on bioconcentrators will have a significant role in screening ecosystems. (71 refs)

- 79-2484 Aflatoxin B₁ Mutagenesis, DNA Binding, and Adduct Formation in *Salmonella typhimurium*.** (Eng) Stark, A. A. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139); Essigmann, J. M.; Demain, A. L.; Skopek, T. R.; Wogan, G. N. *Proc Natl Acad Sci USA* 76(3): 1343-1347; 1979.

The quantitative relationship between the binding of aflatoxin B₁ (AFB₁) to *Salmonella typhimurium* strain TM677 DNA, in the form of specific adducts, and forward mutation to 8-azaguanine resistance (8AGR) was studied. *Salmonella* was treated with AFB₁ in liquid suspension culture in the presence of a rat liver postmitochondrial supernatant. A linear relationship was obtained between the induced 8AGR fraction and the concentration of AFB₁. The efficiency of mutagenesis was high: 0.1% of all cells became 8AGR at 0.32 μ M AFB. DNA purified from mutagenized cells was analyzed for AFB₁ adduct formation by high-pressure liquid chromatography after adduct liberation. AFB₁ exposures at 0.16 and 0.32 μ M for 35 min produced 15 and 22 AFB₁-DNA adducts per genome, respectively, and induced 8AGR fractions of 4.9×10^{-4} and 9.6×10^{-4} . Seventy percent of the AFB₁ bound to DNA was identified chromatographically as 2,3-dihydro-2-(N⁷-guanyl)-3-hydroxyaflatoxin B₁, at the two AFB₁ levels used. These findings support the hypothesis of a cause-effect relationship between DNA adduct formation by AFB₁ and mutagenesis and carcinogenesis. The possibility cannot be ruled out, however, that the mutagenic activity of AFB₁ in *Salmonella* results from interactions with macromolecules other than DNA, since the DNA contained only 2% of the total covalently bound AFB₁. (25 refs)

- 79-2485 Cytotoxic Effects of Aflatoxin B₁ on Pulmonary Cells In Vitro (Meeting Abstract).** (Eng) Oglesby, L. A. (North Carolina State Univ. at Raleigh, Raleigh, NC) *Diss Abstr Int [B]* 39(9): 4225; 1979 (no refs)

- 79-2486 Induction of Both Epoxide Hydratase and a Second Microsomal Membrane Polypeptide by Hepatocarcinogens (Meeting Abstract).** (Eng) Murray, R. K. (Univ. Toronto, Toronto, Ontario M5S 1A8, Canada); Sharma, R. N.; Gurtoo, H. L.; Griffin, M. J.; Cameron, R. G.; Farber, E. *Fed Proc* 38(3, part 2): 660; 1979 (2 refs)

- 79-2487 Conditions for Induction of Bacteriophage from Lysogenic *Bacillus megaterium* with**

Aflatoxin B₁. (Eng) Whittaker, B. L. (Dept. Poultry Science, Ohio State Univ., Columbus, OH 43210); Chipley, J. R. *Appl Environ Microbiol* 37(3): 554-558; 1979.

An attempt was made to determine whether or not aflatoxin B₁ (AFB) was an effective inducing agent for lysogenic bacteria and to characterize some of the parameters involved in induction. A lysogenic strain of *Bacillus megaterium* (NRRL-B-3695) and an indicator strain of this species (NRRL-B-3694) were used. Cultures of the lysogenic strain were incubated for various periods of time in the presence of AFB. Plaque-forming units as well as colony-forming units were then determined. The results indicated that bacteriophage lysogenizing *B. megaterium* could be induced with AFB. The optimum AFB concentration for induction was 25 µg/ml of early-log-phase culture. Evidence suggested that (1) higher concentrations of AFB formed hydrophobic complexes that would not efficiently induce *B. megaterium*; (2) the toxic effect of AFB severely limited the number of cells that could be induced prior to the killing action of the toxin; and (3) AFB concentrations <25 µg/ml were not efficient inducers of bacteriophage production and they did not demonstrate the toxic effect observed at higher concentrations. (20 refs)

79-2488 Investigations of In Vitro Liver Metabolism of Aflatoxin B₁ by the Young Dairy Calf (Meeting Abstract). (Eng) Bodine, A. B. (Clemson Univ., Clemson, SC 29631) *Diss Abstr Int B* 39(8): 3764; 1979 (no refs)

79-2489 Chemical Compounds in Food-producing Animals. Criteria and Procedures for Evaluating Assays for Carcinogenic Residues. (Eng) Gardner, S. (Food and Drug Admin., 5600 Fishers Lane, Rockville, MD 20857) *Fed Regist* 44(55): 17070-17114; 1979.

Procedures and criteria proposed by the Food and Drug Administration to ensure the absence of cancer-causing residues in edible products of animals to which drugs, food additives, or color additives have been administered are detailed. Evaluation of the carcinogenic risk involves a number of steps including threshold assessment (use, residues of toxicological concern, and potential toxicological significance), metabolic studies in target animals to identify residues of concern, chronic toxicity testing, metabolic studies to select marker residues and target tissues (the level of a marker residue in a particular tissue is in a known relationship to the level of the total residue of carcinogenic concern in all edible tissues), evaluation of a compound's effect on pools of carcinogenic or potentially carcinogenic substances endogenous to target animals, and determination of the acceptable level of risk. Procedures

for establishing withdrawal periods, for ensuring compliance, for waiver of requirements, and for implementation are summarized. (79 refs)

79-2490 Preserved Foods as Possible Cancer Hazards: WA Rats Fed Salted Fish Have Mutagenic Urine. (Eng) Fong, L. Y. (Dept. Biochemistry, Univ. Hong Kong, Hong Kong); Ho, J. H.; Huang, D. P. *Int J Cancer* 23(4): 542-546; 1979.

The mutagenicity of foods traditionally and commonly consumed by the southern Chinese, including two samples of dried shrimp and four samples of different species of salted fish, was tested in the *Salmonella typhimurium* microsome mutagenicity assay using tester strains TA98 and TA100. Mutagenic activities toward both tester strains were found in all preparations. In most cases, these activities were enhanced by liver microsome activation. Urine collected from Wistar albino (WA) rats regularly fed salted fish also showed mutagenic activity. The level of this activity decreased markedly when the rats were transferred from a salted fish diet to a Purina rat chow diet. The data suggest that there are mutagenic substances in some preserved Chinese foods. At least one of them, salted fish, has been suspected, based on epidemiological and experimental evidence, of being a possible cocarcinogenic factor in the development of nasopharyngeal carcinoma in the southern Chinese. (27 refs)

79-2491 Pesticides. (Eng) Abdulla, M. (Unit Community Care Sciences, Natl. Board Health and Social Welfare, Dalby, Sweden); Dencker, I.; Haglund, G.; Persson, K.; Norden, A.; von Rosen, G. *Scand J Gastroenterol Suppl* 14(52): 217-222; 1979.

Residue levels of some common pesticides in the normal diet of elderly persons in a region of Sweden were measured and found to be appreciably less than those in many parts of the world. (39 refs)

79-2492 The Effect on Sister-Chromatid Exchanges of Drugs and Dyes by Intercalation and Photoactivation. (Eng) Speit, G. (Institut für Humangenetik und Anthropologie der Universität, D-7800 Freiburg i.Br., W. Germany); Vogel, W. *Mutat Res* 59(2): 223-229; 1979.

Intercalating dyes [acridine orange (AO), proflavin (PF), and methylene blue (MB)] and drugs [chlorpromazine (CPZ), promazine, and chlorprothixene] were tested for their ability to induce sister chromatid exchanges (SCE's) in a clonal derivative of the V79 Chinese hamster cell line with and without photoactivation by visible light. All three

drugs induced SCE's, the frequency increasing at concentrations between 0.25 and 1.0 $\mu\text{g/ml}$ and then leveling off at a nearly twofold increase at concentrations between 1.0 and 5 $\mu\text{g/ml}$. All drugs showed cytotoxic effects, making tests at higher concentrations impossible. Similar dose-effect curves were obtained with the dyes, which caused cytotoxicity and induced SCE's at concentrations 10 times lower than those of the drugs. Visible light alone increased the frequency of SCE's slightly. However, it doubled the toxicity and the effectiveness of PF and AO in inducing SCE's. In contrast, the results obtained with MB and CPZ were similar before and after photoactivation. (23 refs)

- 79-2493 Formation of Nitrosamines from Food Dyes and Effect of Additives Thereon.** (Eng) Banerjee, T. S. (Central Food Lab., Calcutta, India); Roy, B. R. *J Inst Chem (India)* 50(part 3): 134-138; 1978.

The production of nitrosamines by food dyes containing secondary and tertiary amino groups (indigocarmine, Fast Green FCF, Green S, Blue FCF, and Metanil Yellow) was studied. On reaction with sodium nitrite and acid, all five dyes produced nitrosamines, the amounts being greater with the dyes containing tertiary amino groups. Ascorbic acid, glucose, and sodium bisulfite inhibited the reaction, possibly by reduction of nitrous acid. It has been hypothesized that nitrosamines must be transformed into diazoalkanes to be carcinogenic and that this is possible only if both carbon atoms attached to the nitrogen carry hydrogen atoms. Therefore, in view of the structure of the dyes in this study, the nitrosamines produced may not be carcinogenic. (43 refs)

- 79-2494 Order Denying Petition for Food Additive Regulation on Gentian Violet.** (Eng) Luther, L. W. (Bureau Veterinary Medicine, Food and Drug Admin., Dept. Health, Education and Welfare, 5600 Fishers Lane, Rockville, MD 20857) *Fed Register* 44(63): 19035-19038; 1979.

Responses to a petition proposing that food additive regulations be amended to provide for the safe use of 1 lb of a 1.60% gentian violet premix per ton of poultry feed as an aid in controlling fungus and mold growth are presented. The safety of gentian violet as an additive to animal feed has not been established, and there is some evidence that it may be toxic, mutagenic, and carcinogenic to animals. Also, the effectiveness of Gentian Violet in inhibiting mold growth in animal feed has not been established. Furthermore, the environmental impact analysis report as submitted is incomplete, data verifying the chemical identity and composition of the gentian violet proposed for use are lacking, and there are no data demonstrating that the product will possess a uniform identity, strength, quali-

ty, and purity for a given period of time. Thus, the Food and Drug Administration is denying the petition. (39 refs)

- 79-2495 Neoplasms and Pigmentation of Thyroid Glands in F344 Rats Exposed to 2,4-Diaminoanisole Sulfate, a Hair Dye Component.** (Eng) Ward, J. M. (Carcinogenesis Testing Program, Tumor Pathology Branch, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014); Stinson, S. F.; Hardisty, J. F.; Cockrell, B. Y.; Hayden, D. W. *J Natl Cancer Inst* 62(4): 1067-1073; 1979.

The pathology of the thyroid lesions induced in rats by 2,4-diaminoanisole sulfate (2,4-DAAS), a hair dye component, is reported in detail. The compound was fed at dietary levels of 0.12% (low dose) or 0.5% (high dose) to groups of 50 male and 50 female inbred F344 rats for 78 wk. By 107 wk after the initial exposure, 58% of the male rats and 42% of the female rats administered the high dose had thyroid neoplasms, vs only 7%-8% of the controls. Follicular cell carcinomas were the primary type of neoplasm induced. None of the controls had these tumors. The carcinomas were papillary, cystic, or solid, and they were highly invasive locally but did not metastasize. A brown pigment was present as granules primarily in thyroid follicular cells in all exposed rats. The amount of pigment, as determined by an image-analyzing computer, revealed that the cross-sectional area occupied by the pigment granules and the optical density of the granules were dose-related. This pigment may represent a reaction product of 2,4-DAAS and iodine. The manufacturers of human hair dyes have recently agreed to remove 2,4-DAAS from commercial hair dye preparations. (15 refs)

- 79-2496 Determination of Cadmium, Lead and Copper in Wine by Differential Pulse Anodic Stripping Voltammetry.** (Eng) Oehme, M. (Dept. Chemistry, Univ. Oslo, Box 1033 Blindern, Oslo 3, Norway); Lund, W. *Fresenius Z Anal Chem* 294(5): 391-397; 1979.

The simultaneous determination of cadmium, lead, and copper in wine by differential pulse anodic stripping voltammetry at a hanging mercury drop electrode is described. The samples are decomposed in a mixture of sulfuric acid and hydrogen peroxide at 180 C. The procedure was evaluated by recovery tests and compared with other wet digestion methods. Pb levels in 10 wine samples (65-230 ppb) were below the accepted max level, but some wines contained relatively large amounts of Cu (0.08-1.04 ppm). Cd levels were low (1.4-6.6 ppb). (19 refs)

- 79-2497 Ganglioneuroblastoma and Fetal Hydantoin-Alcohol Syndromes.** (Eng) Seeler, R. A. (Div.

Pediatric Hematology, Cook County Hosp., 1825 W. Harrison St., Chicago, IL 60612); Israel, J. N.; Royal, J. E.; Kaye, C. I.; Rao, S.; Abulaban, M. *Pediatrics* 63(4): 524-527; 1979.

A ganglioneuroblastoma developed in a 35-mo-old boy with both the fetal hydantoin (FHS) and fetal alcohol syndromes. The mother had consumed at least a pint of alcohol a day during the pregnancy, and she had been on a regimen of diphenylhydantoin (100 mg 3x/day) until the third or fourth month of pregnancy. Chromosome analysis showed a normal male karyotype. Skeletal survey showed a bilateral absence of the ossification centers of the terminal phalanges of the fifth toes. There were no intracranial calcifications or evidence of bone metastasis. The left kidney was displaced by a suprarenal mass that was identified as a ganglioneuroblastoma. The boy also showed postnatal growth deficiency, mental retardation, fine motor dysfunction, short palpable fissures, pronounced facial hirsutism, metopic ridging, ocular hypertelorism, and a broad nasal bridge. Two other cases of FHS associated with neuroblastoma were reported in 1976. These three cases should establish the relationship between FHS and the development of neural crest tumors, since three such cases should occur by chance only once in 60 yr. The association with tumor has been recognized only in children with the dysmorphic features of FHS. It is not known whether there is an increased risk of malignancy in exposed children without dysmorphic features. (9 refs)

79-2498 Response of the Rat to Saccharin with Particular Reference to the Urinary Bladder. (Eng) Chowanec, J. (Sch. Pathology, Middlesex Hosp. Medical Sch., London, England); Hicks, R. M. *Br J Cancer* 39(4): 355-375; 1979.

Male and female Wistar rats were administered sodium saccharin for life (2 yr), either in their drinking water or diet. The max palatable saccharin dose in the drinking water was 2 g/kg/day and, even then, there was some voluntary restriction of fluid intake in the males. By contrast, 4 g/kg/day was palatable in the diet. Control rats of both sexes received saccharin-free diets and drinking water. Mild urothelial hyperplasias developed by 85 wk in rats of both sexes receiving saccharin in drinking water or diet; the incidence was statistically significant in both the bladders and kidneys of rats receiving the higher dose in the diet, but only in the kidneys of rats receiving the lower dose in the drinking water. Telangiectasia of the *vasa recta* was significant in saccharin-treated rats of both sexes at both doses. Of the three bladder tumors that were seen at 95 or 102 wk, all were in males receiving the higher saccharin dose in the diet. No consistent relationship between bladder epithelial hyperplasias and crystalluria could be demonstrated, although all three bladder tumors were associated with some form of mineralization. The results suggest a particular susceptibility of males to saccharin

treatment. However, rather than initiating carcinogenesis, saccharin may promote or enhance the development of latent tumor cells. (78 refs)

79-2499 Dissociation of Mutagenic from Chemotherapeutic Activities of Drugs (Meeting Abstract). (Eng) Batzinger, R. P. (Johns Hopkins Univ., Baltimore, MD) *Diss Abstr Int [B]* 39(10): 4838; 1979 (2 refs)

79-2500 Concentration and Separation of Arsenic from Polluted Water by Ion Exchange. (Eng) Sandhu, S. S. (Claflin Coll., Orangeburg, SC 29115); Nelson, P. *Environ Sci Technol* 13(4): 476-478; 1979.

A technique for the concentration and separation of arsenic from polluted environmental samples, which contain metal ions reported to interfere with As determination by the silver diethyldithiocarbamate (SDDC) method, is described. Aqueous samples are digested with potassium permanganate and eluted through chromatograph columns packed with Amberlite ion-exchange resin. As retained by the resin is extracted with HCl prior to its determination by the SDDC method. The technique facilitates the quantitative determination of As at levels below 0.005 mg/liter. (18 refs)

79-2501 Concentration and Analysis of Polycyclic Aromatic Hydrocarbons in Water. (Ger) Faltusz, E. (Staatliche Milchwirtschaftliche Lehr- und Forschungsanstalt, Am Maierhof 7, D-7988 Wangen im Allgau, W. Germany) *Fresenius Z Anal Chem* 294(5): 385-390; 1979.

A simple and rapid adsorption method for the concentration and analysis of polycyclic aromatic hydrocarbons (PAH) in water is described. The magnesium salt added to the sample is precipitated, the precipitate is dissolved by ammonium chloride, and the organic substances are extracted with a very small volume of cyclohexane. The PAH are then separated gas chromatographically with the use of an electron-capture detector. (20 refs)

79-2502 Gas-Liquid Chromatographic-Mass Fragmentographic Determination of Low Levels of Diethylcarbonate in Beverages. (Eng) van Lierop, B. H. (Food Inspection Service, Nijenoord 6, Utrecht, Netherlands); Nootenboom, H. *J Assoc Off Anal Chem* 62(2): 253-256; 1979.

Combined gas-liquid chromatography-mass fragmen-

tography was used to determine the levels of diethyl carbonate, an impurity present in diethyl pyrocarbonate (DEPC), in samples of fruit drinks, wine, and beer. DEPC is added to beverages to prevent fermentation. Sixteen of 138 samples had >30 ppb diethyl carbonate. (5 refs)

- 79-2503 Comparison and Evaluation of Some Experimental Designs for Use in Carcinogen Screening.** (Eng) Elashoff, R. M. (Dept. Biomathematics, Sch. Medicine, Univ. California, Los Angeles, CA 90024); Preston, D. L.; Fears, T. R. *J Natl Cancer Inst* 62(5): 1209-1219; 1979.

The development and evaluation of experimental designs for routine in vivo screening of chemicals for potential carcinogenicity are discussed. The current one-stage 50-animal/group screen used by NCI is described, and a two-stage alternative, which would use 35 animals/group is proposed. This alternative allows for retesting of equivocal compounds. The proposed designs were evaluated in terms of sensitivity, specificity, and decision-making using simulated data on 2,000 compounds. Despite the large number of tests made for each compound, the false-positive rate was <0.07 for the current one-stage screen and <0.05 for the proposed two-stage alternative. The two-stage screen, however, would permit 30% more decisions per test period with a savings of 28% in the expected number of animals needed per compound tested. (6 refs)

- 79-2504 The Relationship Between Mutagenic and DNA-Damaging Activity of Pesticides and Their Potential for Carcinogenesis (Meeting Abstract).** (Eng) Rashid, K. A. (Pennsylvania State Univ., University Park, PA) *Diss Abstr Int [B]* 39(10): 4726; 1979 (no refs)

- 79-2505 The Use of Adult Hamster Hepatocytes and Hamster Embryo Cells to Metabolize Carcinogens in the Ames Mutagenesis Assay (Meeting Abstract).** (Eng) Raineri, R. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Poiley, J. A.; Pienta, R. J.; Andrews, A. W. *In Vitro* 15(3): 209; 1979 (no refs)

- 79-2506 Genetic Differences in Metabolism of Polycyclic Aromatic Carcinogens and Aromatic Amines by Mouse Liver Microsomes. Detection by DNA Binding of Metabolites and by Mutagenicity in Histidine-dependent *Salmonella typhimurium* In Vitro.** (Eng) Levitt, R. C. (Developmental Pharmacology Branch, Natl. Inst. Child Health and Human Development, NIH,

Bethesda, MD 20014); Pelkonen, O.; Okey, A. B.; Nebert, D. W. *J Natl Cancer Inst* 62(4): 947-955; 1979.

Genetic differences in the DNA binding of carcinogenic (or noncarcinogenic) metabolites in vitro were compared with genetic differences in the mutagenicity of these metabolites in the *Salmonella typhimurium* assay. The metabolism of various carcinogens and other chemicals by genetically regulated increases in cytochrome P₁-450 levels was studied with the use of liver microsomes from 3-methylcholanthrene (3-MC)-treated C57BL/6N and DBA/2N mice. Genetic differences were seen in the DNA binding of metabolites in vitro for dibenz(a,h)anthracene, benz(a)anthracene, benzo(a)pyrene (BP), 3-MC, 7,12-dimethylbenz(a)anthracene (DMBA), 2-acetylaminofluorene, and dopamine (approx in descending order). Any difference for dibenz(a,c)anthracene or ben-zidine was very small. Marked genetic differences were observed in the in vitro mutagenicity assay (with tester strains TA1538, TA98, or TA100) for 6-aminochrysene, dibenz(a,h)anthracene, dibenz(a,c)anthracene, β -naphthylamine, 3-MC, 2-acetylaminofluorene, BP, benz(a)anthracene, and α -naphthylamine (approx in descending order); the difference for DMBA was very small. Therefore, important discrepancies for certain carcinogens existed between genetic differences in metabolites binding to DNA and genetic differences in metabolites causing mutation in the bacterial assay. The data may be useful for future studies with any of these chemicals in the extrapolation of genetic differences observed in vitro to differences in the risk of cancer associated with the *Ah* locus in the intact animal. (63 refs)

- 79-2507 In Vitro Mutagenicity Assays of Chemical Carcinogens and Related Compounds with *Salmonella typhimurium*.** (Eng) Simmon, V. F. (Dept. Toxicology, SRI International, Menlo Park, CA 94025) *J Natl Cancer Inst* 62(4): 893-899; 1979.

The mutagenicity of 101 chemical carcinogens and non-carcinogens of various classes was determined in the *Salmonella typhimurium*/microsome assay system with the use of tester strains TA1535, TA1536, TA1537, TA1538, TA98, and TA100. Assays were conducted in the presence and absence of a metabolic activation system prepared from the livers of randomly bred Sprague-Dawley rats that had been pretreated with Aroclor 1254. The test chemicals were incorporated into the agar with the bacteria and the metabolic activation system. Mutagens were defined as chemicals that induced a reproducible dose-related increase in the number of histidine-independent revertants. With the use of these procedures, 65% of the organic carcinogens and 25% of the noncarcinogens were found to be mutagenic. The aromatic amines, polycyclic aromatic hydrocarbons, and alkylating agents had the highest correlation between mutagenicity and carcinogenicity. The nitrosamines and the miscellaneous chemicals had the poorest correlation. (16 refs)

79-2508 Energy-related Pollutants in the Environment: Use of Short-Term Tests for Mutagenicity in the Isolation and Identification of Biohazards. (Eng) Epler, J. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Larimer, F. W.; Rao, T. K.; Nix, C. E.; Ho, T. *Environ Health Perspect* 27: 11-20; 1978.

To investigate the potential genetic (and carcinogenic) hazards associated with the developing synthetic fuel technologies, a coupled chemical and biological analysis was made of the products, process streams, and effluents of existing or proposed energy-generating or -conversion systems. One phase of the study deals with known compounds expected to occur in the environment through energy production, conversion, or use; another phase deals with actual samples from existing or experimental processes. To approach the problems inherent in testing large numbers of compounds, a tier system of mutagenicity tests was set up. The *Salmonella* histidine-reversion system is used in the first tier; the standard test for sex-linked recessive lethals in *Drosophila* is used in the second tier; and the specific locus test in the mouse is used in the third tier. The tiered system compared with alternative mutagenicity tests. Data obtained when a group of substituted nitroso compounds were assayed in *Salmonella*, yeast, and *Drosophila* systems are presented. All systems showed a high correlation of mutagenic response with carcinogenicity. The feasibility of applying mutagenicity testing to environmental effluents and crude products from the synthetic fuels technology was tested by screening a condensate sample from an experimental coal gasification process in the *Salmonella* test. The results showed that biological testing can be carried out, but perhaps only with the appropriate analytical separation scheme. The results of comparative studies in other test systems generally validated the results of the initial screening carried out in the *Salmonella* assay. Although the applicability of the *Salmonella* assay results to other genetic assays needs further validation, short-term tests appear to be useful as prescreening devices in testing large numbers of hazardous environmental compounds and complex mixtures. (41 refs)

79-2509 Selection of Carcinogens and Related Compounds Tested for Mutagenic Activity. (Eng) Poirier, L. A. (Lab. Carcinogen Metabolism, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014); Weisburger, E. K. *J Natl Cancer Inst* 62(4): 833-840; 1979.

The rationale for selecting 102 chemical carcinogens and noncarcinogens to be tested for mutagenicity in a National Cancer Institute program to determine the extent of correlation between carcinogenesis and mutagenesis in standard assays is given. The chemicals were divided into five major categories: 37 aromatic amines, 11 polycyclic aromatic hydrocarbons, 8 nitrosamines and nitrosamides, 16 alkylating agents, and a miscellaneous category consisting of 11 heterocyclic compounds, 7 amines, ureas, and

acylating agents, 5 antimetabolites, 4 inorganic chemicals, and 3 promoters. The chemicals were further classified as procarcinogens (requiring metabolic activation to exert their biologic activities), ultimate carcinogens (direct-acting chemicals not requiring metabolic activation), and noncarcinogens (compounds shown to be inactive in one or more adequate carcinogenicity tests). The selection and categorization of the compounds are documented by an extensive bibliography. (168 refs)

79-2510 In Vitro Assays for Recombinogenic Activity of Chemical Carcinogens and Related Compounds with *Saccharomyces cerevisiae* D3. (Eng) Simmon, V. F. (Dept. Toxicology, SRI International, Menlo Park, CA, 94025). *J Natl Cancer Inst* 62(4): 901-909; 1979.

The utility of *Saccharomyces cerevisiae* D3 as a prescreen for a broad spectrum of potential carcinogens was tested with the use of a standard protocol that was maintained throughout the experiments. A total of 101 carcinogens, noncarcinogens, metals, and promoters representing a wide variety of chemical classes was tested to determine whether they increased mitotic recombination in *S. cerevisiae* D3. A metabolic activation system prepared from homogenates of livers from rats that had been pretreated with Aroclor 1254 (a mixture of polychlorinated biphenyls) was incorporated in the assay. All of the 20 ultimate carcinogens and 18/48 procarcinogens increased mitotic recombination. Of the noncarcinogens, 6/21 also increased mitotic recombination. An improved metabolic activation procedure appeared to be required to increase the probability of detecting procarcinogens by this method. The carcinogens thioacetamide, natulan, auramine, safrole, and 1'-hydroxysafrole increased mitotic recombination in *S. cerevisiae* D3 (the last compound was marginally positive), but they were negative in assays with *Salmonella typhimurium*. (22 refs)

79-2511 Mutagenic Activity of Chemical Carcinogens and Related Compounds in the Intraperitoneal Host-mediated Assay. (Eng) Simmon, V. F. (Life Sciences Div., Dept. Toxicology, SRI International, Menlo Park, CA, 94025); Rosenkranz, H. S.; Zeiger, E.; Poirier, L. A. *J Natl Cancer Inst* 62(4): 911-918; 1979.

The mutagenicities of 79 carcinogens, noncarcinogens, and structurally related compounds toward *Salmonella typhimurium* strains TA1530, TA1535, and TA1538 and toward *Saccharomyces cerevisiae* D3 were investigated in the ip host-mediated assay. Fewer than half of the carcinogens were mutagenic toward the *Salmonella* strains. The insensitivity of the system was most marked with the aromatic amine and polycyclic hydrocarbon procarcinogens. Under the test conditions, < 10% of the carcinogens showed clear mutagenic activity toward *S. cerevisiae* D3. However, none of the noncarcinogens was significantly mutagenic toward

either *S. typhimurium* or *S. cerevisiae* D3. Overall, the ip host-mediated assay does not seem suitable for routine preliminary screening of large numbers of potential carcinogens. The median lethal doses to mice of 46 compounds were determined. (23 refs)

79-2512 Evaluation of the Mutagenicity and DNA-modifying Activity of Carcinogens and Noncarcinogens in Microbial Systems. (Eng) Rosenkranz, H. S. (Dept. Microbiology, New York Medical Coll., Valhalla, NY, 10595); Poirier, L. A. *J Natl Cancer Inst* 62(4): 873-892; 1979.

The predictive value of two short-term microbial assays in identifying potential carcinogens was evaluated with the use of 99 chemical carcinogens and noncarcinogens. The mutagenicity of the chemicals was determined in a standard *Salmonella typhimurium* (ST) assay with strains TA1535 and TA1538; their DNA-modifying capacity was determined with normal and DNA polymerase-deficient *Escherichia coli* (EC) strains. The chemicals consisted of: alkylating agents (15); nitrosamines, hydrazines, and related substances (8); heterocyclics (10); aromatic amines (36); polycyclic aromatic hydrocarbons (11); amides, ureas, and acylating agents (7); antimetabolites (5); inorganics (4); and promoters (3). Twenty-one compounds were known noncarcinogens, 21 were ultimate carcinogens, and 45 were procarcinogens. Of the noncarcinogens, 35% were positive in the ST system, 30% were positive in the EC system, and 25% were positive in both systems. Of the ultimate carcinogens, 100% were mutagenic in EC, 79% in ST, and 79% in both systems. Of the procarcinogens, 52% and 67% were positive in the ST and EC assays, respectively, and 48% were positive in both systems. A tabulation of the combined data for ultimate carcinogens and procarcinogens indicated that 77% of the compounds were mutagenic: 61% and 74% in the ST and EC assays, respectively, and 59% in both assays. For prescreening procedures with microbial assays, it is suggested that ST strains TA98 and TA100 be included and that the standard EC DNA polymerase-deficient assay be run in tandem with the ST mutagenicity assay. When the standard EC DNA polymerase-deficient assay does not give interpretable results because of the lack of growth inhibition zones, a modified assay with the use of liquid suspension should be performed. (59 refs)

79-2513 Initial National Cancer Institute Studies on Mutagenesis as a Prescreen for Chemical Carcinogens: An Appraisal. (Eng) Poirier, L. A. (Nutrition and Metabolism Section, Lab. Carcinogen Metabolism, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD, 20014); de Serres, F. J. *J Natl Cancer Inst* 62(4): 919-926; 1979.

A National Cancer Institute (NCI) collaborative study of mutagenesis as a prescreen for chemical carcinogens was evaluated. The initial objectives were to develop a list of carcinogens and related compounds that would be tested and to select mutagenic systems offering the greatest promise of discriminating between carcinogens and noncarcinogens of widely varying chemical structures. The efficacy of several in vitro and in vivo assays to detect carcinogens from a list of 102 compounds was determined. *Salmonella typhimurium* and polymerase A-deficient *Escherichia coli* in vitro were the most effective systems studied. Together, they detected 82% of the organic carcinogens tested. Potential prescreening systems that were thought to be currently insufficiently sensitive for the routine screening of potential carcinogens included (1) the development of resistance to thymidine overloading, methotrexate, or cytosine arabinoside by L5178y mouse lymphoma cells; (2) genetic lesions (eg, mitotic recombination in *Saccharomyces cerevisiae* D3; (3) ip host-mediated assays using *S. typhimurium*, *S. cerevisiae* and L5178y cells; and (4) thymidine uptake as a reflection of DNA repair. The present evaluation of the NCI collaborative study has revealed a correlation between carcinogenic and mutagenic activity. (39 refs)

79-2514 Production of a Mutagen from Ponceau 3R by a Human Intestinal Anaerobe. (Eng) Hartman, C. P. (Bethesda Res. Labs., Rockville, MD, 20850); Andrews, A. W.; Chung, K. T. *Infect Immun* 23(3): 686-689; 1979.

The metabolic reduction of the azo dye Ponceau 3R by the human intestinal bacteria *Fusobacterium* sp. 2 was studied. The reaction was characterized by a lag period of 30 min followed by a 96% reduction within 90 min and 100% reduction within 3 hr. One of the metabolites obtained was 2,4,5-trimethylaniline (41.9%), which was mutagenic in the Ames *Salmonella* mammalian microsome mutagenicity assay when activated by rat liver S9 preparations. Also obtained in large quantity was 2,4-dimethylaniline (38.4%), indicating that a substantial part of Ponceau 3R is the closely related dye Ponceau 2R, which differs in structure from Ponceau 3R in that it lacks a ring methyl group. Although 2,4-dimethylaniline is mutagenic in the Ames assay in quantities > 250 µg, such large quantities were not generated under the conditions studied. None of the other dimethylanilines isolated from the extract of the reduced dye showed any mutagenicity in quantities up to 1,000 µg. (9 refs)

79-2515 In Vitro Metabolic Activation of Chemical Carcinogens to Mutagens and Its Relationship to Cancer Induction in Vivo (Meeting Abstract). (Eng) Brusick, D. J. (Litton Bionetics, Inc., 5516 Nicholson Lane, Kensington, MD, 20795). *In Vitro* 15(3): 208; 1979. (no refs)

79-2516 Aryl-Monoalkyl and Cyclic Triazenes: Direct-acting Mutagens. (Eng) Thomas, H. F. (Dept. Medical Genetics, Univ. Wisconsin, Madison, WI, 53706); Brown, D. L.; Hartman, P. E.; White, E. H.; Hartman, Z. *Mutat Res* 60(1): 25-32; 1979.

The mutagenicity of a linear aryl-monoalkyl triazene, methyl-p-tolyl triazene (MTT), and a cyclic triazene, δ^2 -triazoline, for *Salmonella typhimurium* and for *Hemophilus influenzae* cell-free DNA was tested. Both triazenes were direct-acting mutagens. MTT caused reversion of the *hisG46* base-substitution mutation but not of the *hisD3052* frameshift mutation. The strains carrying the *hisG46* mutation were equally sensitive to mutagenesis, independent of genetic background. Mutation frequency induced by MTT was strongly enhanced by liquid preincubation before plating. It was decreased by the presence of caffeine in the plating medium and increased by a pool of common L-amino acids. This is the first report of mutagenicity by a cyclic triazene. (33 refs)

79-2517 The Mutagenic Action of Nitroimidazoles. IV. A Comparison of the Mutagenic Action of Several Nitroimidazoles and Some Imidazoles. (Eng) Voogd, C. E. (Lab. Chemotherapy, Natl. Inst. Public Health, P.O. Box 1, Bilthoven, The Netherlands); Van Der Stel, J. J.; Jacobs, J. J. *Mutat Res* 66(3): 207-221; 1979.

The mutagenicity of 51 imidazoles was determined in fluctuation tests with *Klebsiella pneumoniae* as the test organism and in the Ames test with *Salmonella typhimurium* TA100 as the tester strain. Of the 33 nitroimidazoles tested, 31 were mutagenic for *K. pneumoniae*, whereas only 2/18 imidazoles without a nitro group were mutagenic. All eight nitroimidazoles tested in the Ames assay were mutagenic for *S. typhimurium*. Regarding the methylnitroimidazoles, mutagenic activity was increased when a methyl group was present on the atom next to that with a nitro group in the imidazole nucleus. Mutagenic activity was decreased when there was an unsubstituted atom between the atoms with the methyl and nitro groups. For molecules with a more complex chemical structure, no clear relationship existed between chemical structure and mutagenicity. Also, there was no direct relationship between antimicrobial action, growth inhibition, and mutagenicity. The possible existence of nitroimidazole derivatives with strong antimicrobial but weak mutagenic actions cannot be excluded. (27 refs)

79-2518 Comparison of the Genetic Activity of 5-Nitroimidazole Derivatives in *Escherichia coli*, *Neurospora crassa*, *Saccharomyces cerevisiae* and *Drosophila melanogaster*. (Eng) Mohn, G. R. (Environmental Mutagenesis Branch, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709); Ong, T. M.; Callen, D. F.; Kramers, P. G.; Aaron, C. S. *J Environ Pathol Toxicol* 2(3): 657-670; 1979.

Four 5-nitroimidazoles, including metronidazole [MNZ: 1-(2-hydroxyethyl)-2-methyl-5-nitroimidazole], 6-(2-dimethylamino-ethoxy)-2-(5-nitro-1-methyl-2-imidazolylmethylene)-1-tetralone sulfate (compound 2), 3-(5-nitro-1-methyl-2-imidazolyl)methyleneamino-5-morpholinomethyl-2-oxazolidone hydrochloride (compound 3), and 1-(3-morpholinopropyl)-2-(5-nitro-1-methyl-2-imidazolyl)benzimidazole (compound 4), were tested for mutagenic activity in several assay systems. MNZ was moderately mutagenic toward *Escherichia coli*. Compounds 2 and 3 were strongly mutagenic even at concentrations that did not inactivate the colony-forming ability of the cells. Compound 4 showed no effect toward *E. coli*. Similar results were obtained with *Neurospora crassa*, *Saccharomyces cerevisiae*, and *Drosophila melanogaster*, with compound 2 exhibiting a higher effect than compound 3. These biological effects were explained partly on the basis of differences in chemical structure. The findings indicate that conjugation of an unsaturated center with the 5-nitro grouping via the carbon at position 2 enhances mutagenicity and that attachment of an aromatic ring directly to position 2 of the 5-nitroimidazole nucleus reduces the mutagenic potential. (29 refs)

79-2519 Isolation and Identification of the Metabolite of N-(5-Nitro-2-furfurylidene)-3-amino-2-oxazolidone (Furazolidone). (Eng) Tatsumi, K. (Faculty Pharmaceutical Sciences, Kyushu Univ., 3-1-1 Maidashi, Higashi-ku, Fukuoka, 812, Japan); Ou, T.; Yamada, H.; Yoshimura, H.; Koga, H.; Horiuchi, T. *J Pharmacobio Dyn* 1(4): 256-261; 1978.

The in vitro and in vivo metabolites of furazolidone [N-(5-nitro-2-furfurylidene)-3-amino-2-oxazolidone] were isolated, and the mutagenicities of one metabolite and the parent compound for *Salmonella typhimurium* strain TA100 were determined. Incubation of furazolidone with milk xanthine oxidase yielded two reduction products. The first was identified as 3-(4-cyano-2-oxobutylideneamino)-2-oxazolidone (COO), and the structure of the second was not identified. COO was also isolated from the urine of male rabbits given a single 100-mg/kg dose of furazolidone po. Furazolidone was highly mutagenic for *S. typhimurium*, whereas COO was nonmutagenic. The data suggest that COO is not an active metabolite of furazolidone. (26 refs)

79-2520 Electrochemical Properties of Polycyclic Compounds Studied by the Polarographic Method in Anhydrous Systems. VI. The Influence of Proton-Donor on Reduction of Carcinogenic Aromatic Hydrocarbons in Anhydrous Environment. (Eng) Vachalkova, A. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia); Podany, V.; Bahna, L. *Neoplasma* 25(6): 679-684; 1978.

The electrochemical behavior of a series of carcinogenic and

noncarcinogenic polycyclic aromatic hydrocarbons (PAH) was studied in an anhydrous environment (in dimethylformamide or dimethyl sulfoxide) in the presence of a proton donor (phenol). Based on changes in polarographic behavior induced by phenol, the PAH were divided into three groups. The Group 1 PAH were noncarcinogenic or only marginally carcinogenic. They responded to increasing phenol concentrations with higher diffuse current values for the first and second polarographic waves; the values of the half-wave potentials remained practically unchanged. Group 2 was characterized by high carcinogenicity. With increasing phenol concentration, a diffuse and irreversible new wave appeared between the two original waves of these compounds. The new wave increased at the expense of the second original wave. Group 3 contained only 1,2,5,6-dibenz(a)anthracene, which shows relatively high carcinogenicity. With the addition of phenol to this compound, the original second wave increased at the expense of the third wave until the latter disappeared at the half-wave potential value of the original second wave. This new polarographic wave was of a diffuse and irreversible character. Thus, the polarographic method described can be used to distinguish carcinogenic from noncarcinogenic PAH. (7 refs)

- 79-2521 Tobacco-specific Nitrosamines: Occurrence, Formation, Carcinogenicity, and Metabolism.** (Eng) Hecht, S. S. (Div. Environmental Carcinogenesis, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595); Chen, C. H.; Hoffmann, D. *Acc Chem Res* 12(3): 92-98; 1979.

The occurrence, formation, carcinogenicity, and metabolic activation of tobacco-specific nitrosamines are reviewed. Both N'-nitrosanornicotine (NNN) and 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK), but not 4-(N-methyl-N-nitrosamino)-4-(3-pyridyl)butanal (NNA), have been detected in tobacco and tobacco smoke. NNN has been proved to be carcinogenic in mice, rats, and hamsters. In a comparison bioassay in strain A mice, NNK was more carcinogenic than NNN, but NNA did not show significant activity. Nitrosoanabasine was somewhat carcinogenic in rats and noncarcinogenic in hamsters. In vitro and in vivo experiments demonstrated that both NNN and N-nitrosopyrrolidine undergo metabolic α -hydroxylation in the rat, and it is likely that α -hydroxylation is the critical step in the metabolic activation of these compounds. Since NNN and NNK are derived predominantly from nicotine by nitrosation during the curing and smoking of tobacco, human exposure might be reduced by appropriate management of the curing processes. Enzymes mediating the metabolic activation and detoxification of tobacco-specific nitrosamines might be induced or inhibited by environmental modifiers. (70 refs)

- 79-2522 The Fate of Intratracheally Instilled ^3H -Styrene Oxide in the Isolated Perfused Rat Lungs.**

(Eng) Uotila, P. (Dept. Physiology, Univ. Turku, SF-20520 Turku 52, Finland). *Res Commun Chem Pathol Pharmacol* 23(3): 561-573; 1979.

The metabolism and covalent binding of intratracheally instilled ^3H -styrene oxide (100 nanomoles) in the isolated perfused lungs of control and cigarette smoke-exposed male Wistar rats were investigated. After the instillation, the lungs were perfused for 16 min and the nonrecirculating perfusate was collected in two fractions: 0-8 min and 8-16 min. About 40% of the instilled dose was in the 0- to 8-min perfusion medium, mainly in the form of unmetabolized styrene oxide. However, the radioactivity in the 8- to 16-min perfusion medium and lung tissue itself was mainly in the form of water-soluble metabolites, probably glutathione conjugates. The amount of styrene glycol was very small. Exposure of the rats to cigarette smoke had no effect on the metabolism of styrene oxide or on the amount of the compound that was covalently bound to the protein and nucleic acid fractions of the perfused lungs. (26 refs)

- 79-2523 Comparison of the Induction by Cigarette Smoke Condensates of Sister-Chromatid Exchanges in Chinese Hamster Cells and of Mutations in *Salmonella typhimurium*.** (Eng) De Raat, W. K. (Div. Technology, Society TNO, P.O. Box 217, Delft, Netherlands). *Mutat Res* 66(3): 253-259; 1979.

Cigarette smoke condensates (CSC's) prepared from three European brands of cigarette were tested for the induction of sister chromatid exchanges (SCE's) in Chinese hamster ovary cells and for mutations in *Salmonella typhimurium* TA98 and TA100. Brand A (low-tar brand) produced 21 mg CSC/3 cigarettes, brand B 55 mg/3 cigarettes, and brand C 69 mg/3 cigarettes. Rat liver microsomal (S9) fractions were prepared from rats pretreated with Aroclor 1254, phenobarbital, or 3-methylcholanthrene or from untreated rats. The results of the SCE tests showed that an SCE-inducing compound was present in the CSC's. None of the four metabolic activation systems enhanced the amount of SCE-inducing compound. The three CSC's did not differ much in their capacity to induce SCE. In the *Salmonella* test, revertants were produced only when S9 fractions from rats treated with inducers of the enzyme systems necessary for metabolic activation were added. TA98 was much more sensitive than TA100. Apparently, the SCE and *Salmonella* tests are sensitive to different components of CSC. (13 refs)

- 79-2524 Liver and Lung Cell-mediated Mutagenesis of V79 Cells with Chemical Carcinogens (Meeting Abstract).** (Eng) Langenbach, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE, 68105); Tompa, A.; Kuszynski, C.; Kennedy, S.; Huberman, E. *In Vitro* 15(3): 209; 1979. (no refs)

79-2525 Application of In Vitro Transformation and Metabolism to the Study of Cocarcinogenesis (Meeting Abstract). (Eng) Nesnow, S. (US Environmental Protection Agency, Research Triangle Park, NC, 27711); Leavitt, S. *In Vitro* 15(3): 208; 1979. (no refs)

79-2526 Effects of Caffeine on Sister-Chromatid Exchanges (SCE) In Vivo. (Eng) Basler, A. (Dept. Human Genetics and Anthropology, Universitätsstr. 1, Geb 23.12, D-4000 Dusseldorf 1, W. Germany); Bachmann, U.; Roszinsky-Kocher, G.; Rohrborn, G. *Mutat Res* 59(2): 209-214; 1979.

Chinese hamsters were treated with caffeine (2 doses of 20, 100, 200, or 400 mg/kg given 24 hr apart, by stomach tube) and/or benzo(a)pyrene (BP: 2 doses of 450 mg/kg ip) or cyclophosphamide (CP: 2 doses of 5 mg/kg ip) and the effects of these agents on the frequency of sister chromatid exchanges (SCE's) and chromosome aberrations in bone marrow cells was determined. The SCE frequency increased with increasing dose of caffeine up to 2 x 200 mg/kg, which resulted in a mean number of 6.20 SCE's/metaphase; the mean number of SCE's/metaphase was 10.57 after BP and 12.35 after CP. Simultaneous po treatment with caffeine reduced the frequency of SCE's induced by CP or BP. Nonsignificant increases in metaphases with structural chromosome aberrations were induced by BP or caffeine; simultaneous administration of the two agents caused a synergistic effect. (18 refs)

79-2527 Bioassay Procedure for the Detection of Mutagenic Metabolites in Human Urine with the Use of Sister Chromatid Exchange Analysis. (Eng) Guerrero, R. R. (Pasadena Foundation Medical Res., 99 N. El Molino Ave., Pasadena, CA, 91101); Rounds, D. E.; Hall, T. C. *J Natl Cancer Inst* 62(4): 805-809; 1979.

A sensitive, reproducible, short-term bioassay system for the detection of activated mutagenic metabolites in urine from humans exposed to promutagens is described. Human diploid fibroblasts were grown in medium containing 5%-20% urine from 8 smokers, 7 nonsmokers, and 2 individuals undergoing cyclophosphamide (CP) chemotherapy for cancer (5.0 and 7.5 mg/kg/wk iv, respectively). The cells were then subjected to sister chromatid exchange (SCE) analysis. Activated CP metabolic products in urine specimens produced up to a ten-fold increase in SCE's over preinjection SCE levels for the same individuals. Linear dose-response curves over a 5%-20% urine concentration range were obtained with cells grown in urine specimens from the nonsmokers and the cigarette smokers. This test system proved to be sensitive to ambient exposure levels of environmental mutagens, and it demonstrated that urine from smokers was significantly more mutagenic than was urine from nonsmokers. Replicate experiments showed highly reproducible SCE values for each individual as well as for av SCE values for each group of

subjects. The ability of this bioassay system to detect trace mutagenic activity in human urine reproducibly makes it an attractive choice for the monitoring of humans who have been exposed to environmental and/or industrial mutagens. (30 refs)

79-2528 Accessibility of Deoxyribonucleic Acid in Chromatin to the Covalent Binding of the Chemical Carcinogen Benzo(a)pyrene. (Eng) Jahn, C. L. (Dept. Bacteriology and Immunology, Univ. North Carolina at Chapel Hill, Sch. Medicine, Chapel Hill, NC); Litman, G. W. *Biochemistry* 18(8): 1442-1449; 1979.

The role of chromatin structure in the binding of benzo(a)pyrene (BP) to DNA was investigated. A model system utilizing rat liver microsomes to activate [³H]BP in the presence of calf thymus nuclei was used to examine the ability of BP to bind regions of DNA that differ in their accessibility in chromatin. [³H]BP-modified nuclei were digested with staphylococcal nuclease and DNase I, and the specific activity (counts/minute of [³H]BP/absorbance at 260 nanometers of DNA) of the DNA remaining undigested was determined. Both enzymes resulted in characteristic changes in specific activity as a function of digestion. No changes occurred during digestion of isolated [³H]BP-DNA, and BP had no effect on the kinetics of digestion of DNA or nuclei, indicating that the specific activity changes seen in nuclear digests were due to preferential binding to DNA in regions of chromatin differing in enzyme susceptibility. The nucleosomal sites of [³H]BP binding were determined by electrophoretic analysis of the resistant DNA and by examining the specific activity as a function of digestion of (1) nucleosome multimers isolated by sucrose gradient sedimentation of [³H]BP-modified nuclei partially digested with staphylococcal nuclease and of (2) monomer subfractions obtained by KCl precipitation of H1-containing monomers. In addition, the distribution of [³H]BP in fragments obtained from a DNase I digest of nuclei was compared with that of an isolated monomer fraction. These data led to the conclusion that BP binds to the spacer region and the outermost portions of the nucleosome core. (41 refs)

79-2529 Analysis of Prehistoric Levels of Benzo(a)pyrene in Permafrost Soil. (Rus) Il'nitskii, A. P. (Cancer Res. Center, Moscow, USSR); Vinogradov, V. N.; Riabchun, V. K.; Mishchenko, V. S.; Gvil'dis, V. Iu.; Chernen'kii, B. I.; Belitskii, G. A.; Shabad, L. M. *Dokl Akad Nauk SSSR* 245(1): 254-257; 1979.

In an attempt to estimate the natural level of carcinogenic polycyclic aromatic hydrocarbon (PAH) during prehistoric geological periods, levels of benzo(a)pyrene (BP) were analyzed in samples of permafrost soil. Spectrofluorometric assay of soil samples taken at depths ranging from 0.06-0.16 m (active layer) to 0.80-0.90 m ($\geq 10,000$ yr old) confirmed the presence of BP in all layers of permafrost. The natural

level of BP ranged from 1 to 5 $\mu\text{g/kg}$ of dry soil. This level was comparable to the current background level of PAH and, thus, could be due to the existence of natural sources of these compounds. (9 refs)

- 79-2530 Binding of Benzo(a)pyrene into Lung and Thymocyte Nuclear Fractions.** (Eng) Blazsek, I. (Dept. Medical Chemistry, Univ. Helsinki, Siltavuorenpenger 10 A, 00170 Helsinki 17, Finland); Vauhkonen, M.; Hemminki, K. *Res Commun Chem Pathol Pharmacol* 23(3): 611-626; 1979.

The in vitro binding of ^3H -benzo(a)pyrene to rat lung cells and thymocytes was studied. In most lung cell fractions, the specific radioactivity of protein exceeded that of RNA and DNA, but in the chromatin fraction, RNA was most actively labeled. The labeling of DNA in the matrix fraction was threefold that in the chromatin fraction, and the matrix sediment proteins were more actively labeled than the proteins of other fractions. A high level of binding to RNA was observed in the thymocytes. Again, binding to protein was greater in the matrix fraction than in the other fractions. Nonhistones were more actively labeled than histones, the difference being 30-fold in the lung fractions and 10-fold in the thymocyte fractions. Highly radioactive peaks were associated with matrix sediment proteins with approx mol wts of 55,000 and 130,000 daltons. (29 refs)

- 79-2531 Metabolism of Benzo(a)pyrene to Reactive Intermediate(s) via Prostaglandin Biosynthesis.** (Eng) Sivarajah, K. (Lab. Pulmonary Function and Toxicology, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC, 27709); Anderson, M. W.; Eling, T. E. *Life Sci* 23(26): 2571-2578; 1978.

The oxidation of benzo(a)pyrene (BP) and other polycyclic aromatic hydrocarbons to electrophilic metabolites during the formation of prostaglandins (PG) and thromboxanes from arachidonic acid (AA) by guinea pig lung microsomal protein was examined. In the presence of NADPH or AA, electrophilic metabolites of [^{14}C]-BP were generated that were nonextractable from microsomal protein and thus assumed to be covalently bound. The total amount of BP metabolized in the presence of NADPH was 2-2.5 times the amount of BP metabolized in the presence of AA. Only 4%-5% of BP metabolized by the NADPH-mediated mixed-function oxidase system was covalently bound, whereas 12%-15% of the BP metabolized in the presence of AA was covalently bound to tissue protein and DNA. Quinones were the major metabolites produced by the AA-dependent system, and dihydrodiols were the major metabolites formed by the NADPH-dependent system. 7,12-Dimethylbenz(a)anthracene and 7,8-BP dihydrodiol, but not 3-hydroxy-BP, were also oxidized by PG synthetase to reactive metabolites. (27 refs)

- 79-2532 Effects of a Cytochrome P-450 Inhibitor on Covalent Binding of [^3H]Benzo(a)Pyrene in Nil Cell Fractions.** (Eng) Sloane, N. H. (Dept. Microbiology and Molecular Genetics, Harvard Medical Sch., Boston, MA, 02115); Chu, L. N.; Amos, H. *IRCS Med Sci (Cancer)* 7(2): 81; 1979.

1-Benzylimidazole (BI) inhibited the covalent binding of ^3H -benzo(a)pyrene to both the nuclear and cytoplasmic fractions of Nil cells. Second cycle cells initially exposed to the chemicals and subsequently grown in their absence showed a preferential concentration of BI in the nuclear fraction. (4 refs)

- 79-2533 Metabolism of Lipid Peroxides During Chemical Carcinogenesis.** (Rus) Lankin, V. Z. (Inst. Chemical Physics, Moscow, USSR); Poliakov, V. M.; Arkhangel'skaia, A. V.; Gurevich, S. M. *Biull Eksp Biol Med* 87(3): 270-273; 1979.

The metabolism of lipid peroxides during chemical carcinogenesis was studied in random-bred albino rats that were inoculated sc with benzo(a)pyrene (BP: 5 mg in 0.5 ml of olive oil). Rats were sacrificed at various times after BP injection, and phospholipid levels and enzymatic and nonenzymatic activities of phospholipid peroxidation in the liver and tumor tissue were assessed. BP caused a marked increase in the level of lecithin and cephalin in the liver microsomes and mitochondria that was max at 14 wk, the time of tumor appearance. Tumor development was associated with a drastic decrease in phospholipid levels. These levels were significantly lower in the tumor tissue than in the liver (5.0-5.5 mg/g and 25.2 mg/g, respectively). In contrast, glutathione peroxidase activity was significantly greater in the tumor (46.7 IU at 16 wk, 69.8 IU at 20 wk, and 67.4 IU at 24 wk than in the liver (37.0 IU). These findings indicate that the detoxification of phospholipid peroxides was significantly more effective in the tumor tissue than in the liver. (14 refs)

- 79-2534 A New Assay for Glutathione S-Transferase Using (^3H)-Benzo(a)pyrene 4,5-Oxide as Substrate. Inducibility by Various Chemicals in Different Rat Tissues Compared to that of Aryl Hydrocarbon Hydroxylase and Epoxide Hydratase.** (Eng) Van Cantfort, J. (Laboratoire de Chimie Medicale, Institut de Pathologie, B-4000 Sart-Tilman, Liege 1, Belgium); Manil, L.; Gielen, J. E.; Glatt, H. R.; Oesch, F. *Biochem Pharmacol* 28(4): 455-460; 1979.

A new assay for glutathione S-transferase (GST) uses ^3H -benzo(a)pyrene 4,5-oxide (^3H -BP 4,5-oxide) as a substrate. It is performed under conditions that are optimal with respect to both the pH and the concentration of the two substrates (2mM glutathione and 80 μM BP 4,5-oxide). The substrate and product are separated by a differential extraction, and the radioactivity in the aqueous fraction is counted by liquid

scintillation spectrometry. Other than a trace of ^3H -BP 4,5-oxide, the aqueous fraction contained only one labeled product that was identified as a glutathione conjugate. The assay was used to determine the effect of cigarette smoke inhalation on GST activity in liver, kidney, and lung and to compare this effect with that of the drug metabolizing enzyme inducers phenobarbital (PB), 3-methylcholanthrene (3-MC), and 16 α -cyanopregnenolone and with the effect on aryl hydrocarbon hydroxylase (AHH) and epoxide hydratase (EH). Liver AHH activity was enhanced by the three inducers, but only 3-MC and cigarette smoke significantly induced enzyme activity in lung and kidney. EH activity was induced by 16 α -cyanopregnenolone and PB in liver but not in other tissues. GST activity was also inducible, but the inductions were quantitatively weaker than those of the other two enzyme activities and were limited to the liver. These results indicate that GST is insensitive to the administration of chemicals to animals, and they suggest that the role of this enzyme in the metabolism of polycyclic hydrocarbons might be very limited. (25 refs)

79-2535 Identification of Benzo(a)pyrene Metabolites by Gas Chromatograph-Mass Spectrometer.

(Eng) Takahashi, G. (Dept. Pathology, Chest Disease Res. Inst., Kyoto Univ., Sakyo-ku 606, Kyoto, Japan); Kinoshita, K.; Hashimoto, K.; Yasuhira, K. *Cancer Res* 39(5): 1814-1818; 1979.

Combined gas chromatography-mass spectrometry was used in an attempt to separate and identify benzo(a)pyrene (BP) and 38 of its metabolites (12 phenols, 6 quinones, 6 dihydroxyl compounds, 7 diols, 4 stereoisomeric tetraols, 2 diol epoxides, and 1 epoxide). All derivatives, after being trimethylsilylated, were developed on Dexsil-300 and OV-1 columns. The 7 diols and 4 stereoisomeric 7,8,9,10-tetraols were separated successfully, but not the 12 phenols. The 8- and 11-isomers appeared separately but the other 10 isomers formed 3 peaks on the OV-1 column. The five phenols previously reported to be present in animal tissue were applied to the Dexsil-300 column. The 6-, 7-, and 9-phenols were separated, but the 1- and 3-phenols eluted together. The quinones were converted to related dihydroxyl derivatives under silylation. The chromatographic separation of 4/6 dihydroxyl derivatives was successful on the OV-1 column, but the 6,12- and 7,10-isomers formed in a single peak. The two diol epoxides were converted to 7,8,9-trihydroxy-7,8-dihydro-BP under silylation. Therefore, it was impossible to identify the diol epoxides themselves. However, the presence of stereoisomeric 7,8,9,10-tetraols and/or 7,8,9-trihydroxy-7,8-dihydro-BP strongly suggests that the test material contains diol epoxides. The mass spectra and retention times of all the BP derivatives studied are given. (26 refs)

79-2536 Formation of DNA Adducts in 10T1/2 Mouse Embryo Fibroblasts Incubated with Benzo(a)-

pyrene or Dihydrodiol Oxide Derivatives. (Eng) Brown, H. S. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia Univ., New York, NY, 10032); Jeffrey, A. M.; Weinstein, I. B. *Cancer Res* 39(5): 1673-1677; 1979.

The extent of DNA binding and the types of adducts formed when confluent cultures of the mouse embryo fibroblast cell line 10T1/2 were exposed to radioactive benzo(a)pyrene (BP) or 7,8-dihydroxy-9,10-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (BPDE) were analyzed. The cells metabolized BP to its diastereoisomeric 7,8-dihydrodiol-9,10-oxides, (\pm)-7 β ,8 α -dihydroxy-9 α , 10 α -epoxy-7,8,9,10-tetrahydro-BP (BPDE I) and (\pm)-7 β ,8 α -dihydroxy-9 β ,10 β -epoxy-7,8,9,10-tetrahydro-BP (BPDE II), which formed covalent adducts with cellular DNA. Detailed analysis by high-pressure liquid chromatography indicated that a deoxyguanosine adduct, N²-(10S-[7R,8S,9R-trihydroxy-7,8,9,10-tetrahydrobenzo(a)pyrene]yldeoxyguanosine, was the predominant DNA adduct formed in cells exposed to BP. This adduct is also the major one found in human tissues exposed to BP. Exposure of cells to BPDE I and BPDE II, rather than to BP, produced several additional DNA adducts. During a 68-hr posttreatment incubation, about 50%-70% of the adducts were removed from the cellular DNA. No major differences between the relative rates of excision from cellular DNA of the individual BPDE I and BPDE II adducts were found. These results provide evidence that when 10T1/2 cells are exposed to BP, there is stereoselective synthesis and covalent binding of BPDE mainly to the N² group of deoxyguanosine. This similarity with results obtained in human tissues makes the 10T1/2 cell an attractive model system for studying the action of BP and related polycyclic hydrocarbons. (36 refs)

79-2537 In Vitro Chemical Transformation of Fetal Mouse Brain Cells (Meeting Abstract). (Eng)

Markovits, P. (Institut Curie, 75005-Paris, France); Coulomb, B.; Levy, S.; Papadopoulos, D.; Maunoury, R. *In Vitro* 15(3): 212-213; 1979. (no refs)

79-2538 Metabolic Activation of Carcinogens by X-Irradiated and Non-Irradiated Syrian Hamster Embryo Cells (Meeting Abstract). (Eng) Dooley, J. (Dept.

Environmental Health, Univ. Cincinnati, Cincinnati, OH, 45267); Soukup, S.; Warshawsky, D.; Christian, R. *In Vitro* 15(3): 219; 1979. (no refs)

79-2539 Benzo(a)pyrene Metabolism and Binding in Type II Alveolar Cells Clonally Derived From Adult Rat Lung (Meeting Abstract). (Eng) Teel, R. W.

(Loma Linda Univ., Loma Linda, CA, 92350); Douglas, W. H. *In Vitro* 15(3): 219; 1979. (1 ref)

79-2540 Radioimmunoassay for Benzo(a)pyrene (Meeting Abstract). (Eng) Kado, Y. N. (Univ. California, Berkeley, CA). *Diss Abstr Int [B]* 39(9): 4253; 1979. (no refs)

79-2541 The Murine *Ah* Locus: In Utero Toxicity and Teratogenesis Associated with Genetic Differences in Benzo[a]pyrene Metabolism (Meeting Abstract). (Eng) Nebert, D. W. (NICHD, NIH, Bethesda, MD); Shum, S. *Pediatr Res* 13(4, part 2): 423; 1979. (no refs)

79-2542 Collagen Changes Associated with Chemical Carcinogenesis in Lung Organ Cultures (Meeting Abstract). (Eng) Hussain, M. Z. (Univ. California, San Francisco, CA, 94143); Tolentino, M.; Lee, S. D.; Belton, J. C.; Bhatnagar, R. S. *Fed Proc* 38(3, part 2): 1338; 1979. (no refs)

79-2543 Lymphocyte Aryl Hydrocarbon Hydroxylase (AHH) Inducibility in Acute Leukemia of Childhood (AL) (Meeting Abstract). (Eng) Blumer, J. L. (Dept. Pediatrics, Case Western Reserve Univ., Cleveland, OH); Dunn, R.; Gross, S. *Pediatr Res* 13(4, part 2): 429; 1979. (no refs)

79-2544 Oncogenesis-related Enzymes [Plasminogen Activator (PA), Arylhydrocarbon Hydroxylase (AHH)] in Leukemic and Normal Human Lymphocyte Lines in the Absence and Presence of Polyaromatic Carcinogens (PAHC's) (Meeting Abstract). (Eng) Freedman, H. J. (Roswell Park Memorial Inst., Buffalo, NY, 14260); Minowada, J.; Wilkens, H. J.; Back, N. *Fed Proc* 38(3, part 1): 369; 1979. (1 ref)

79-2545 Induction and Maintenance of Aryl Hydrocarbon Hydroxylase Activity in Mouse 3T3 Cells (Meeting Abstract). (Eng) Bittner, M. A. (Univ. Michigan, Ann Arbor, MI). *Diss Abstr Int [B]* 39(10): 4839; 1979. (no refs)

79-2546 Induction and Properties of Aryl Hydrocarbon Hydroxylase in Bovine Pancreatic Ducts. (Eng) Kahng, M. W. (Dept. Pathology, Univ. Maryland Sch. Medicine, 31 S. Greene St., Baltimore, MD, 21201); Jones, R. T.; Trump, B. F. *J Natl Cancer Inst* 62(5): 1251-1255; 1979.

The inducibility and characteristics of aryl hydrocarbon hydroxylase (AHH) in cultured bovine pancreatic ducts were

studied fluorometrically. AHH was present and inducible in all pancreatic ducts exposed to 20 μ g benz(a)anthracene (BA)/ml medium. AHH activity in the control tissue ranged from 1.0 to 3.0 units (U)/mg DNA, whereas the activity in the BA-treated tissue was 4.2-28.5 U/mg DNA. The induction ratio, defined as the ratio of the activity of enzyme preparations from BA-treated ducts to control enzyme activity, ranged from 5 to 18. After 12 hr of BA exposure, AHH activity in the treated tissue was 12 times that in the control tissue, and it continued to increase to 15, 19, and 31 times that in the control tissue at 24, 48, and 72 hr, respectively. The BA-induced AHH activity had a broad pH optimum between 7.1 and 7.7, and max activity was found at pH 7.4. The AHH activity was linear with respect to incubation time up to 30 min. A study was also made of the effect of benzo(a)-pyrene concentration on BA-induced AHH activity. The apparent Michaelis constant for the substrate was 0.5 μ M, and the max velocity was 8.6 U/mg DNA. BA-induced AHH activity was inhibited 65% by 100 μ M 7,8-benzoflavone, whereas control enzyme activity was stimulated 100% by the same concentration of 7,8-benzoflavone. (15 refs)

79-2547 Relationship Between Amount and Type of Dietary Fat in Promotion of Mammary Carcinogenesis Induced by 7,12-Dimethylbenz[a]anthracene. (Eng) Hopkins, G. J. (Dept. Clinical Biochemistry, Flinders Medical Center, Bedford Park, S. Australia 5042, Australia); Carroll, K. K. *J Natl Cancer Inst* 62(4): 1009-1012; 1979.

The relationship between the amount and type of dietary fat in the promotion of 7,12-dimethylbenz(a)anthracene (DMBA)-induced mammary carcinogenesis was investigated in female Sprague-Dawley rats. The rats were fed semipurified diets containing various fats, either alone or in combination, to provide different amounts of dietary fat and linoleic acid. One week before commencing the diets, each rat received an intragastric dose of 5 mg DMBA. Rats fed diets containing mixtures of 3% sunflower seed oil and 17% tallow or coconut oil developed twice as many tumors as those fed 3% sunflower seed oil or 20% of either saturated fat alone. Tumor yields in the rats fed the mixed-fat diets were comparable to those in rats fed a 20% lard diet, which provided about the same amount of linoleic acid. No further increase in tumor yield was observed in rats fed a 20% sunflower seed oil diet that contained more than five times as much linoleic acid. These results show that a certain amount of polyunsaturated fat, as well as a high level of dietary fat, is required to promote mammary carcinogenesis. (21 refs)

79-2548 Non-random Duplication of Chromosome 15 in Murine T-Cell Leukemias Induced in Mice Heterozygous for Translocation T(14:15)6. (Eng) Wiener, F. (Dept. Tumor Biology, Karolinska Institutet, S-104 01

Stockholm 60, Sweden); Spira, J.; Ohno, S.; Haran-Ghera, N.; Klein, G. *Int J Cancer* 23(4): 504-507; 1979.

The chromosome constitution of 7,12-dimethylbenz(a)anthracene (DMBA)-induced T-cell leukemias in C57BL x CBAT6T6 F₁ mice was studied. The CBAT6T6-derived chromosome T(14;15)6 was regularly duplicated, whereas the C57BL-derived normal chromosome 15 was only present in one copy. It was concluded that the gene(s) that tends to duplicate in parallel with the neoplastic transformation of a prothymocyte to an overt leukemic cell has a greater chance of duplicating and/or may have a stronger promoting effect on leukemogenesis if located on the CBA-derived, structurally rearranged T(14;15)6 than the corresponding gene(s) located on the C57BL-derived normal chromosome 15. (11 refs)

79-2549 Ultrastructural Alterations in Experimental Lingual Leukoplakia and Carcinoma. (Eng)

Marefat, M. P. (Dept. Oral Medicine and Oral Pathology, Harvard Sch. Dental Medicine, 188 Longwood Ave., Boston, MA 02115); Albright, J. T.; Shklar, G. *Oral Surg* 47(4): 334-342; 1979.

Epidermoid carcinomas, preceded by dysplastic leukoplakic lesions, were produced on the right lateral borders of the tongues of 12 male and 12 female Syrian hamsters by the application of 9,10-dimethyl-1,2-benz(a)anthracene (DMBA: 0.5% soln in acetone, 3x/wk) after the area had been scratched with a root canal broach. Animals were killed at 12-13 wk and at 15-16 wk after the start of treatment. Electron microscope studies of both the dysplastic leukoplakic lesions and the well-differentiated epidermoid carcinomas revealed clumping of the tonofilaments and widening of the intercellular spaces in addition to the cellular pleomorphism, altered nuclear-cytoplasmic ratio, and prominent nucleoli observed with the light microscope. In the carcinomas, there were variations in the morphology of the mitochondria, with tubular forms in evidence. The basal lamina in the carcinomas was discontinuous. Pseudopodia arising from basal cells were seen in areas of basal lamina discontinuity. (30 refs)

79-2550 Differential Rotational Mobility of a Membrane-bound Fluorochrome in Cell Types of Increasing Oncogenic Potential. (Eng) Monti, J. A. (Univ. Alabama in Birmingham, Medical Center, Birmingham, AL 35294); Sarraf, A. M.; Christian, S. T.; Saxholm, H. J. *Arch Biochem Biophys* 193(2): 496-501; 1979.

The interaction of the fluorescent phospholipid 4-nitrobenzo-2-oxa-1,3-diazole(aminocaproyl)-phosphatidylcholine NBD-PC, which was used as an extrinsic membrane probe, with mouse embryo C3H/10T1/2 clone

8 (10T1/2) cells and 10T1/2 cells transformed by 7,12-dimethylbenz(a)anthracene (DMBA: Types I-III) or 3-methylcholanthrene (3-MC: Type III) was determined. Specifically, differences in the fluorescence polarization and temperature-dependent emission intensity of incorporated NBD-PC among the various cells were compared. The effects of temperature on the emission intensity of membrane-bound NBD-PC differed for the 10T1/2 cells and the chemically transformed Type I (nononcogenic), Type II (moderately oncogenic), and Type III (highly oncogenic) cells. The behavior of NBD-PC bound to Type III cells transformed by DMBA and 3-MC was similar. The rotational mobility of the membrane-bound fluorochrome increased in the order: 3-MC-Type III > DMBA-Type III > Type II > Type I > 10T1/2. There were significant differences in NBD-PC polarization values between the 10T1/2 cells and the transformed cells, but there were no statistically significant differences in polarization values among the different transformed cells. The data suggest that the dynamic properties of the plasma membranes are sufficiently different for normal and chemically transformed cells to be differentiated on this basis. (36 refs)

79-2551 Suppressive Effect of 3-Methylcholanthrene on Phagocytic Activity of Mouse Peritoneal Macrophages for *Torulopsis glabrata*. (Eng) Tewari, R. P.

(Dept. Medical Sciences, Southern Illinois Univ. Sch. Medicine, Springfield, IL 62708); Balint, J. P.; Brown, K. A. *J Natl Cancer Inst* 62(4): 983-988; 1979.

The effect of methylcholanthrene (3-MC) on the phagocytic activity of C3H and CFW mouse peritoneal macrophages for *Torulopsis glabrata*, an organism occasionally seen as an opportunistic fungal infection in cancer patients, was investigated. Macrophages were maintained in glass scintillation vials or on cover slips in Leighton tubes with the use of Hanks' balanced salt soln plus 30% horse serum. Graded amounts of 3-MC (0, 1, 5, 10, 25, 50, or 100 µg/ml) were incorporated into the medium, and the macrophages were parasitized with viable cells of *T. glabrata*. Macrophages from C3H mice, a strain highly susceptible to 3-MC carcinogenesis, were more prone to the suppressive effect of 3-MC than were the macrophages from CFW mice, a relatively resistant strain. A significant suppressive effect on the phagocytosis of macrophages from C3H mice was observed with 5 µg/ml 3-MC, whereas up to 50 µg/ml did not alter the phagocytic activity of CFW macrophages. However, 100 µg/ml also suppressed the phagocytosis of CFW macrophages. Suppression of C3H macrophage phagocytosis was observed after 6 hr exposure to 3-MC, whereas a similar effect on CFW macrophages was seen after 12 hr. Treatment with 100 µg/ml 3-MC impaired the fungicidal activity of both C3H and CFW macrophages. These results indicate that there is a correlation between the suppressive effect of 3-MC on macrophage activity and the strain susceptibility of mice to chemical carcinogenesis. (38 refs)

- 79-2552 Rimantadine but Not Amantadine Protects Fischer Rat Embryo Cells from Transformation Induced by 3-Methylcholanthrene or Benzo(a)pyrene.** (Eng) Price, P. J. (Center Disease Control, 1600 Clifton Road, N.E., Atlanta, GA 30333); Mansfield, J. I.; Hassett, C. M. *In Vitro* 15(2): 82-85; 1979.

The in vitro oncogenic potential and the ability to protect cells from polycyclic hydrocarbon-induced transformation of the antiviral drugs amantadine hydrochloride (AM) and rimantadine hydrochloride (RM) were tested using a serial line of Fischer rat embryo cells (F1706) that previously had been shown to be an accurate indicator of chemicals known to be oncogenic in animal studies. Neither compound (0.5 µg/ml RM, 1.0 or 2.0 µg/ml AM) was found to have transforming activity. At these levels, which were slightly toxic, RM, but not AM, protected the cells from transformation induced by 3-methylcholanthrene (0.1 or 0.2 µg/ml) and benzo(a)pyrene (0.5 µg/ml). (11 refs)

- 79-2553 Effects of Donor Age on Neoplastic Transformation of Adult Mouse Bladder Epithelium In Vitro.** (Eng) Summerhayes, I. C. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Franks, L. M. *J Natl Cancer Inst* 62(4): 1017-1023; 1979.

An attempt was made to demonstrate the effects of aging on neoplastic transformation in long-term primary cultures of the bladder, an organ in which an age-associated tumor incidence has been found in humans. Neoplastic transformation of C57BL/1crf-a' mouse bladder epithelium was induced in long-term primary cultures by a single 24-hr treatment with 7,12-dimethylbenz[a]anthracene on day 2 of culture. Transformed foci appeared earlier (40-60 days) and at a higher frequency (28%) in cultures from old donors (28-30 mo), compared with 100 days and 0.9% in cultures from young adult donors (5-7 mo). After transplantation into syngeneic mice, transformed cells produced carcinomas. Spontaneous epithelial transformation occurred in dimethyl sulfoxide-treated old cultures after the same time interval (40-50 days) as in the carcinogen-treated cultures, but at a lower frequency (5.3%). Spontaneous epithelial transformation did not occur in cultures from young donors. (18 refs)

- 79-2554 Immunity to a 3-Methylcholanthrene-induced Fibrosarcoma in the C57BL/6 Mouse: In Vivo Analysis by the Adoptive Tumor Neutralization Test.** (Eng) Poupon, M. F. (Laboratoire d'Immunochimie, Institut de Recherches Scientifiques sur le Cancer, B. P. No. 8, 94800 Villejuif, France); Lespinats, G.; Kolb, J. P.; Payelle, B. *J Natl Cancer Inst* 62(4): 989-994; 1979.

The adoptive tumor neutralization test (modified Winn

test) was used to study antitumor immunity in C57BL/6 mice made immune to a 3-methylcholanthrene (3-MC)-induced fibrosarcoma (MC-B6-1). Spleen cells from mice immunized against this tumor partially or completely protected recipient mice from the growth of MC-B6-1 tumor cells when both spleen and tumor cells were inoculated sc. In the case of partial immunity, the tumor growth rate was decreased only during the first, and not the second, phase of tumor development. Protection in the Winn test was a specific phenomenon between MC-B6-1 and a second MCA-induced fibrosarcoma (MC-B6-2). T cells were required for successful immunity, with neither B cells nor macrophages from normal or immunized mice provided any protection against tumor growth. The degree of protection increased with the number of transferred immune spleen cells and with the number of challenges received by the immunized mice. The protective effect was abrogated when the immune spleen cells were exposed to 1,000 R radiation. When immune spleen cells were injected at a distance from the tumor cells, no protection was conferred. However, 49% of these mice were protected against a subsequent challenge. Immune spleen cells did not protect nude mice from the growth of the MC-B6-1 fibrosarcoma. The results indicate that it is possible to demonstrate an antitumor reaction to a chemically induced tumor by the Winn test. (21 refs)

- 79-2555 Effect of Carrageenan on the Non-specific Resistance of Mice to Injected Syngeneic Tumour Cells, Alone or in Mixtures.** (Eng) Wu, R. L. (Dept. Bacteriology, Univ. Sydney, Sydney, Australia); Kearney, R. *Br J Cancer* 39(3): 241-246; 1979.

The mechanisms of nonspecific resistance to two syngeneic methylcholanthrene-induced fibrosarcomas (H-1 and H-2) were investigated using inbred male CBA/H/WEHI mice. Sc inoculation of 0.05×10^5 H-1 or H-2 cells produced tumors in a small number of normal mice, whereas the same number of cells mixed with 10^6 viable, nonreplicating, mitomycin C-treated tumor cells (MCT cells) produced tumors in all normal animals. The effect was reduced when the MCT cells were heat-killed or freeze-thawed, and tumor growth was completely inhibited when latex particles were mixed with the graft. It is likely that the latex particles were toxic for the cells. Carrageenan pretreatment (3 consecutive daily ip doses of 0.5 mg) significantly enhanced the growth of H-1 cells alone, but it had no effect on the growth of MCT-H-1 grafts. Inoculation of MCT-H-1 cells ip did not enhance the growth of sc inoculated H-1 cells. The results support the concept of a nonspecific macrophage "surveillance" system that appears to be crucial in controlling tumor growth, since it determines the establishment of small numbers of tumor cells while they can still be easily destroyed. (24 refs)

- 79-2556 Mutagenesis of Human Cells by 3-Methylcholanthrene.** (Eng) Curren, R. D.

(Dept. Biochemical Oncology, Microbiological Assoc., 5221 River Rd., Bethesda, MD 20016); Homer, C. J.; Price, P. J.; Freeman, A. E. *Mutat Res* 60(1): 109-113; 1979.

The mutagenicity of 3-methylcholanthrene (3-MC: 10 µg/ml) for cultures of human foreskin epithelial cells mixed with xeroderma pigmentosum (XP) fibroblasts derived from a female patient was studied. High mutagenicity was observed in these cultures. Similar results were obtained in other experiments with lower doses of 3-MC and with benzo(a)pyrene. Mutant clones that showed resistance to 6-thioguanine or ouabain (OUA) retained their resistant phenotype even during growth in the absence of the selective agent. All five of the OUA-resistant clones had a male karyotype and a wild-type UV-sensitivity, indicating that they were not derived from the extremely UV-sensitive female XP cells. Thus, the induced mutants must have originated from the foreskin explants. It is suggested that the skin epithelial cells metabolized 3-MC to mutagenic products and that the XP cells may have served merely as nonspecific stabilizers of the culture conditions, permitting survival of the small mutant epithelial population. (19 refs)

79-2557 Biochemical Characterization of Alien H-2 Antigens Expressed on a Methylcholanthrene-induced Tumor. (Eng) Rogers, M. J. (Lab. Cell Biology, NCI, Bethesda, MD 20014); Appella, E.; Pierotti, M. A.; Invernizzi, G.; Parmiani, G. *Proc Natl Acad Sci USA* 76(3): 1415-1419; 1979.

Alien H-2 antigens expressed on a methylcholanthrene-induced tumor (C-1) of BALB/c (H-2d) origin were characterized. Membranes or purified antigens from C-1 tumor cells were used to inhibit the precipitation of highly purified H-2a by various alloantisera. The membranes completely inhibited the precipitation of an authentic H-2a molecule, especially by antisera against private specificity 23, indicating that a structural antigen identical to normal H-2Kk is expressed on the C-1 tumor. The alien H-2 molecule was expressed in very small amounts on the membranes of C-1 tumor cells, having a specific activity 1/10 that of H-2Kk antigens on normal cells. Like normal H-2 antigens, the alien antigens have a mol wt of 48,000, are glycoproteins, and are noncovalently bound to β_2 -microglobulin. They differ from normal H-2 antigens in their susceptibility to papain digestion, behavior during gel filtration, and overall stability. Both genetic and epigenetic mechanisms have been suggested to explain the appearance of previously unexpressed phenotypes. Present evidence seems to favor a derepression mechanism for the alien H-2 molecule. A direct mutation of the H-2 gene could cause the structural differences seen in the alien H-2 molecule by affecting the primary structure. Alternatively, the post-translational modification apparatus of the cell might be haplotype specific, so that an H-2Kk molecule would be incorrectly processed in an H-2d cell. (31 refs)

79-2558 Early Influences of the Tumor Promoter 12-O-Tetradecanoylphorbol 13-Acetate (TPA) but Not of 4-O-Methyl-TPA on the Cell Cycle of Hela Cells (Meeting Abstract). (Eng) Kinzel, V. (Institut für Experimentelle Pathologie, Deutsches Krebsforschungszentrum, Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Richards, J.; Stohr, M. *Hoppe Seylers Z Physiol Chem* 360(3): 300; 1979 (2 refs)

79-2559 Effect of Dietary Antioxidants on Epoxide Hydratase and on DNA Modification by Benzo[a]pyrene (Meeting Abstract). (Eng) Kahl, R. (Pharmakologisches Institut der Universität, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany); Klaus, E. *Hoppe Seylers Z Physiol Chem* 360(3): 294; 1979 (1 ref)

79-2560 Effect of the Tumor Promoter 12-O-Tetradecanoylphorbol 13-Acetate on the Synthesis of Epstein-Barr Virus (EBV) DNA in Producer and Nonproducer Cell Lines (Meeting Abstract). (Eng) Hudewentz, J. (Institut für Virologie, Zentrum für Hygiene, Universität Freiburg, Hermann-Herderstrasse 11, D-7800 Freiburg, W. Germany); Bornkamm, G. W.; zur Hausen, H. *Hoppe Seylers Z Physiol Chem* 360(3): 287-288; 1979 (1 ref)

79-2561 Inhibition of Epidermal Growth Factor Binding to Surface Receptors by Tumor Promoters. (Eng) Brown, K. D. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Dicker, P.; Rozengurt, E. *Biochem Biophys Res Commun* 86(4): 1037-1043; 1979.

The effect of the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) on the binding of radiolabeled epidermal growth factor (EGF) to surface receptors was investigated. Unlabeled EGF [0-20 nanograms (ng)/ml] and TPA (0-1,000 ng/ml) produced a potent, dose-dependent inhibition of [125]EGF (2 ng/ml) binding to Swiss mouse 3T3 cells. The inhibition of radiolabeled EGF binding by TPA was competitive, with no change in max binding capacity but with an approx 10-fold decrease in apparent binding affinity. At 25 ng/ml [125]EGF, the inhibition of binding by 100 ng/ml TPA was completely abolished. EGF inhibited radiolabeled EGF binding at 0 or 37°C, whereas TPA was inhibitory only at 37°C. The inhibitory effect of EGF was rapidly reversed when it was removed from the binding medium, whereas the effect of TPA persisted after its removal. The inhibition of binding was 52% in cells pretreated with saturating concentrations of EGF and 63% in nonpretreated cells, which suggests that TPA and EGF interact with different binding sites. This finding was further supported by ex-

periments in which TPA and EGF triggered biological responses in a synergistic manner. Treatment of 3T3 cells with TPA concentrations as high as 1 $\mu\text{g/ml}$ for 8 hr did not reduce the max binding capacity, but treatment with 2 ng/ml EGF caused a 50% decrease in this value. This indicates that TPA, in contrast to EGF, does not induce "down-regulation" of EGF receptors. The results suggest that TPA does not bind directly to EGF receptors. (19 refs)

79-2562 Inhibition of Metabolic Cooperation in Mamalian Tissue Culture Cells by Phorbol Myristate Acetate (Meeting Abstract). (Eng) Yotti, L. P. (Dept. Human Development, Michigan State Univ., East Lansing, MI, 48824); Trosko, J. E. *In Vitro* 15(3): 208-209; 1979. (no refs)

79-2563 Changes Induced in the Intercellular Connections of the Skin During Experimental Carcinogenesis (Meeting Abstract). (Eng) Komitowski, D. (German Cancer Res. Center, Inst. Experimental Pathology, Heidelberg, Germany). *J Cutan Pathol* 5(5): 270; 1978. (no refs)

79-2564 Modulation of Plasminogen Activator Synthesis in Chick Embryo Fibroblasts by Cyclic Nucleotides and Phorbol Myristate. (Eng) Wilson, E. L. (Dept. Clinical Science and Immunology, Medical Sch., Univ. Cape Town, Cape Town, South Africa); Reich, E. *Cancer Res* 39(5): 1579-1586; 1979.

The effects of phorbol-12-myristate-13-acetate (PMA) and a temperature-sensitive Rous sarcoma virus (ts-68) on plasminogen activator (PA) synthesis by chick embryo fibroblasts (CEF) were studied. Intracellular PA showed a dose- and duration-dependent increase in response to PMA. PMA also increased PA levels in ts-68-infected cells at both the permissive (37 C) and non-permissive (41 C) temperatures, the inductive effects of PMA and the effects of the shift to 37 C being synergistic. Viral transformation lowered the threshold concentration for a response to PMA. The transcriptional events required for PA induction were evident within < 1 hr, and translation was effective by 2 hr. Actinomycin D pretreatment (1 $\mu\text{g/ml}$) blocked the inducing and synergizing effect of PMA on PA synthesis in ts-68-infected CEF at both 37 and 41 C; blocked the inducing and synergizing effect of the sarcoma transformation, irrespective of any preexisting enzyme induction by PMA; and blocked the deinduction of PMA- or virus-determined enzyme levels regardless of the presence or absence of either inducing stimulus. The PA induced by PMA did not differ from that induced by ts-68. PA induction was strongly but reversibly inhibited by exposure of the CEF to AMP or cholera toxin. However, many of the morphological changes accompanying transformation were

not affected by concentrations of cyclic nucleotides that suppressed PA production. Thus, the two phenomena are at least partially independent expressions of transformation in this system. (39 refs)

79-2565 Reversible Inhibition by Retinoids of 3-Methylcholanthrene-induced Neoplastic Transformation in C3H/10T1/2 Clone 8 Cells. (Eng) Merriman, R. L. (Dept. Experimental Therapeutics, Grace Cancer Drug Center, Roswell Park Memorial Inst., New York State Dept. Health, Buffalo, NY); Bertram, J. S. *Cancer Res* 39(5): 1661-1666; 1979.

Cultured C3H/10T1/2 clone 8 mouse embryo fibroblasts were used to study the effects of retinoids on 3-methylcholanthrene (3-MC)-induced neoplastic transformation. At concentrations that did not affect cell survival (0.005-0.5 $\mu\text{g/ml}$), retinyl acetate inhibited 3-MC-induced transformation. Complete inhibition of transformation was observed when 3-MC-treated cells were continuously treated with retinyl acetate (0.1 $\mu\text{g/ml}$) starting 7 days after 3-MC exposure (2.5 $\mu\text{g/ml}$; 24 hr). A brief exposure to retinyl acetate (0.5 mg/ml) on days 7-14 after 3-MC treatment caused a 70% inhibition of transformation, and delaying retinyl acetate exposure up to 3 wk after 3-MC still decreased transformation by 80%. Nontoxic concentrations of retinol and retinal were approx equal in potency to retinyl acetate. The inhibition of neoplastic transformation by retinoids was fully reversible on maintaining initiated cultures for 3-5 wk in drug-free medium. Growth of three established transformed C3H/10T1/2 clone 8 cell lines in the presence of normal cells was not inhibited by 1- $\mu\text{g/ml}$ doses of retinyl acetate or retinol. Thus, in extension of the in vivo studies, it was shown that retinoids inhibit chemically induced neoplastic transformation in cultured mouse fibroblasts and, furthermore, that this inhibition is not related to toxicity. The results indicate that retinoids delay the progression of preneoplastic cells to fully neoplastic cells. (47 refs)

79-2566 Retinoid Inhibition of Carcinogen-induced Transformation in Cryopreserved Golden Syrian Hamster Embryo Cells (Meeting Abstract). (Eng) Traul, K. A. (Pfizer Inc., Maywood, NJ, 07607); Nanna, U.; Wolff, J. S.; Jensen, K. E. *In Vitro* 15(3): 218; 1979. (no refs)

79-2567 Role of Vitamin A in Chemical Carcinogenesis in the Rat Mammary Gland. (Rus) Khandudza, K. L. (Dept. Biophysics, State Univ., Moscow, USSR); Esakova, T. D.; Tarusov, B. N. *Biull Eksp Biol Med* 87(3): 273-275; 1979.

The effect of large po doses of vitamin A on the concentration of 7,12-dimethylbenz(a)anthracene (DMBA) in various or-

gans and tissues was studied in female Wistar rats. Animals received 150,000 IU vitamin A po for 3 days or 250,000 IU for 5 days; control rats received 1.25 ml of corn oil for 5 days. The rats were then inoculated iv with a lipid emulsion of DMBA (2 mg) and sacrificed 10 min or 4, 5, or 6 hr later. At 10 min after DMBA administration, the max concentration of DMBA was detected in the liver, followed by the adrenals, spleen, kidneys, blood, and mammary gland. The DMBA concentration decreased with time in all organs but the mammary gland. Vitamin A decreased the level of DMBA in all organs and in the blood, increased its rate of metabolism, and decreased the level of lipid-soluble DMBA metabolites (the decrease depended on the dose of vitamin A). (13 refs)

79-2568 Tumor Prevention by Vitamin A: In Vitro Studies with Cultured Human Fibroblasts. (Ger)

Kohl, F. V. (I. Medizinische Universitäts-Klinik Hamburg-Eppendorf, Martinistrasse 52, D-2000 Hamburg 20, W. Germany); Rudiger, H. W. *J Cancer Res Clin Oncol* 93(2): 149-160; 1979.

The effects of vitamin A palmitate (VAP), all-trans-retinoic acid (RA), and Ro 10-9359 [ethyl-all-trans-9-(4-methoxy-2,3,6-trimethylphenyl)-3,7-dimethyl-2,4,6,8-nonatetraenoate] on the metabolism of ³H-labeled benzo(a)pyrene were studied in normal diploid human fibroblast cultures. All three compounds caused a concentration-dependent inhibition of the metabolism of BP into active water-soluble metabolites. The concentrations necessary for 50% inhibition were approx 110 µg/ml for VAP, 12.5 µg/ml for Ro 10-9359, and 6.25 µg/ml for RA. The binding of BP metabolites to DNA was also reduced considerably by all three substances. In another experiment, VAP reduced ³H-leucine incorporation to the fibroblasts from 100% to 77%. The inhibiting effect of VAP on the overall protein synthesis of the fibroblasts was less than one-third of that produced by cycloheximide and actinomycin D. The inhibition by the three substances of the formation of water-soluble metabolites was due to a preferential inhibition of the de novo synthesis of BP-metabolizing enzymes, and not to direct inhibition of these enzymes. The findings indicate that the protective effect of vitamin A against certain carcinogens is based on its ability to inhibit activation of the procarcinogen. (33 refs)

79-2569 The Role of Vitamin A and Ascorbic Acid in Relation to Respiratory System Cancers (Meeting Abstract). (Eng)

Lopez-S, A. (Dept. Medicine, Louisiana State Univ. Sch. Medicine, New Orleans, LA); Yates, B.; Johnson, W. D.; Nze, R. *Clin Res* 27(2): 553A; 1979. (no refs)

79-2570 Radiorespirometric Analysis of Glucose Metabolism During the Feeding with 3'-Methyl-4-(dimethylamino)azobenzene in Rat. (Eng) Koji-

ma, S. (Faculty Pharmaceutical Sciences, Teikyo Univ., Suarashi 1091-1, Sagamiko-machi, Tsukui-gun, Kanagawa 199-01, Japan); Shiki, Y.; Kubodera, A. *Radioisotopes* 28(2): 78-83; 1979.

Glucose metabolism was analyzed by a radiorespirometric technique during the administration of 3'-methyl-4-(dimethylamino)azobenzene (3'-Me-DAB) to male Donryu rats. The rats were given a diet containing 0.06% 3'-Me-DAB for 30 wk starting at age 5 wk. Glucose(U-¹⁴C) was given ip (2.5 µCi), and the differential respiratory ¹⁴CO₂ radioactivity was detected continuously for 2 hr. The results of this assay were correlated with δ-fetoprotein (AFP) levels and glycolytic enzyme activities. The peak time (PT: the time when respiratory ¹⁴CO₂ radioactivity reached a max) occurred at 4 wk after 3'-Me-DAB feeding, and the peak height (PH: max radioactivity at PT) was reached at 4-5 wk. This coincided with the appearance of AFP at 3-6 wk. AFP was not detected at 7-10 wk, but it reappeared from the 11th wk onward. After 3'-Me-DAB administration, hexokinase activity decreased up to the 3rd wk, increased during the 4th wk, and returned to control levels at the 6th wk. Pyruvate kinase activity was inhibited immediately after 3'-Me-DAB feeding, but it recovered to control levels at the sixth wk. In tumor-bearing rats, hexokinase and phosphofructokinase activities were lower than those in control rats, but pyruvate kinase activity was twice that in controls. These results suggest that the changes in PT could be related to the activation of hexokinase, followed by activation of the direct oxidative pathway for the conversion of glucose. (27 refs)

79-2571 Reactivity of the Liver to Glucocorticoids During Chemical Hepatocarcinogenesis. (Rus)

Me-chitov, V. I. (Lab. Tumor Biochemistry, Cancer Res. Center, Moscow, USSR); Adler, V. V.; Shapot, V. S. *Biull Eksp Biol Med* 87(3): 270-279; 1979.

Wistar rats with a transplanted Zajdela hepatoma and Wistar rats that were kept on a diet containing 3-methyl-4'-dimethylaminoazobenzene were subjected to partial hepatectomy. One hr after surgery rats were inoculated ip with the glucocorticoid dexamethasone (1 mg/100 g), and 1 hr prior to sacrifice they were inoculated with ³H-thymidine (100 µCi/rat). Dexamethasone inhibited ³H-thymidine incorporation in both the normal hepatocytes and the primary hepatoma cells. Tyrosine aminotransferase inducibility was retained in the hepatoma cells, but hormonal induction of tryptophan pyrrolase was lost. (14 refs)

79-2572 Nitroxide Radicals Generated from Carcinogenic Aminoazo Dyes During Their Metabolism In Vivo and in Enzymatic System In Vitro. (Eng)

Kimura, T. (Biophysics Div., Natl. Cancer Center Res. Inst., Tokyo 104, Japan); Kodama, M.; Nagata, C. *Biochem Pharmacol* 28(4): 557-560; 1979.

The type of nitroxide radical formed from aminoazo dyes during their metabolism by rats and in an enzymatic system *in vitro* is described. 3'-Methyl-N,N-dimethyl-4-aminoazobenzene (3'-methyl-DAB) was incubated with liver microsomes from methylcholanthrene (MC)-treated rats. After incubation, electron spin resonance (ESR) measurement detected a free radical with a signal consisting of six hyperfine splittings. A much larger ESR signal having the same hyperfine structure was observed when 3'-methyl-DAB was replaced by 3'-methyl-N-methyl-4-aminoazobenzene (3'-methyl-MAB). The same ESR signal was also observed when DAB or MAB was incubated with liver microsomes, but the signals were smaller than those of the 3'-methyl substitutes. Comparison of those ESR signals with those generated by authentic samples indicated that the free radical generated enzymatically from 3'-methyl-DAB (or -MAB) was a nitroxide radical. The same type of free radical was also detected *in vivo*. After *po* administration of 3'-methyl-DAB to rats, the livers were removed and extracted with benzene. The ESR signal of the extract was the same as that observed in the *in vitro* experiment. The role of the nitroxide radical in carcinogenesis of aminoazo dyes is not known. However, the finding that the proximate form of aminoazo dyes are easily converted to the nitroxide radical *in vivo* suggests a causal role for this radical in aminoazo dye carcinogenesis. (9 refs)

- 79-2573 Carcinogenic Azo Dyes. IX. Detection of New Metabolites of 3'-Methyl-4-(dimethylamino)azobenzene in Rat Bile.** (Eng) Mori, Y. (Gifu Coll. Pharmacy, Mitahora-higashi 5-6-1, Gifu 502, Japan); Hori, T.; Toyoshi, K.; Horie, M. *J Pharmacobio Dyn* 1(3): 192-201; 1978.

Metabolites appearing in the bile of male Wistar rats administered a *po* dose of 3'-methyl-4-(dimethylamino)azobenzene (3'-Me-DAB; 45 mg/kg) were studied by the ion cluster technique using a mixture of unlabeled and deuterated compounds. The major metabolites appeared to be 3'-carboxyaminoazo dyes and their azo reduced compounds and 3-amino-6-hydroxytoluene and 3-aminobenzyl alcohol and their N-acetylated compounds. Other metabolites were 3'-Me-4-aminoazobenzene (3'-Me-4-AB), 3'-CHO-AB, 3'-CHO-4-(methylamino)azobenzene (3'-CHO-MAB), 3'-Me-MAB, 3'-Me-4'-OH-AB, 3'-Me-4'-OH-MAB, 3'-Me-4'-OH-DAB, 3'-CH₂OH-MAB, 3'-CH₂OH-DAB, 3'-CH₂OH-AB, 3'-Me-4'-OH-AB-NAc, 3-aminobenzaldehyde and its N-acetylated metabolite, 3'-Me-N-OH-AB, and 3'-Me-4-nitroazobenzene. The parent compound, 3'-Me-DAB, was hardly excreted in the bile. This is the first documentation of the occurrence of oxidative metabolism at the ring methyl group of 3'-Me-DAB in the rat. (11 refs)

- 79-2574 Histochemical Comparison of Focal Losses of RNase and ATPase Activities in Preneoplastic Rat Livers.** (Eng) Daoust, R. (Institut du Cancer de Montre-

al, Centre Hospitalier Notre-Dame, 1560 est rue Sherbrooke, Montreal, P.Q., Canada H2L 4M1). *J Histochem Cytochem* 27(2): 653-656; 1979.

To assess the significance of enzyme-deficient foci as putative premalignant lesions, parallel histochemical analyses of RNase and ATPase activities were carried out in serial sections of livers from male Wistar rats fed 4-dimethylaminoazobenzene (DAB; 0.06% of a basal low-protein diet). The RNase activity of the centrilobular liver parenchyma was markedly increased in animals given the basal diet without DAB; ATPase activity was not altered significantly. The cirrhotic liver parenchyma observed after 30 days of DAB feeding showed intense RNase and ATPase activities, whereas the trabecular tissues showed no RNase but high ATPase activity. After 60 days, some areas of the regenerating parenchyma were RNase- and ATPase-negative, and the hepatocytes of the hyperbasophilic foci and hepatocellular carcinomas that developed after 90 days were consistently deficient in RNase and ATPase activities. The RNase and ATPase activities of the regenerating livers of untreated hepatectomized rats were essentially normal. The results indicate that groups of hepatocytes show a simultaneous loss of nuclease and canalicular ATPase activities at early stages of hepatocarcinogenesis. These foci of altered cells may represent a subpopulation capable of progressing to neoplasia, since hyperbasophilic areas and tumor cells exhibit similar enzyme deficiencies. (36 refs)

- 79-2575 Carcinogenic Azo Dyes--Detection of New Metabolites of 3'-Methyl-4-(methylamino)azobenzene in Rat Bile.** (Eng) Mori, Y. (Gifu Coll. Pharmacy, 5-6-1, Mitahora-higashi, Gifu 502, Japan); Horie, M.; Toyoshi, K. *Radioisotopes* 28(2): 72-77; 1979.

The metabolism of 3'-methyl-4-(methylamino)azobenzene (3'-Me-MAB) in rat bile was studied by an ion cluster technique. A mixture of unlabeled and labeled 3'-Me-MAB (10 mg in 1 ml cottonseed oil) was administered to male Wistar rats *po* via a stomach tube, and the bile was collected for 24 hr and hydrolyzed enzymically. During the 24-hr collection period, 28.7% of the administered dose was excreted into the bile. The hydrolyzed bile was chromatographed on an Amberlite XAD-2 column. The column was washed with water, and the metabolites were eluted with methanol and separated by thin-layer chromatography. The mass spectrum of each metabolite was measured and compared with those of authentic samples. Eight radioactive peaks were recognized in the chromatograms. The main metabolites were identified as follows: 3'-methyl-4-aminoazobenzene, 3'-methyl-4'-hydroxymethylaminoazobenzene, 3-acetaminotoluene, 3'-CHO-AB, 3'-CHO-MAB, 3'-COOH-AB, 3'-COOH-MAB, and 3'-CH₂OH-AB. The last three metabolites were oxidized at the ring methyl group and were detected for the first time as metabolites in bile. It is suggested that ring-methylated aminoazo dyes such as 3'-Me-MAB and 3'-methyl-4-dimethylaminoazobenzene may be bifunctional carcinogens. (10 refs)

- 79-2576 Analysis of 2-Acetylaminofluorene in Laboratory Animal Chow by Reverse Phase Liquid Chromatography.** (Eng) West, R. W. (Natl. Center Toxicological Res., Jefferson, AR, 72079); Oller, W. L. *J Liq Chromatogr* 1(2): 181-185; 1978.

A high-speed liquid chromatographic procedure for the analysis of 2-acetylaminofluorene (2-AAF) in laboratory animal chow is described. The extract is passed through alumina to remove coextracted compounds that would interfere with resolution of the 2-AAF peak on the analytical column. Background absorbance is reduced, and baseline resolution of 2-AAF is achieved. Reverse-phase liquid chromatography allows minimal clean-up of extracts with good column stability and high sample throughput. (2 refs)

- 79-2577 Enhancement of 2-Acetylaminofluorene Liver Carcinogenesis in Rats Fed a Choline-devoid Diet.** (Eng) Lombardi, B. (Dept. Pathology, Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA, 15261); Shinozuka, H. *Int J Cancer* 23(4): 565-570; 1979.

The effects of feeding a choline-deficient (CD) or a choline-supplemented (CS) diet on liver tumor induction by 2-acetylaminofluorene (AAF) were investigated in male Sprague-Dawley rats. The rats were fed a CD or a CS diet containing 0.0075% AAF. Three to eight animals were killed 1, 3, 4, and 6 mo thereafter. Well- to moderately well-differentiated hepatocellular carcinomas developed in 50% and 75% of rats fed the CD + AAF diet for 4 and 6 mo, respectively. Cholangiocarcinomas developed after 6 mo in 38% of the animals. No tumors developed in rats fed the CS + AAF diet. The results confirm those obtained previously with DL-ethionine and azaserine, showing that feeding a CD diet greatly potentiates the induction of liver tumors in rats by chemical carcinogens. (19 refs)

- 79-2578 Transformation of Cultured Mouse Mammary Glands by Aromatic Amines and Amides and Their Derivatives.** (Eng) Tonelli, Q. J. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111); Custer, R. P.; Sorof, S. *Cancer Res* 39(5): 1784-1792; 1979.

The transformation model of the cultured whole mammary gland of mice was applied to a study of the carcinogenic aryl amines and amides and their activated derivatives. Mouse mammary glands in whole organ culture, previously demonstrated by others to undergo lobuloalveolar development, functional differentiation, and glandular involution, are transformable by these compounds. The transformation, which involves an escape from the hormonal controls of these processes, results in the formation of nodulelike alveolar lesions that are morphologically similar to presumptive preneoplastic lesions (hyperplastic alveolar nodules) that occur in mice during viral and chemical mammary tumorigenesis. The

transformation system discriminates between carcinogens and noncarcinogens of analogous structures in the fluorenyl and naphthylenic chemical series, and it is sensitively responsive to the presence of the amino or amide group or their derivatives. In a series of fluorenyl carcinogens, N-2-fluorenylacetamide displayed very high transforming activity > N-hydroxy-N-2-fluorenylacetamide (high activity) > N-acetoxy-N-2-fluorenylacetamide (moderate activity) > 2-fluorenamine and 2-nitrofluorene (low activity). In contrast, the noncarcinogen fluorene had little if any activity. The two naphthylenic carcinogens, 1-naphthylamine and 2-naphthylamine, displayed moderate transforming activity, but the noncarcinogen naphthalene had no activity. Control glands treated only with the vehicle (dimethyl sulfoxide) were not transformed. Considerable structural changes at the cellular level were caused by the carcinogens, much less by the noncarcinogens, and essentially none by the vehicle. It remains to be determined whether the glands transformed by these carcinogens can progress to tumors in vivo. These and previous results with carcinogenic polycyclic aromatic hydrocarbons suggest that that mouse mammary gland in whole organ culture may be transformable by a broad spectrum of chemical carcinogens. (38 refs)

- 79-2579 A High Pressure Liquid Chromatography Procedure for the Separation of Metabolites of 2-Acetylaminofluorene from Cells in Culture.** (Eng) Raineri, R. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD, 21501); Poiley, J. A.; Ernst, M. K.; Hillesund, T.; Pienta, R. J. *J Liquid Chromatography* 1(4): 457-467; 1978.

High-pressure liquid chromatography (HPLC) was used to determine picomole levels of the carcinogenic metabolite N-hydroxy-2-acetylaminofluorene (N-hydroxy-AAF) formed from N-2-acetylaminofluorene (AAF) by cells in culture. The extract from the cells and cell incubation medium was given a preliminary cleanup using thin-layer chromatography. N-hydroxy-AAF was then isolated using HPLC with a Waters Bondapak C₁₈/Corasil column and an acetonitrile-water elution system. Ring hydroxylation products were eluted with 23% acetonitrile, and AAF and the deacetylation product 2-aminofluorene (AF) were eluted with 33% acetonitrile. Resolution of N-hydroxy-AAF was accomplished using a linear gradient of 33%-99% acetonitrile. When cells in culture were incubated with 22 nanomoles/ml of AAF for 6 hr, 690 picomoles of N-hydroxy-AAF/10⁶ cells were formed by hamster hepatocytes compared with 13 and 8 picomoles/10⁶ cells for human embryo and hamster embryo cells, respectively. The rate of formation of N-hydroxy-AAF by hamster liver microsomes was 140 nanomoles/hr incubation/mg microsomal protein. (10 refs)

- 79-2580 Reactivity of the Antibodies to DNA Modified by the Carcinogen N-Acetoxy-N-acetyl-2-aminofluorene.** (Eng) Sage, E. (Centre de Biophysique

Moleculaire, C.N.R.S., 45045 Orleans Cedex, France); Fuchs, R. P.; Leng, M. *Biochemistry* 18(7): 1328-1332; 1979.

Rabbits were immunized with double-stranded native DNA (nDNA) that had been modified by in vitro reaction with N-acetoxy-N-acetyl-2-aminofluorene (AAAF), and the resulting antibodies were purified by affinity chromatography. This modified DNA is designated nDNA-AAF. The interactions between the purified antibodies or the Fab fragments and several ligands were studied. By radioimmunoassay, nDNA-AAF, heat-denatured DNA (dDNA)-AAF, and guanosine monophosphate (GMP)-AAF bound to the antibodies with about the same affinity. GMP modified by 2-aminofluorene (GMP-AF) interacted slightly less, and GMP modified by N-hydroxy-N-acetyl-2-aminofluorene (N-OH-AAF) did not interact with the antibodies. The values of the association constants deduced from fluorescence measurements for the binding of the Fab fragments to nDNA-AAF, dDNA-AAF, and GMP-AAF in 50 mM sodium chloride, pH 7.5, were of the same order of magnitude. The association constants for nDNA-AAF and dDNA-AAF and the amounts of precipitated antibodies decreased as the ionic strength increased. It is deduced that 1-1.5 phosphate groups interact by charge-charge interactions with the Fab fragments. The absorption and circular dichroism spectra of GMP-AAF, nDNA-AAF, and dDNA-AAF bound to the Fab fragments showed that the Fab fragments induced similar perturbation to the three ligands. These results indicate that the immunodeterminant group is the dGMP-AAF residue. (25 refs)

79-2581 Chronic Exposure to Low Concentrations of Halothane-Nitrous Oxide: Reproductive and Cytogenetic Effects in the Rat. (Eng) Coate, W. B. (Inhalation Toxicology Dept., Hazleton Labs. America, Inc., 9200 Leesburg Turnpike, Vienna, VA, 22180); Kapp, R. W.; Lewis, T. R. *Anesthesiology* 50(4): 310-318; 1979.

The reproductive, teratogenic, and mutagenic (cytogenetic) effects of low concentrations of halothane (HT) + nitrous oxide were determined in adult male and female Sprague-Dawley rats. The rats were exposed for 60 days prior to mating to 1 or 10 ppm HT + 50 or 500 ppm N₂O, respectively, for 7 hr/day, 5 days/wk. Inseminated females were exposed 7 hr/day from days 1 through 15 or from days 6 through 15 of their putative gestation. The former were allowed to deliver normally; fetuses of the latter were delivered by cesarean section on day 20. The males were exposed for an additional 40 wk (after the 3-wk mating period), after which bone marrow and spermatogonial cells were harvested for cytogenetic analysis. Exposure to 10 ppm HT + 500 ppm N₂O resulted in decreased ovulation and implantation efficiency and in slightly retarded fetal development (also seen at the lower level exposure). No major teratologic effect or unequivocal abortifacient effect of exposure of pregnant females during organogenesis or prior exposure of males was

observed. However, cytogenetic damage to both bone marrow and spermatogonial cells was found at both levels of exposure. (17 refs)

79-2582 Interstitial Cell Carcinomas of the Testis in BALB/c Male Mice Ingesting Methoxychlor. (Eng) Reuber, M. D. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501). *J Cancer Res Clin Oncol* 93(2): 173-179; 1979.

BALB/c and C3H male and female mice were fed 750 ppm methoxychlor [1,1'-(2,2,2-trichloroethylidene)bis(4-methoxy)benzene] or 100 ppm 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) in the diet for 2 yr. Interstitial cell carcinomas of the testis developed in 27/51 BALB/c male mice ingesting methoxychlor compared with 8/71 control mice. The testicular tumors were more malignant, developed at an earlier age, and were larger, less differentiated histologically, and more invasive in methoxychlor-treated mice than in control mice. They varied from well to poorly differentiated and undifferentiated and were capable of metastasis. They were also occasionally bilateral in treated mice. BALB/c mice receiving DDT and C3H male mice receiving methoxychlor or DDT did not develop testicular tumors. C3H male and female mice ingesting methoxychlor or DDT were more susceptible to the development of liver tumors than BALB/c mice. Methoxychlor-treated BALB/c mice also had lung tumors. The carcinogenicity of methoxychlor for the testis of BALB/c males is probably related to its estrogenic activity. (30 refs)

79-2583 Uptake of Benzo(a)pyrene, Carbaryl, DDT and Parathion in Cultured Human Cells: Re-evaluation. (Eng) Murakami, M. (Inst. Physical and Chemical Res., Wako-shi, Saitama 351, Japan); Fukami, J. *Bull Environ Contam Toxicol* 21(4/5): 478-482; 1979.

The uptake of the pesticides carbaryl, 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT), and parathion and of benzo(a)pyrene (BP) by cultured human embryonic lung cells was investigated. Labeled chemicals were added to the culture medium at a concentration of 4×10^{-6} M. The medium was removed after 24 hr, and the cells were fractionated into acid-soluble, ethanol-soluble, and acid- and ethanol-insoluble fractions. DDT was easily removed from the cells by ethanol; a very small amount remained in the acid- and ethanol-insoluble fraction. The insoluble fraction contained 30 times as much carbaryl and 6x as much parathion as DDT. About 40% of the BP added to the medium was extracted with ethanol. In another experiment, the cells were incubated with the chemicals for 4 or 48 hr and ghost monolayers were prepared by detergent treatment (Triton X-100). The amount of chemicals associated with the ghost monolayers increased with duration of incubation. The association of DDT with the ghost monolayer was considera-

bly less than that of the other three chemicals. These results indicate that although a large amount of DDT is taken up by the cells, only a small fraction accumulates in the acid- and ethanol-insoluble fraction or the detergent-resistant cytoskeleton. DDT, therefore, associates mainly with surface membrane structures by a nonspecific absorption process. Carbaryl and parathion are taken up by the cells to a lesser degree than DDT, but they are more closely associated with cellular components such as proteins. (6 refs)

- 79-2584 Carcinogenic Risk Assessment: Ethylene Dibromide.** (Eng) Ramsey, J. C. (Toxicology Res. Lab., Health and Environmental Res., Dow Chemical Co., Midland, MI, 48640); Park, C. N.; Ott, M. G.; Gehring, P. J. *Toxicol Appl Pharmacol* 47(2): 411-414; 1979.

The incidence of cancer following ethylene dibromide (EDB) exposure predicted by a one-hit carcinogenesis model using parameters derived from a bioassay in rats was compared with that observed in a group of 156 workers employed in EDB production. The parameter estimates were derived from the age-specific incidence of tumor formation in male rats treated with 40 mg/kg/day EDB by gavage in corn oil. The one-hit model estimated that an almost 100% lifetime incidence of cancer should be expected in workers exposed for 40 yr to 0.4 ppm EDB at citrus fumigation centers. The duration of exposure to EDB at two locations (at 1 of which employees were also exposed to carbon tetrachloride and chloroform) was determined by work history records of each employee and by an industrial hygiene survey of airborne concentrations of EDB at one of the locations. Time-weighted av (TWA) concentrations of EDB were conservatively assumed to be 3.0 ppm (23 mg/m³). The effect of a different exposure concentration was also conducted at an assumed TWA of 0.9 ppm (6.9 mg/m³). The one-hit model predicted either 85 or 54 neoplasms above the normal background incidence at TWA EDB concentrations of 3.0 or 0.9 ppm, respectively, compared with the 8 neoplasms observed to date in both employee groups combined. It is concluded that the use of the one-hit model results in highly exaggerated risk estimates in humans. (8 refs)

- 79-2585 Whole-Body Autoradiography and Allied Tracer Techniques in Distribution and Elimination Studies of Some Organic Solvents.** (Eng) Bergman, K. (Dept. Toxicology, Univ. Uppsala, S-751 23 Uppsala, Sweden). *Scand J Work Environ Health* 5(suppl. 1): 263 pp.; 1979.

Whole-body autoradiography was used to study the distribution and elimination of ¹⁴C-labeled benzene, toluene, xylene, styrene, methylene chloride, chloroform, carbon tetrachloride, and trichloroethylene in mice following inhalation exposure. All solvents were rapidly taken up by fatty and nervous tissues, where they were retained for various periods of time

depending largely on their fat solubility. Unexpectedly long retention times were noted for the chlorinated solvents, particularly chloroform, which was retained in the cerebellum, meninges, and spinal nerves. Metabolites appeared rapidly in several organs, primarily the liver and kidney, and they were excreted in the urine and bile. Toluene and xylene metabolites were completely extractable, whereas firmly bound metabolites of benzene, styrene, and the chlorinated solvents were found in the liver, kidney, and bronchi. Three-compartment pharmacokinetics were observed for most of the solvents studied, although a two-compartment model fit the elimination rate curves for styrene, methylene chloride, and chloroform in the exhaled air. The fat solubilities of the solvents influenced their pharmacokinetics, lower rate constants generally being obtained for the expiration of the more fat-soluble compounds. (478 refs)

- 79-2586 Reactions of Trichloroethylene Epoxide in Aqueous Systems.** (Eng) Henschler, D. (Inst. Toxicology, Univ. Wurzburg, Versbacher Strasse 9, D-8700 Wurzburg, W. Germany); Hoos, W. R.; Fetz, H.; Dallmeier, N.; Metzler, M. *Biochem Pharmacol* 28(4): 543-548; 1979.

The reactivity of trichloroethylene epoxide (TE) in aqueous systems was studied in an effort to determine its role in the metabolism, mutagenicity, and carcinogenicity of trichloroethylene (T). TE decomposed completely in unbuffered water into several acid products. Under C-C fission, it decomposed to carbon monoxide and formate. Glyoxylic acid and dichloroacetic acid were also formed. These results are contrary to those obtained in vivo, where T is metabolically transformed exclusively to trichlorinated C₂-compounds. It is suggested that T is epoxidized by mixed function oxygenases and that TE rearranges immediately within the hydrophobic premise of the enzyme, due to the catalytic action of the trivalent iron of P-450, to the nonreactive chloral. Thus, under normal in vivo conditions, the highly reactive epoxide is protected from the decomposition reactions observed in vitro. This hypothesis is consistent with the very low mutagenic and doubtful carcinogenic potential of T. (23 refs)

- 79-2587 Evidence for Possible Lack of Safety for Human Consumption of Dichloroacetate, a "Sister Compound" and Frequent Ingredient of the Non-Vitamin "B₁₅" (Pangamate) (Meeting Abstract).** (Eng) Herbert, V. (Bronx Veterans Admin. Hosp., New York, NY); Gardner, A.; Colman, N. *Am J Clin Nutr* 32(4): 952; 1979. (3 refs)

- 79-2588 Mutagenic and Alkylating Metabolites of Halo-Ethylenes, Chlorobutadienes and Dichlorobutenes Produced by Rodent or Human Liver Tissues. Evidence for Oxirane Formation by P450-linked Microso-**

mal Mono-oxygenases. (Eng) Bartsch, H. (International Agency for Res. Cancer, 150 Cours Albert-Thomas, F-69372 Lyon, Cedex 2, France); Malaveille, C.; Barbin, A.; Planche, G. *Arch Toxicol* 41(4): 249-277; 1979.

The abilities of various haloolefins to induce *his+* revertants in *Salmonella typhimurium* strains TA100 and TA1530 in the presence of mouse or human postmitochondrial liver supernatants were studied. With mouse liver microsomes, mutagenicity decreased in the following order: 3,4-dichlorobutene-1 > 1-chlorobutadiene > 2-chlorobutadiene > vinyl bromide > vinylidene chloride (VDC) > vinyl chloride (VC). 1,1,2-Trichloroethylene and 1,1-difluoroethylene were marginally mutagenic, and tetrachloroethylene and vinyl acetate were nonmutagenic. With human liver fractions, VC, vinyl bromide, VDC, and 2-chlorobutadiene were mutagenic. 1,4-Dichloro-2,3-epoxybutane was less mutagenic than 1,4-dichlorobutene-2, the mutagenicity of which was increased by liver microsomes. The mutagenicities of VC and VDC were increased up to twofold in the presence of liver microsomes from rats pretreated with phenobarbital or 3-methylcholanthrene, but pregnenolone-16 α -carbonitrile, aminoacetonitrile, and disulfiram pretreatment decreased the mutagenic effects. VC and, probably, vinyl bromide were epoxidized by mouse liver microsomes. 2-Chlorobutadiene, but not 1,1-difluoroethylene, 1,1-dichloroethylene, or 1,1,2-trichloroethylene, yielded an alkylating intermediate. The alkylating activity of 2-chloro- and 1-chlorobutadiene, 3,4-dichlorobutene-1, and 1,4-dichlorobutene-2 and its 2,3-epoxide derivative was not related quantitatively to mutagenicity. The data indicate that oxidation of the double bond in certain haloolefins is a common pathway in the formation of biologically active intermediates. (78 refs)

79-2589 Y-Chromosomal Nondisjunction in Dibromochloropropane-exposed Workmen. (Eng) Kapp, R. W. (Genetic Toxicology, Hazleton Lab. America, Inc., Vienna, VA, 22180); Picciano, D. J.; Jacobson, C. B. *Mutat Res* 64(1): 47-51; 1979.

The incidence of Y-chromosome nondisjunction (YFF) in the sperm of 18 workers exposed to 1,2-dibromo-3-chloropropane (DBCP) from 6 to 18 mo (mean, 15.2 mo) was investigated. In semen samples from 15 individuals with no known exposure to DBCP, an av of 41.5% (36.7%-46.3%) of the spermatozoa were positive for Y chromosomes (YF bodies), and the av YFF frequency was 1.2% (0.8%-1.8%). Samples from the DBCP-exposed workers showed an av YF frequency of 41.8% (36.3%-46.3%) and an av YFF of 3.8% (2.0-5.3%). The differences in YF frequency between the exposed and nonexposed groups were not significant, whereas the differences in YFF frequency were very highly significant. (15 refs)

79-2590 A Teratogenicity and Tissue Distribution Study on Dibromochloropropane in the Rat. (Eng)

Ruddick, J. A. (Food Directorate, Health Protection Branch, New Res. Center, Tunney's Pasture, Ottawa, Ontario K1A 0L2, Canada); Newsome, W. H. *Bull Environ Contam Toxicol* 21(4/5): 483-487; 1979.

The teratogenicity and tissue distribution of dibromochloropropane (DBCP) was studied in primiparous Wistar rats. Groups of rats received 0, 12.5, 25.0, or 50.0 mg/kg DBCP po on gestation days 6-15. Necropsies were performed on day 22. Another group of pregnant females was treated with 25 mg/kg DBCP po on gestation days 6-15 and sacrificed at 1, 3, 6, 12, or 24 hr after the last DBCP dose; the fetuses were assayed for DBCP. DBCP was not teratogenic at the doses given, but it was toxic to both the dams and fetuses. There was a significant difference between the wt of fetuses from the 50.0-mg/kg-dosed dams and that of control fetuses. This fetotoxic effect is related to the maternal toxicity of DBCP. Heart, brain, fat, and blood contained detectable amounts of DBCP at 12 hr after administration, but by 24 hr only fat still contained DBCP (0.17 ppm). Apparently, there was no accumulation of DBCP in the tissues examined (spleen, brain, heart, lung, kidney, liver, and blood), except fat. (5 refs)

79-2591 The Effect of Chloroform Ingestion on Some Hepatic Carcinogen Metabolising Enzymes in Rats. (Eng) Capel, I. D. (Res. Dept., Marie Curie Foundation, The Chart, Oxted, Surrey RH8 0TL, England); Williams, D. C. *IRCS Med Sci (Cancer)* 7(2): 82; 1979.

The ingestion of chloroform at 0.15 mg/kg/day for 14 days did not adversely affect gross liver wt or microsomal protein content in male Sprague-Dawley rats. However, the ingestion of chloroform at 0.15 or 15 mg/kg/day increased benzo(a)pyrene binding by DNA in the presence of 1,1,1-trichloropropene-2,3-oxide, suggesting that epoxide hydratase activity was increased. (6 refs)

79-2592 Radiographic Study of Gastric Hyperplasia Induced by Polychlorinated Biphenyls in the Rhesus Monkey. (Eng) Silverman, S. (Dept. Veterinary Radiology, Univ. California, Davis, 4301 X Street, Davis, CA, 95817); Rosenquist, C. J.; McNulty, W. P. *Invest Radiol* 14(1): 65-69; 1979.

The effect of 3,4,3',4'-tetrachlorobiphenyl (TCB: 3 ppm in the diet) on three immature male rhesus monkeys was studied radiographically. Clinical signs of toxicity, including swelling and erythema of the eyelids and periorbital edema, developed in 14-17 days. By day 23, two animals showed vomiting, anorexia, and wt loss; the third had no gastric distress. Radiographic examination of the first two monkeys revealed thickening of the gastric wall along the greater curvature of the distal body and antrum and narrowing and rigidity of the junction of the body and antrum. By day 62, the lesion in-

volved a larger area, thickening was increased, and nodularity and irregularity of the mucosal and serosal surfaces were observed. Both of these animals died within 62 days. The third TCB-treated monkey showed no more than a possible lesion on radiographic examination, and there was no evidence of further gastric involvement on subsequent examinations. Radiographically, polychlorinated biphenyl-induced lesions in the rhesus monkey strongly resemble an infiltrating-type carcinoma and therefore provide an excellent model for evaluating radiographic diagnoses of early infiltrative-type lesions. (6 refs)

- 79-2593 Changes in Peroxisomes in Preneoplastic Liver and Hepatoma of Mice Induced by α -Benzene Hexachloride.** (Eng) Tsukada, H. (Dept. Pathology, Cancer Res. Inst., Sapporo Medical Coll., S-1, W-17, Chuo-ku, Sapporo 060, Japan); Gotoh, M.; Mochizuki, Y.; Furukawa, K. *Cancer Res* 39(5): 1628-1634; 1979.

Changes in the number and size of peroxisomes (POS's) in hepatomas and hyperplastic nodules (HPN's) induced in the male DD mouse liver by α -benzene hexachloride (BHC) were determined histochemically and electron microscopically. The mice were divided into two groups: one was fed a diet containing 500 ppm BHC and the other was fed the BHC diet plus, 4 wk before sacrifice, 1% ethyl- α -p-chlorophenoxyisobutyrate (CPIB). CPIB is a well-known POS inducer. Six mice from each group were examined 16-36 wk after the start of the BHC diet. HPN were found in 1/6 mice at 16 wk and 5/6 mice at 20 wk, irrespective of CPIB treatment. At 24 and 28 wk, HPN were found in 5/6 mice and hepatomas in 2/6 mice of both groups. At 32 and 36 wk, all mice had HPN, and 3/6 mice in each group had hepatomas. Although most of the hepatomas were well-differentiated and contained a considerable number of POS's they showed no response to the POS-inducing stimulus of CPIB. However, the size of these organelles was increased; ie, the induction of peroxisomal protein synthesis may not be lost completely, in contrast to the induction of POS proliferation. In agreement with previous findings, hepatocytic hyperplasia of the hepatocytes was found to originate within the centrilobular zone of the liver. As a result of this proliferation, HPN eventually formed. Within the nodules, hepatocytic proliferation and "maturation" occurred simultaneously. Maturation was characterized by an increase in the number of POS's and in the inducibility of these organelles by CPIB. Cells within the HPN that did not show further maturation or lost their POS inducibility during maturation continued to proliferate, resulting in hepatoma formation. These results are similar to those of rat hepatocarcinogenesis studies, except that abnormal matrical tubules of POS's were not formed in the mouse HPN cells in response to CPIB. (28 refs)

- 79-2594 Effect of Acrylonitrile on Primary Syrian Golden Hamster Embryo Cells in Culture: Transformation and DNA Fragmentation.** (Eng) Parent, R. A. (Food

and Drug Res. Labs., P.O. Box 107, Route 17C, Waverly, NY, 14892); Casto, B. C. *J Natl Cancer Inst* 62(4): 1025-1029; 1979.

The effect of acrylonitrile (ACN) on the transformation of primary Syrian golden hamster embryo cells (HEC) in culture was established by two approaches. The focus assay involved the plating of small numbers of HEC in tertiary culture and counting the chemically transformed foci. The second assay involved the enhancement of viral transformation performed by inoculating the HEC with an oncogenic adenovirus after ACN treatment and counting the resulting virus-transformed foci 3 wk later. Application of 50 or 100 μ g/ml ACN produced foci of morphologically transformed cells. When similar cells were pretreated with simian adenovirus (SA7) and subsequently treated with ACN (25-200 μ g/ml), up to an 8.9-fold increase in the frequency of virus-transformed foci was noted over that found in cultures treated only with SA7. When [3 H]thymidine-labeled primary Syrian golden HEC were treated with ACN and cellular DNA was subsequently subjected to alkaline sucrose gradients, a shift in the sedimentation pattern reminiscent of that observed for chemical carcinogens was noted. These observations support recent studies indicating that ACN may be carcinogenic. (23 refs)

- 79-2595 Sensitivity of Hexokinase and Glucokinase to Exposure to Hormones During Hepatocarcinogenesis.** (Rus) Kil'dema, L. A. (Inst. Experimental and Clinical Medicine, Tallin, USSR). *Vopr Med Khim* 24(1): 36-41; 1979.

The effect of hydrocortisone (HC) and insulin (IS) on the activity of key carbohydrate metabolism enzymes during diethylnitrosamine (DENA)-induced hepatocarcinogenesis was evaluated in Wistar rats. The rats received 2.5 mg/kg/day DENA in the drinking water, 6 days/wk, for 8 mo. The hormones were given at the end of the 3rd, 5th, 6th, 7th or 8th mo (HC: 5 mg/kg/day, for 7 days, im; IS: 2 IU/100 g, bid, for 2 days sc). Animals were sacrificed after hormone administration, and the activity and isoenzyme spectrum of hexokinase (HK) and glucokinase (GK) in the liver were assessed. Administration of HC to control rats caused a 25% decrease in HK activity (primarily due to a decrease in the activity of isozyme II) and a 23%-38% decrease in GK activity. IS did not affect HK activity, but it increased the activity of GK by 177%. The first precancerous changes (focal hyperplasia) were detected after 5 mo of DENA treatment: after 6-7 mo, the rats had 3- to 4-mm-diameter tumor nodules and hepatomas, and after 8 mo, they developed highly differentiated hepatocarcinomas. The effect of HC on HK and GK activities was less pronounced in rats with DENA-induced hepatomas than in controls: there was a 17% and 12% decrease in HK and GK activity, respectively, after 5 mo of DENA treatment and a 8% and 12% decrease after 8 mo. IS did not affect HK activity, but it increased GK activity by 189% after 5 mo, and by 188% after 8 mo. (21 refs)

- 79-2596 Study of Mutagenicity of Nitroso Compounds in *Escherichia coli*.** (Rus) Pogodina, O. N. (Inst. Cytology, Leningrad, USSR). *Genetika* 14(12): 2113-2118; 1978.

The mutagenic activity of two carcinogenic nitrosamines was studied in two strains of *Escherichia coli*. Incubation of the *HfrH thi-* and *his-* strains with 5% dimethylnitrosamine (DMNA) and 1% diethylnitrosamine (DENA) for 1 hr decreased the survival of the treated cells. The *thi-* strain was slightly more sensitive (62.0% and 58.7% of the cells survived treatment with DMNA and DENA, respectively, compared with 66.0% and 68.8% for *his-* strain). Both carcinogens lacked mutagenic activity. However, in vitro hydroxylation of the compounds with 0.1 M sodium nitrite resulted in a 1.5-fold increase in the mutagenic activity of DMNA and 3.5-fold increase in the mutagenic activity of DENA. Both forward and backward mutations were induced. (22 refs)

- 79-2597 Antigenic and Morphological Differences in Diethylnitrosamine-induced Hepatomas as a Function of Dose.** (Spa) Uribarrena, R. (Departamento de Investigaciones Medicas, Universidad de Navarra, Pamplona, Spain); Perez Miranda, M.; Pons, F.; Cabarcos, A.; Ayensa, C.; Amezcaga, A. *Rev Esp Oncol* 25(2): 221-240; 1978.

The morphology and antigenic characteristics of hepatomas induced by diethylnitrosamine (DENA) were studied in Wistar rats as a function of dose. The total dose administered in drinking water was 8 mg/kg/day in Group A and 4 mg/kg/day in Group B (30 rats each). The high dose produced cirrhotic lesions and then hepatomas after 22 wk, but hepatomas without preceding or concurrent cirrhosis were found in Group B. In this group, the first tumor appeared after 30 wk. The malignant liver tissue was characterized by a high albumin level and the exclusive presence of β -transferin, which is not found in cirrhotic or normal livers. Circulating neoantigen was found exclusively in the tumor-bearing animals; it was present in 18/35 sera. The antigenic spectrum of the Group A rats was more complex than that of the Group B rats, in whom the max number of bands was 3. The increased antigenic complexity found in Group A was probably due to the toxic effects of DENA, which produced morphological and protein changes that had nothing to do with the neoplastic character of the cell. (25 refs)

- 79-2598 Volatile Nitrosamine Contamination of Laboratory Animal Diets (Letter to Editor).** (Eng) Edwards, G. S. (Thermo Electron Corp., Waltham, MA, 02154); Fox, J. G.; Policastro, P.; Goff, U.; Wolf, M. H.; Fine, D. H. *Cancer Res* 39(5): 1857-1858; 1979.

The presence of N-nitroso contaminants in a small sampling

of laboratory animal diets is reported. Low levels of the carcinogen N-nitrosodimethylamine (NDMA: 1-4 ppb) were found in 11/12 samples of commercial pelleted diets for laboratory animals. Higher levels (5-50 ppb) of NDMA were found in 3/7 samples of the NIH open-formula rat and mouse ration. The fish meal used in this diet contained NDMA in quantities sufficient to account for most of the contamination. In addition, several samples of the NIH diet contained low levels of N-nitrosopyrrolidine (0-2.1 ppb). The implications of these findings with respect to carcinogenicity testing are summarized briefly. For example, nitrosamine dietary contaminants could act synergistically with other (test) carcinogens or cocarcinogens to increase tumor incidence or alter the target organ. (13 refs)

- 79-2599 Increased Excision of O⁶-Methylguanine from Rat Liver DNA after Chronic Administration of Dimethylnitrosamine.** (Eng) Montesano, R. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, 150 cours Albert Thomas, 69372 Lyon Cedex 2, France); Bresil, H.; Margison, G. P. *Cancer Res* 39(5): 1798-1802; 1979.

The effect of chronic exposure of male BDIV rats to dimethylnitrosamine (DMN) on the ability of the liver to excise O⁶-methylguanine (OMG) from DNA in vivo was examined. The rats were given DMN (2 mg/kg/day, 5 days/wk) by stomach tube for a total of 9 wk, the final dose being of ¹⁴C-labeled material. Control rats received only the labeled DMN. Liver DNA was isolated 2-12 hr later, and normal and alkylated purines were determined after hydrolysis in mild acid chromatography on Sephadex G-10. The levels (measured as disintegrations per minute/micromole of parent base) of 7-methylguanine in the DNA of the experimental rats were the same as or slightly higher than those of the control animals, and the persistence of this product was similar in both groups. This was also true for 3-methyladenine. In contrast, the initial amount of OMG in the liver DNA of the experimental rats was one-third of the amount found in the control rats, and the rate of loss of this product from DNA was higher in the experimental animals. These differences were reflected in the alkylation product ratios: the 3-methyladenine:7-methylguanine ratios were closely similar in the two groups of animals at all times, whereas the OMG:7-methylguanine ratio was initially three times higher in the control animals and fell more slowly. DNA synthesis (as measured by the incorporation of [³H]thymidine) was higher in the liver, kidney, and lung of the experimental rats. (43 refs)

- 79-2600 Kupffer Cell Sarcoma in Rats after Exposure to Small Doses of Dimethylnitrosamine and N-2-Acetylaminofluorene During Hepatic Regeneration.** (Eng) Chopra, P. (Dept. Pathology, All India Inst. Medical Sciences, New Delhi 110016, India); Manga, A.; Nayak, N. C. *J Natl Cancer Inst* 62(4): 1089-1095; 1979.

CHEMICAL CARCINOGENESIS

The occurrence of primary liver sarcomas in normal, CCl₄-treated, or 70% hepatectomized (He-x) adult male Wistar rats treated with dimethylnitrosamine (DMN: 0.8 mg/100 g, once or twice at an interval of 14 days) or N-2-acetylaminofluorene (AAF: 2 mg/100 g/day for 3 days) is reported. Sarcomas occurred in 3.8% of rats treated with CCl₄ (100 µl/100 g) + DMN, 7.8% of rats treated with He-x + DMN, 9.2% of rats treated with He-x + AAF, 5.3% of normal animals treated with DMN, and 0% of normal animals treated with AAF. The differences in incidence between groups were not particularly significant. The sarcomas showed a consistent pattern in almost all animals. The tumor cells contained evidence of phagocytosis and many structural features attributed to Kupffer's cells. They grew along the sinusoids or filled these spaces, and sometimes they invaded the hepatic vein tributaries. Thus, it appeared that the sarcomas originated from Kupffer cells. In 7/13 rats in which the lungs were examined, multiple tumor emboli resembling those in the liver were present in the blood vessels. Reticuloendothelial cell hyperplasia was observed in the spleen, and a tumor identical to those in the liver was observed in one rat. This animal also had an intestinal tumor that resembled those in the liver and spleen. (24 refs)

- 79-2601 Persistence of Methylated Bases in Ribonucleic Acid of Syrian Golden Hamster Liver after Administration of Dimethylnitrosamine.** (Eng) Margison, G. P. (Paterson Labs., Christie Hosp., Manchester M20 9BX, England); Margison, J. M.; Montesano, R. *Biochem J* 177(3): 967-973; 1979.

Syrian golden hamster liver ribosomal RNA was isolated up to 96 hr after the hamsters were injected ip with ¹⁴C-dimethylnitrosamine (DMN: 25 or 2.5 mg/kg). 7-Methylguanine, 3-methylcytosine, O⁶-methylguanosine and 1-methyladenosine concentrations were measured after acidic or enzymic hydrolysis of the RNA to bases or mononucleosides followed by ion-exchange chromatography. After the high dose, alkylation appeared to be max at 12 hr, followed by a decrease, which was most rapid between 24 and 48 hr. After the low dose, the amounts of alkylation products were seven times lower, and no changes in the rates of loss of the products were observed between 7 and 96 hr. After both the high and low dose, the relative amounts of the four alkylation products appeared not to change between 7 and 96 hr, posttreatment. These results suggest that base-specific excision repair does not exist for RNA alkylation products in this system. (32 refs)

- 79-2602 Mediated Mutagenesis of Dimethylnitrosamine in *Neurospora crassa* by Various Metabolic Activation Systems.** (Eng) Whong, W. Z. (Cancer Res. Center, Thermo Electron Corp., Waltham, MA); Ong, T. *Cancer Res* 39(5): 1525-1528; 1979.

Studies were conducted to determine whether dimethylni-

trosamine (DMN) can be converted to metabolites mutagenic to *Neurospora crassa* in different in vivo and in vitro activation systems. The activation of DMN to mutagenic metabolites by liver, lung, and kidney of male Sprague-Dawley rats and male Swiss outbred albino mice was also compared in the in vivo and in vitro assays. Four metabolic activation systems (growth-mediated, mycelium extract-mediated, host-mediated, and organ homogenate-mediated) were used to study the mutagenicity of DMN in both forward and reverse mutation systems in the *ad-3* (adenine-3) region of *N. crassa*. DMN was not mutagenic in *Neurospora* if conidia alone were treated. It was highly mutagenic, however, if conidia were treated with this compound under any of the four activation systems. Quantitative differences in DMN-induced mutation frequencies were observed between in vivo (growth- and host-mediated) and in vitro (mycelium extract- and organ homogenate-mediated) activations. The efficiency of the conversion of DMN to mutagenic metabolites by the rodent organs appeared to be in a reversed order between the host-mediated (liver > kidney > lung) and the organ homogenate-mediated (lung > kidney > liver) assays. Inductions of reverse mutations in strain N23 indicated that DMN induces base-pair substitutions in *N. crassa*. (23 refs)

- 79-2603 Persistence of DNA Damage During Development of Liver Angiosarcoma in Rats Fed Dimethylnitrosamine.** (Eng) Abanobi, S. E. (Dept. Pathology, Univ. Toronto, Medical Sciences Building, Toronto, Ontario M5S 1A8, Canada); Farber, E.; Sarma, D. S. *Cancer Res* 39(5): 1592-1596; 1979.

Liver DNA damage induced by dimethylnitrosamine (DMN) and the subsequent repair of this damage under conditions that lead to the development of cancer were studied. Chronic feeding of a diet containing 50 ppm DMN to male Wistar rats resulted in liver DNA damage monitored as slow sedimentation of the DNA, compared with that of control liver DNA, in alkaline sucrose gradients. The damage in rat liver DNA could be seen within 2 days after DMN treatment, and it was progressive with duration of feeding up to 8 wk. Extended feeding up to 15-31 wk did not result in a proportionate increase in DNA damage. The DNA damage observed at 8 wk persisted until 31 wk, at which time liver angiosarcoma was present. Despite the fact that the DNA damage induced by DMN appears to involve the bulk of the liver DNA, the tumors developed were entirely from vascular endothelium. (25 refs)

- 79-2604 Evidence for Several Demethylase Enzymes in the Oxidation of Dimethylnitrosamine and Phenylmethylnitrosamine by Rat Liver Fractions.** (Eng) Kroege-Koepeke, M. B. (Frederick Cancer Res. Center, P.O. Box B, Frederick, MD, 21501); Michejda, C. J. *Cancer Res* 39(5): 1587-1591; 1979.

An attempt was made to provide additional evidence for the

existence of a number of nitrosamine demethylases, some of which are membrane-bound and others which are apparently soluble and probably perform their function in the cytoplasm. The steady-state kinetics and isotope effects were examined for the demethylation of dimethylnitrosamine (DMN) and phenylnitrosamine (PMN), as well as their deuterated analogs, using the S-9 supernatant fraction, the microsomal pellet, and the postmicrosomal supernatant from rat livers. The isotope effect (ratio of max rates for the deuterated and light substrates) using the S-9 fraction from Long-Evans rat livers was 1.82 for DMN and 5.38 for PMN. Phenobarbital induced dimethylnitrosamine demethylase activity in the microsomal pellet of both Long-Evans and Sprague-Dawley rats, but it repressed this activity in the postmicrosomal supernatant from Long-Evans rats and increased it markedly in the postmicrosomal supernatant from Sprague-Dawley rats. There was nitrosamine demethylase activity in the so-called "pH 5 enzymes" and in the supernatant from that preparation. The latter activity showed substantially different characteristics from those found in the other fractions. (12 refs)

79-2605 Chromatographic Determination of Volatile N-Nitrosamines in Natural and Waste Waters.

(Rus) Kosmenko, L. D. (N. N. Petrov Res. Inst. Oncology, Leningrad, USSR); Kosmenko, V. G. *Gig Sanit* (2): 53-57; 1979.

The efficacy of various chromatographic techniques for the detection of volatile nitrosamines in bodies of water and wastewater was tested. Four dialkyl nitrosamines, dimethylnitrosamine (DMNA), diethylnitrosamine (DENA), di-n-propylnitrosamine (DPNA), and di-n-butylnitrosamine (DBNA), were analyzed in water samples from the Neva River at Leningrad and in wastewater samples from a hydrolysis plant. The most reliable results were obtained with the simultaneous use of gas-liquid or liquid chromatography and spectrophotometry. The Neva River water contained < 5 µg/ml DMNA, DENA, DPNA, or DBNA, the wastewater < 20 µg/ml. (15 refs)

79-2606 Vascular Liver Tumors Induced in *Mastomys (Praomys) natalensis* by Single or Twofold Administration of Dimethylnitrosamine. (Eng) Wayss, K. (Inst. Experimental Pathology, German Cancer Res. Center, Neuenheimer Feld 280, 6900 Heidelberg, W. Germany); Banasch, P.; Mattern, J.; Volm, M. *J Natl Cancer Inst* 62(5): 1199-1207; 1979.

A study was made of the morphology and morphogenesis of vascular liver tumors induced in the multimammate mouse *Mastomys (Praomys) natalensis* by dimethylnitrosamine (DMNA: single dose of 10 mg/kg or 2 doses of 5 mg/kg, ip). Benign hemangioendotheliomas (HET's) developed in 164/173 rodents, malignant HET's in 10/173. There were no

differences associated with number of injections or interval between injections (1-7 days), but males reacted to the carcinogen somewhat earlier and more strongly than females. The first histologic alterations were focal dilatations of the sinusoids. The benign HET's that subsequently developed were characterized by solitary or multiple distended blood lacunae, and they resembled peliosis hepatis macroscopically. The lacunae were frequently thrombosed. The coating endothelium appeared only slightly altered. In advanced stages, entire liver lobes could be affected in the form of a diffuse hemangioendotheliomatosis, and in some animals the histologic picture largely corresponded to that of cavernous hemangioma in humans. Rupture of the HET's often gave rise to hemorrhaging into the abdominal cavity. Unequivocal hemangiosarcomas were found in 10 animals. In more advanced stages, the tumor cells displaced the liver parenchyma, and metastases were observed to the lung in one animal and to the adrenal cortex in another. It is possible that sinus dilatations, benign HET's, and hemangiosarcomas are only different stages of a uniform pathogenetic series. (52 refs)

79-2607 Induction of 6-Thioguanine Resistance in Chinese Hamster Lung Cells Treated with Dimethylnitrosamine, 2-Aminoanthracene or 7,12-Dimethylbenz(a)anthracene in the Presence of Rat Liver Microsomes. (Eng) Huang, S. L. (Northrop Services, Inc., Environmental Sciences Group, P.O. Box 12313, Research Triangle Park, NC, 27709); Whong, W. Z. *Toxicol Lett* 3(4): 209-214; 1979.

The mutagenicity of dimethylnitrosamine [DMN (5-100 mM)], 2-aminoanthracene [AA (25-200 µg/ml)], and 7,12-dimethylbenz(a)anthracene [DMBA (25-200 µg/ml)] was studied at the hypoxanthine guanine phosphoribosyl transferase (HGPRT) locus in Chinese hamster lung cells. These compounds induced 6-thioguanine resistance in the presence of rat liver microsomes, but not in their absence. The frequencies of mutations induced by these compounds were dose-dependent. (18 refs)

79-2608 The Percutaneous Absorption of N-Nitrosodietanolamine Through Excised Human Skin (Meeting Abstract). (Eng) Bronaugh, R. L. (Div. Toxicology, Food and Drug Admin., Washington, DC); Congdon, E. R.; Scheuplein, R. J. *J Invest Dermatol* 72(4): 204; 1979. (no refs)

79-2609 The Percutaneous Absorption of N-Nitrosodietanolamine Through Excised Human Skin (Meeting Abstract). (Eng) Bronaugh, R. L. (Div. Toxicology, Food and Drug Admin., Washington, DC); Congdon, E. R.; Scheuplein, R. J. *Clin Res* 27(2): 522A; 1979. (no refs)

79-2610 Dedifferentiation of Liver and Pancreas Induced by Chemical Carcinogens (Meeting Abstract). (Eng) Bockman, D. E. (Dept. Anatomy, Medical Coll. Georgia, Augusta, GA); Black, O.; Webster, P. D. *Gastroenterology* 76(5, part 2): 1104; 1979. (no refs)

79-2611 Carcinogenic Effects of Di(2-hydroxypropyl)nitrosamine (DHPN) in Male Wistar Rats: Promotion of Pancreatic Cancer by a Raw Soya Flour Diet. (Eng) Levison, D. A. (Dept. Pathology, Univ. Dundee, Dundee, Scotland); Morgan, R. G.; Brimacombe, J. S.; Hopwood, D.; Coghill, G.; Wormsley, K. G. *Scand J Gastroenterol* 14(2): 217-224; 1979.

The effect of raw soya flour (RSF) on the carcinogenicity of di(2-hydroxypropyl)nitrosamine (DHPN, 0.2 mg/wk ip for 4 wk and then 0.1 mg/wk for 20 wk) in the pancreas was studied in four groups of male Wistar rats (16 rats/group). DHPN-treated and untreated rats were given a diet of RSF or heated (control) soya flour (HSF). Pancreatic wts were significantly increased in rats fed RSF compared with those of rats fed HSF; DHPN significantly reduced this hypertrophy. Zymogen-containing nodules with large pleomorphic nuclei and many mitotic figures were observed in the pancreases of rats given RSF, RSF + DHPN, and, to a lesser extent, HSF + DHPN. Among the other tumors found in rats given RSF + DHPN or HSF + DHPN were invasive squamous cell carcinomas of the esophagus (4 and 4, respectively), intestinal adenocarcinomas (2 and 4), hepatic lesions (3 and 4) and lung cancers (5 and 2). It is possible that dietary factors that result in the excessive and sustained release of cholecystokinin may sensitize the pancreas to environmental carcinogens. The number of rats with microscopic hyperplastic pancreatic nodules were 4/16, 15/16, 6/16, and 13/13 (only 13 were suitable for morphological study) in the HSF, RSF, HSF + DHPN, and RSF + DHPN groups, respectively. The corresponding figures for mean number of nodules/section of pancreas were <1, 4, <1, and 8; for mean area occupied by nodules/section of pancreas <2%, 10%, <2%, and 25%; and for number of rats with pancreatic carcinoma, 0/16, 0/16, 0/16, and 2/16 (these tumors were mucin-producing invasive adenocarcinomas). Ten pancreases in the RSF + DHPN group showed macroscopic nodules after 9 mo; two contained, in addition, adenomatous acinar cell nodules, and one contained an adenoma apparently arising from ductal epithelium. (19 refs)

79-2612 Intestinal Tumors Induced by a Single Intraperitoneal Injection of Methyl(acetoxymethyl)nitrosamine in Three Strains of Rats. (Eng) Berman, J. J. (Lab. Experimental Pathology, NCI, Bethesda, MD, 20014); Rice, J. M.; Wenk, M. L.; Roller, P. P. *Cancer Res* 39(5): 1462-1466; 1979.

To determine whether the strain of rat determines the quantity or type of intestinal tumors induced by methyl(acetoxymethyl)nitrosamine (DMN-OAc), 5-wk-old male and female rats of the Sprague-Dawley (SD), Buffalo (BUF), and Fischer (F344) strains were given a single ip injection of DMN-OAc. The dose used, 13 mg (0.1 micromole)/kg, was one-half of the acute ip median lethal dose determined for 5-wk-old male SD rats. In each strain treated with DMN-OAc, a large number of intestinal epithelial tumors developed. The histological features of these tumors were the same in all strains and both sexes. In each strain, females had fewer induced tumors of the small intestine (SI) and large intestine (LI) than males of the same strain. The greatest difference between males and females in the frequency of SI tumors induced was observed in F344 rats. These were 1.7 SI tumors/male F344 rat and only 0.2 SI tumor/female F344 rat. The lowest induced tumor incidence was seen in BUF rats (0.3 SI tumor/male rat and 0.2 SI tumor/female rat). In SD rats, there were 2.0 SI tumors/male and 1.3 SI tumors/female. In all strains and both sexes, many more SI tumors were induced than LI tumors. Tumors occurred throughout the SI and colon and in the cecum, and they tended to occur most frequently in the distal ileum. Localization was the same in all strains and sexes studied. Other tumors induced with high frequency by ip administration of DMN-OAc were schwannomas (primary in peritoneum), testicular mesotheliomas, and splenic angiosarcomas. (13 refs)

79-2613 Cumulative Catalytic Effects of Some Physiologically Active Ions on the Rate of Nitrosation of N-Methylaniline In Vitro. (Eng) Lathia, D. (Dept. Nutrition Physiology, Fachhochschule Niederrhein, Richard-Wagnerstrasse 101, 405 Monchengladbach, W. Germany); Rutten, M. *Nutr and Cancer* 1(2): 19-22; 1979.

The effect of physiological concentrations of thiocyanate and/or iodide ions on the in vitro formation of N-methyl-N-nitrosoaniline (NMNA) was studied. The amount of NMNA formed from 0.1 mM N-methylaniline and 0.1 mM potassium nitrite in the presence of 0.5 mM potassium thiocyanate (KSCN) increased 700% compared with the control reaction. Even at the lowest thiocyanate concentration tested (0.005 mM), the effect was considerable. However, the increase in NMNA formation was remarkably higher in the presence of iodide ions: the total percentage increase of NMNA in the presence of 0.015 mM potassium iodide was 240% higher than that in the presence of 0.025 mM KSCN. Tests of the combined effect of thiocyanate and iodide ions on in vitro NMNA formation gave extremely high yields, indicating a cumulative effect. Thiocyanate levels of 0.545 and 1.82 mM have been detected in the saliva of smokers and nonsmokers, respectively. The present results indicate a relatively high rate of NMNA formation in the presence of 0.5 mM thiocyanate. In addition, data are presented that demonstrate that the iodide concentration in human gastric juice could be sufficient to catalyze the nitrosation reaction significantly. (14 refs)

- 79-2614** **ENU-induced Granular Oligodendroglioma in the Rat (Meeting Abstract).** (Eng) Liwnicz, B. H. (Univ. Cincinnati Coll. Medicine, Cincinnati, OH); Mandybur, T. I.; Alvira, M. *J Neuropathol Exp Neurol* 38(3): 329; 1979. (2 refs)

- 79-2615** **The Development of Experimental Brain Tumours: A Sequential Light and Electron Microscope Study of the Subependymal Plate. II. Microtumours.** (Eng) Pilkington, G. J. (Dept. Neurological Studies, Middlesex Hosp. Medical Sch., London W1P 8AA, England); Lantos, P. L. *Acta Neuropathol (Berl)* 45(3): 177-185; 1979.

The cytoarchitecture of the microtumors induced in the rat brain by transplacental administration of N-ethyl-N-nitrosourea (ENU) is described, and the relevance of this experimental model to human neoplasia is assessed. Pregnant BD-IX rats were given a single ip injection of 30 mg/kg ENU on gestation day 15. The offspring were killed at fortnightly intervals between 2 and 20 wk of age. The subependymal plate region adjacent to the lateral ventricles was examined by light and electron microscopy to study the early stages of tumor development. Microtumors, composed of subependymal plate cells, glioblasts, and various glial cells at different stages of maturation, were found in 16-, 18-, and 20-wk-old rats. The most common site for microtumors was the angle of the lateral ventricles between the corpus callosum and caudate nucleus; others were located at the lateral aspect of the ventricles. It is suggested that most, if not all, cerebral gliomas originate from the undifferentiated cells of the subependymal plate: these mitotically active stem cells provide a susceptible target for the carcinogenic stimulus. The morphology of the gliomas is determined by the diverging processes of differentiation and anaplasia, resulting in a pleomorphic cell population. Sequential studies of ENU-induced gliomas may contribute to an understanding of the pathogenesis of gliomas in humans. (22 refs)

- 79-2616** **The Development of Experimental Brain Tumours: A Sequential Light and Electron Microscope Study of the Subependymal Plate. I. Early Lesions (Abnormal Cell Clusters).** (Eng) Lantos, P. L. (Dept. Neurological Studies, Middlesex Hosp. Medical Sch., London W1P 8AA, England); Pilkington, G. J. *Acta Neuropathol (Berl)* 45(3): 167-175; 1979.

The development of transplacentally induced brain tumors in rats is described, and the role of subependymal plate cells in their origin is elucidated. Pregnant BD-IX rats were given a single ip injection of 30 mg/kg N-ethyl-N-nitrosourea on gestation day 15. The offspring were killed at fortnightly intervals between 2 and 20 wk of age. The subependymal plate region adjacent to the lateral ventricles was examined by light and electron microscopy to study the early stages in the development of brain tumors. Abnormal clusters of undifferentiat-

ed subependymal plate cells were found in regions away from the plate from 8 wk of age onward. In addition, focal cellular hyperplasia within the subependymal plate was seen. The clusters, which often occurred around neurones and blood vessels, were thought to represent the earliest morphologically detectable changes in the development of cerebral gliomas. (28 refs)

- 79-2617** **Paper Chromatographic Analysis of the In Vitro Effect of Nitrosoureas on DNA Composition in the Presence of Lipids.** (Ukr) Danilenko, I. I. (Inst. Epidemiology, Kiev, USSR). *Dopov Akad Nauk Ukr RSR [B]* (1): 52-55; 1979.

DNA specimens isolated from *Salmonella typhimurium* were incubated with lipids (2 mg/ml) and then treated with methylnitrosourea, dimethylnitrosourea, ethylnitrosourea, and nitrosoguanidine. The nucleotide composition of DNA was assessed by paper chromatography. It was found that regardless of the type of mutagen, the presence of lipids increased the alkylation of adenine. (15 refs)

- 79-2618** **Molecular Properties of Steroid-Binding Proteins in Rat Mammary Tumors Induced by N-Nitrosomethylurea (Meeting Abstract).** (Eng) Lewko, W. M. (Dept. Biochemistry, Univ. Louisville Health Sciences Center, Louisville, KY); Cassidy, C. S.; Wittliff, J. L. *J Toxicol Environ Health* 4(2/3): 487-488; 1978. (2 refs)

- 79-2619** **Morphologic and Biochemical Characteristics of Transplantable Neurogenic Tumors Induced by N-Ethyl-N-nitrosourea in Inbred BD IX Rats.** (Eng) Wechsler, W. (Medizinische Einrichtungen, Universität Dusseldorf, Neuropathologisches Institute, Moorenstrasse 5, D-5000 Köln 91, W. Germany); Ramadan, M. A.; Pfeiffer, S. E. *J Natl Cancer Inst* 62(4): 811-817; 1979.

The morphologic and biochemical parameters of three experimental rat tumors, one glioma and two neurinomas, were evaluated during long-term serial transplantation experiments. The tumors were induced transplacentally in inbred BD IX rats by systemic application of N-ethyl-N-nitrosourea (80 mg/kg iv on gestation day 15). Because primary gliomas and neurinomas produced in this way are composed of heterogeneous cell populations, changes in tumor morphology are expected to occur during serial transplantation in syngeneic hosts. These changes were correlated with the expression of two biochemical nervous system markers, S-100 protein and 2',3'-cyclic nucleotide 3'-phosphohydrolase. Several changes were observed during serial transplantation, including increased growth rate (even after 1 passage), preferential growth of anaplastic vs differentiated glial and Schwann's cells, varying degrees of fibrosarcomatous changes

after prolonged serial transplantation, and reduced levels of S-100 protein. In contrast, tumors derived from biochemically differentiated clonal cell lines retained their morphologic and biochemical characteristics to a much greater extent, even after prolonged periods of sc transplantation. (43 refs)

79-2620 Plant Sterols: Role in Large Bowel Cancer (Meeting Abstract). (Eng) Raicht, R. F. (Dept. Medicine, Veterans Admin. Medical Center, New York Univ. Sch. Medicine, New York, NY); Cohen, B. I.; Sarwal, A.; Takahashi, M.; Fazzini, E. *Gastroenterology* 76(5, part 2): 1296; 1979. (no refs)

79-2621 Assessment of Sub-Clinical Zinc-Deficiency for Carcinogenetic and Other Long-Term Studies in Rats. (Eng) Mathur, A. (Dept. Oral Surgery, Faculty Odontology, Univ. Lund, Malmo, Sweden); Wallenius, K.; Abdulla, M. *Arch Oral Biol* 23(12): 1077-1082; 1978.

To determine the influence of zinc on po carcinogenesis, 3-wk-old female Sprague-Dawley rats were fed 0.09 (Group I), 0.77 (Group II), or 3.98 (Group III) micromoles of Zn/kg, and, at 6 wk, were painted 3x/wk with the water-soluble carcinogen 4-nitroquinoline N-oxide. Controls were painted with propylene glycol solvent along. There were no significant differences in wt gain or food consumption between the experimental and control groups, and no animal showed evidence of macroscopic cancer. After 3 wk, the plasma Zn level in Group I was significantly lower than that in Groups II and III, but the levels in Group I later increased to those in Group II. The Cu:Zn ratio in the plasma was significantly higher in Group I than in Groups II and III, and the ratio in Group II was nonsignificantly higher than that in Group III. The liver Zn concentration was always significantly lower in Group I than in Groups II and III. There were no significant differences between the experimental and control animals in each group with respect to the levels of Zn in the plasma or liver or the plasma Cu:Zn ratio. The results suggest that after prolonged intake of the Zn-deficient diet, the plasma Zn level does not accurately reflect the Zn intake and that the Zn concentration in the liver is most affected. The subnormal Zn diet caused subclinical Zn deficiency that could be assessed by laboratory tests. (24 refs)

79-2622 Effect of Storage of Ethyl Methanesulphonate & Hydroxylamine Treated Seeds on Mutation Frequency in Barley *Hordeum vulgare*. (Eng) Singh, R. M. (Dept. Genetics & Plant Breeding, Faculty Agriculture, Banaras Hindu Univ., Varanasi 221 005, India); Agrawal, P. K.; Singh, J.; Singh, B. D.; Singh, R. B. *Indian J Exp Biol* 16(10): 1067-1069; 1979.

Barley seeds were treated with 0.03 M ethyl methanesulphonate and/or 0.08 M hydroxylamine for 6 hr and then planted immediately or stored for 7 or 14 days. Storage reduced germination rate, seedling height, and field survival to some extent, but pollen and ovule fertility were not affected appreciably. However, storage reduced the frequency and spectrum of chlorophyll and viable mutations markedly. Combination treatments had a synergistic effect on mutation induction. (10 refs)

79-2623 Exploratory Monitoring of Air Pollutants for Mutagenicity Activity with the *Tradescantia* Stamen Hair System. (Eng) Schairer, L. A. (Biology Dept., Brookhaven Natl. Lab., Upton, NY, 11973); Van't Hof, J.; Hayes, C. G.; Burton, R. M.; de Serres, F. J. *Environ Health Perspect* 27: 51-60; 1978.

Use of the *Tradescantia* stamen hair genetic system, originally developed for studying the effects of ionizing radiation, for environmental mutagen detection is described. Exposures to the air pollutants SO₂, NO₂, and O₃ and to vapors of mutagens such as 1,2-dibromoethane (DBE) and ethyl methanesulphonate (EMS) demonstrated the usefulness of the system as a detector of chemical mutagens. A significant number of phenotypic changes occurred following exposures to as little as 0.14 ppm DBE. Max sensitivity was obtained with long-term or chronic exposures, because the response increased linearly with duration of exposure up to 21 days. A mobile laboratory designed to support plant culture in the field was used to monitor industrial sites in several states. Environment-controlled growth chambers were installed in a trailer so that both ambient air fumigations and concurrent clean air control exposures could be made. Atmospheric contaminants from petroleum and chemical processing plants generated a significant number of phenotypic pigment changes that were 17%-31% above the control levels; contaminants from steel and copper smelters, automotive combustion products, and photochemical compounds were negative. The *Tradescantia* stamen hair system encompasses the cytogenetic and somatic potential to make it a useful tool for mutagenicity monitoring of ambient air pollution mixtures. (15 refs)

79-2624 A Human Diploid System for Detection of Environmental Mutagens: Ethylmethanesulphonate (EMS) Induction of Methotrexate and Ricin Resistant Mutants (Meeting Abstract). (Eng) Polin, R. A. (Univ. Pennsylvania Sch. Medicine, Philadelphia, PA); Kennett, R. *Pediatr Res* 13(4, part 2): 438; 1979. (no refs)

79-2625 Gastric Carcinoma in Dogs Produced by the Combined Use of N-Ethyl-N'-nitro-N-nitrosoguanidine (ENNG) and Gastrin: With Special Refer-

ence to Development of Scirrhus Carcinoma. (Eng) Kurihara, M. (Dept. Gastroenterology, Juntendo Univ., 3-1-3 Hongo, Bunkyo-ku, Tokyo); Shirakabe, H.; Yamaya, F.; Miyasaka, K.; Maruyama, T.; Izumi, T.; Yasui, A.; Kamano, T. *Acta Pathol Jpn* 29(2): 171-176; 1979.

An attempt was made to induce scirrhus carcinoma of the stomach in eight 4-mo-old beagle dogs by the combined administration of N-ethyl-N'-nitro-N-nitrosoguanidine (ENNG) and gastrin. Two dose regimens were used. (1) Four dogs were given 500 ml/day of a 150- μ g/ml ENNG soln po for experimental months (EM) 1-8 and, after a 1-mo interval, 500 ml/day of a 200- μ g/ml soln for 2.5 mo; starting at EM 5, the dogs were inoculated sc with gastrin at a dose of 5 μ g/kg 2x/wk for 4 mo, 10 μ g/kg/day for 2 mo. (2) Four dogs were given 500 ml/day of the 150- μ g/ml ENNG soln for 1 mo, 500 ml/day of the 200 μ g/ml soln for EM 2-8; starting at EM 5, they were inoculated with gastrin at a dose of 5 μ g/kg/day for 1.5 mo, 10 μ g/kg/day for 5 mo. One of four dogs in the first group and two of four in the second group developed gastric carcinoma. One of these three dogs had an annularly infiltrating advanced carcinoma with marked fibrous thickening of the antral wall that resulted in stenosis of the antrum (carcinoma scirrhus). This lesion resembled *linitis plastica* (Bormann's Type IV carcinoma) in the human stomach. Histological examination showed a signet ring cell carcinoma that involved the submucosal, muscular, subserosal, and serosal layers. The foci of gastric carcinoma in the other two dogs did not differ macroscopically or histologically from those produced by ENNG alone. (7 refs)

79-2626 Neuroendocrine Cells in Serially Passaged Rat Stomach Cancers Induced by MNNG. (Eng) Kobori, O. (1st Dept. Surgery, Faculty Medicine, Univ. Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113, Japan); Oota, K. *Int J Cancer* 23(4): 536-541; 1979.

Five gastric carcinomas, induced in inbred Wistar rats by po administration of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG: 80 mg/liter drinking water for 31 wk), were successfully transplanted to isologous rats. The transplants grew to a size of 10-35 mm in diameter within 8-25 wk of implantation. In one case, serial transplantations were maintained up to the 11th generation, with the occurrence of distant metastasis in the 3rd generation. Histological, histochemical, and ultrastructural comparisons of the original and transplanted tumors revealed that (1) the original tumors were quite well-differentiated, forming papillary or tubular structures, whereas the transplants were more anaplastic and pleomorphic, often showing solid nests; and (2) tumor cells with gastrointestinal differentiation and cells with neuroendocrine differentiation were present and evenly distributed in both the original and the serially transplanted tumors. As it is unlikely that normal and neoplastic neuroendocrine cells were growing side-by-side with and independently of the epithelial neoplastic components, the findings strongly suggest the multidirectional potency of inbred rat stomach carcinoma cells and

the common neoplastic origin of the epithelial and neuroendocrine components. (15 refs)

79-2627 Establishment of a Permanent Cell Line from Gastric Carcinoma Induced by N-Methyl-N'-nitro-N-nitrosoguanidine in the Rat (Meeting Abstract). (Fre) Kobori, O. (Laboratoire d'Immunologie et de Medecine Experimentale, Faculte de Medecine, boulevard Jeanne-d'Arc, F 21033 Dijon, France); Martin, F.; Martin, M. S.; Turc, C. *Gastroenterol Clin Biol* 3(1): 86; 1979. (no refs)

79-2628 Potentiation by Glucocorticoids of the Effects of N-Nitroso Carcinogens on the Tyrosine Aminotransferase Induction and DNA Synthesis in Cultured Rat Liver Cells (Meeting Abstract). (Eng) Hoshino, J. (Abt. Biochemie, Robert Koch-Institut, Nordufer 20, D-1000 Berlin 65, W. Germany); Kroger, H. *Hoppe Seylers Z Physiol Chem* 360(3): 286; 1979. (no refs)

79-2629 Measurement of Mutagenesis in Mammalian Cells. (Eng) Waldren, C. (Eleanor Roosevelt Inst. Cancer Res., Univ. Colorado Medical Center, 4200 E. Ninth Ave., Denver, CO, 80262); Jones, C.; Puck, T. T. *Proc Natl Acad Sci USA* 76(3): 1358-1362; 1979.

An in vitro method for measuring mutagenesis by environmental agents uses stable human-Chinese hamster ovary hybrid cells that have retained a single human chromosome not necessary for cell reproduction. Forward mutations are detected in genes necessary for production of specific human cell-surface antigens. Both mutants form colonies in the presence of specific antisera and complement that destroy the unmutagenized cells. The method is illustrated by tests of X-irradiation, N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), and caffeine. When expressed in terms of number of mutants per 10^4 cells per LD₅₀, MNNG was 8x more potent than X-rays in causing limited marker loss but roughly similar in its ability to cause loss of all or most of the chromosome. Caffeine was without measurable effect. The method permits assessment of lesions that cause loss of all or most of the chromosome as well as various localized gene mutations. The use of this test may help prevent exposure to agents important in cancer and in genetic diseases. (37 refs)

79-2630 The Effect of Bile Acids on Drug Metabolism. (Eng) Kawalek, J. C. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD, 21501). *Nutr and Cancer* 1(2): 13-18; 1979.

The effects of cholic acid (CA), chenodeoxycholic acid

(CDC), deoxycholic acid (DOC), and lithocholic acid (LA) on rat microsomal hydroxylases, epoxide hydrolase (EH), and glutathione S-transferase (GSH S-transferase) were studied. In most cases, the bile acids inhibited the microsomal hydroxylase activity, the inhibition being greatest with DOC and varying with substrate, bile acid, and the liver microsome (S9) preparation used for metabolic activation. In one test, LA caused a steady increase in enzymatic activity. EH activity was slightly stimulated by DOC and CA, initially stimulated then markedly decreased by LA, and markedly decreased (50%) by CDC. GSH S-transferase was inhibited by DOC, LA, and CDC and weakly inhibited by CA. These results demonstrate that the presence of bile acids could affect the metabolism of endogenous substrates, carcinogens, and other xenobiotics. (35 refs)

- 79-2631 Effect of Cholesterol Metabolites and Promoting Effect of Lithocholic Acid in Colon Carcinogenesis in Germ-free and Conventional F344 Rats.** (Eng) Reddy, B. S. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY, 10595); Watanabe, K. *Cancer Res* 39(5): 1521-1524; 1979.

The colon tumor-promoting activity of sodium lithocholate (SLC), cholesterol, cholesterol-5 α ,6 α -epoxide (cholesterol epoxide: CE), and cholestane-3 β ,5 α ,6 β -triol (triol) was studied in female Fischer (F-344) conventional and germ-free rats. The germ-free animal system would indicate whether further modification of these compounds by gut microflora is required for tumor promotion. At 7 wk of age, groups of germ-free and conventional rats were given intrarectal instillations of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG: 2.5 mg/rat 2x/wk for 2 wk) and then intrarectal doses of SLC, cholesterol, CE, or triol (20 mg/rat 3x/wk for 46 wk). Other groups received MNNG for 2 wk and vehicle thereafter for 46 wk or SLC or cholesterol metabolites alone for 48 wk. No tumors were detected in rats given SLC, cholesterol, CE, or triol alone, which suggests that these compounds, or their microbially modified metabolites, were not carcinogenic to colon mucosa under the experimental conditions used. SLC increased MNNG-induced colon tumor incidence in both germ-free and conventional rats. The colon tumor incidence was similar in the MNNG, MNNG + cholesterol, MNNG + CE, and MNNG + triol groups. The results obtained in germ-free rats also indicate that SLC, without being further modified by the gut microflora, acts as a colon tumor promoter. It is concluded that SLC, but not cholesterol, CE, triol, or their microbial products, has a promoting effect on MNNG-induced colon carcinogenesis in rats. (32 refs)

- 79-2632 Response of Two Rodents, *Mastomys natalensis* and *Myiostomys albicaudatus*, to the Pancreatic Carcinogen Azaserine.** (Eng) Roebuck, B. D. (Dept. Pharmacology and Toxicology, Dartmouth Medical Sch., Hanover, NH, 03755); Longnecker, D. S. *J Natl Cancer Inst* 62(5): 1269-1271; 1979.

Two rodent species, *Mastomys natalensis* and *Myiostomys albicaudatus*, were tested for responsiveness to the pancreatic carcinogen azaserine (AS). The animals (aged 28 days) were treated with 30 mg/kg AS ip 1x/wk for 5 wk. They were killed 6 mo after the first AS treatment, and the pancreas was examined for pathological changes. *Myiostomys* was not responsive to AS treatment, but *Mastomys* was the most responsive rodent tested to date. Male *Mastomys* had 84 atypical acinar cell nodules (AACN)/g pancreas, and the females had 75 AACN/g pancreas. In addition to large numbers of AACN in *Mastomys*, 2/4 male animals had acinar cell adenomas. One of these two animals had a circumscribed tumor with marked anaplasia that was regarded as an early carcinoma or a carcinoma in situ. In previous studies, male Wistar rats had 51 AACN/g pancreas and the females had 20 AACN/g pancreas. (8 refs)

- 79-2633 Neuraminidase Effect on the Growth of a Transplantable Nickel Sulfide-induced Rat Tumor.** (Eng) Abandowitz, H. M. (Dept. Biomedical Sciences, Univ. Guelph, Guelph, Ontario, Canada). *Jpn J Med Sci Biol* 31(5/6): 421-424; 1978.

The immunostimulatory potential of a weakly immunogenic nickel sulfide induced fibrosarcoma was determined after treatment of the tumor cells with *Vibrio cholerae* neuraminidase (VCN). The cells were exposed to 50 IU VCN/5 x 10⁶ cells/ml phosphate-buffered saline at 37 C for 30 min. Then, 1 x 10⁶ viable cells were inoculated sc in the leg of two groups of four highly inbred Fischer (F344) rats (8-10 wk old). The VCN-tumor-immunized rats, which remained free of palpable tumors for 32 days, were rechallenged sc in the opposite leg with 5 x 10⁵ freshly harvested viable untreated tumor cells. Administration of VCN-treated tumor cells rendered 50% of the rats resistant to a subsequent challenge with untreated viable tumor cells. When tumors did appear in two VCN-tumor-immunized rats, their time of appearance was delayed by 8 and 11 days and their growth was retarded (12.9 mm in immunized rats, compared with 30.5 mm in control rats). Tumors were palpable in all control rats 4 days after injection of untreated tumor cells. These findings provide preliminary evidence of the potential of VCN enhancement in tumor immunogenicity. (13 refs)

- 79-2634 Basal Cell Carcinoma of the Scrotum in a Former Employee of a Japanese Mustard Gas Factory.** (Jpn) Inada, S. (Dept. Dermatology, Hiroshima Univ. Sch. Medicine, Kasumi, Hiroshima 734, Japan); Yamura, T.; Takiyama, W.; Kamitsuna, A.; Nishimoto, Y. *Jpn J Cancer Clin* 25(1): 67-70; 1979.

In 1977, a basal cell carcinoma of the scrotum was diagnosed in a 72-yr-old man who had worked in a mustard gas factory from 1933 to 1945. He had pleuritis at age 28 and chronic bronchitis at age 62 as a result of exposure to mustard gas;

a slightly differentiated adenocarcinoma of the stomach was resected at age 71. During the patient's employment at the poison gas factory, mustard gas permeated through his protective clothing and often caused acute sinusitis, bronchitis, and dermatitis in areas where perspiration collected. In 1974 and 1975, two of the hyperkeratotic eruptions that occurred on the skin of the patient's trunk and lower extremities were diagnosed histopathologically as Bowen's disease. The mustard gas was considered to be the cause of the Bowen's disease and the basal cell carcinoma. (34 refs)

- 79-2635 Epidermal Carcinogenicity of Bis(2,3-epoxycyclopentyl)ether, 2,2-Bis(p-glycidyloxyphenyl)propane, and m-Phenylenediamine in Male and Female C3H and C57BL/6 Mice.** (Eng) Holland, J. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Gosslee, D. G.; Williams, N. J. *Cancer Res* 39(5): 1718-1725; 1979.

The systemic toxicity and skin carcinogenicity of bis(2,3-epoxycyclopentyl)ether (BECE), 2,2-bis(p-glycidyloxyphenyl)propane (BGOP), and m-phenylenediamine (PD) were compared with those of benzo(a)pyrene by application to the skin of male and female C3H and C57BL/6 mice 3x/wk for 24 mo. BECE and BGOP were also applied as an equal parts mixture to determine whether the materials would interact as skin carcinogens. A comparison of dose responses showed that benzo(a)pyrene was 107×10^3 , 161×10^3 , and 51×10^3 more potent as a skin carcinogen than BECE, BGOP, and their mixture, respectively. PD was not carcinogenic in the skin of either strain at the max exposure rate allowed by systemic toxicity. The two inbred mouse strains used exhibited a constant ratio of sensitivity to the induction of epidermal cancer by diverse chemical agents. Statistical analysis of this difference for various compounds at different dose rates revealed that C57BL/6 mice were 2.4 times more sensitive to epidermal carcinogenesis than C3H mice under identical circumstances of exposure. It is suggested that when carcinogenicity is assessed on a relative basis, the observed constancy in the response ratio would enable these two inbred strains to be used interchangeably. The choice of strain would affect the sensitivity of the assay significantly, but this would become important only in the assay of extremely weak carcinogens. (17 refs)

- 79-2636 The Role of Dietary Fiber in Colon Tumorigenesis Induced by Dimethylhydrazine in the Rat (Meeting Abstract).** (Eng) Barbolt, T. A. (Albany Medical Coll., Union Univ., Jackson, TN, 38301). *Diss Abstr Int [B]* 39(9): 4282-4283; 1979. (no refs)

- 79-2637 Induction of Sister-Chromatid Exchanges (SCE) in Human Leukocytes by Cyclic Nitroso-Compounds (Meeting Abstract).** (Eng) Ho, T. (Biology

Div., Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Tipton, S. C.; Epler, J. L. *In Vitro* 15(3): 209; 1979. (no refs)

- 79-2638 Hodgkin's Disease as a Complication of Crohn's Disease.** (Eng) Hecker, R. (Gastroenterology Unit, Royal Adelaide Hosp., Adelaide, Australia 5000); Sheers, R.; Thomas, D. *Med J Aust* 2(13): 603; 1978.

The case report of a 32-yr-old man who developed intestinal Hodgkin's disease in association with a 10-yr history of colorectal Crohn's disease is presented. Although the association could have been coincidental, the history of Crohn's disease, the presence of histocompatibility antigens HLA-A1 and HLA-B5, and 5 yr of exposure to azathioprine could have been involved. Hodgkin's disease (mixed cellularity type) involved the celiac nodes, paracolic nodes, and the sites of three skip lesions in the ileum, transverse colon, and rectosigmoid junction. (5 refs)

- 79-2639 Reticulum Cell Sarcoma in Azathioprine-treated Systemic Lupus Erythematosus.** (Eng) Hehir, M. E. (Royal Postgraduate Medical Sch., London, England); Sewell, J. R.; Hughes, G. R. *Ann Rheum Dis* 38(1): 94-95; 1979.

The development of a reticulum cell sarcoma in the lung of a 43-yr-old woman who had received 18 mo of azathioprine treatment for systemic lupus erythematosus (SLE) is described. Only two other cases of this association have been reported. Tumor induction may be a possible hazard of immunosuppressive therapy in SLE patients. (4 refs)

- 79-2640 Vasopressin Stimulation of Mouse 3T3 Cell Growth.** (Eng) Rozengurt, E. (Imperial Cancer Res. Fund, P. O. Box 123, Lincoln's Inn Fields, London WC2A 3PX, England); Legg, A.; Pettican, P. *Proc Natl Acad Sci USA* 76(3): 1284-1287; 1979.

In tests conducted to determine whether substances that promote Na⁺ entry can be mitogenic for quiescent cells, vasopressin was shown to be a potent mitogen for Swiss mouse 3T3 cells. The hormone [1-10 nanograms (ng)/ml] caused a striking shift of the dose-response curve for the effect of serum on thymidine incorporation by cultures of 3T3 cells arrested in the G₁/G₀ phase of the cell cycle. In the absence of added serum, the effect of vasopressin on DNA synthesis was greatly potentiated by insulin, epidermal growth factor, and a factor isolated from medium conditioned by simian virus 40-infected baby hamster kidney cells. The mitogenic effect of vasopressin was dependent on time and hormone concentration. In the presence of insulin, the half-maximal effect elicited by the peptide was obtained at 0.6 ng/ml. [Arg]vasopressin and [Lys]vasopressin were equally potent.

The vasopressins were 10^3 -fold more potent than oxytocin. In the presence of a low (2.5%) concentration of serum, the vasopressins stimulated cell proliferation. (21 refs)

- 79-2641 Dissociation of α -Macrofetoprotein and α -Fetoprotein Production During Experimental Injury.** (Eng) Hudig, D. (Dept. Medicine M-013, Univ. California, San Diego, La Jolla, CA, 92093); Sell, S.; Newell, L.; Smuckler, E. A. *Lab Invest* 40(2): 134-139; 1979.

The concentrations of two major fetal serum proteins of the rat, α -macrofetoprotein (AMF) and α -fetoprotein (AFP), were measured following administration of croton oil (0.1 ml injected into a hind footpad), carbon tetrachloride (0.35 ml x 2 ip, or single dose of 25 μ g/100 g po), galactosamine (200-800 mg/kg ip, 1 or 2 doses), or ethionine (0.64 nanomole/100 g ip) and after 70% partial hepatectomy. The greatest elevations in AMF occurred in animals given croton oil or ip CCl_4 and after partial hepatectomy. These concentrations were similar to max fetal rat concentrations at 20 days of gestation. The greatest AFP concentration was seen after partial hepatectomy, 800 mg/kg galactosamine, and po CCl_4 . These concentrations were well below those of 20-day rat fetuses. SGOT values, which are indicative of the degree of lytic hepatic injury, were greatest in animals given po CCl_4 and 800 mg/kg galactosamine. When both AMF and AFP concentrations were elevated, AMF appeared to be an acute-phase reactant, whereas AFP elevations occurred later and were associated with hepatic proliferation. Apparently, the production of AMF and AFP is under completely independent regulation in the adult rat. (36 refs)

- 79-2642 Effects of the Co-mutagen Norharman in Rats.** (Eng) Murasaki, G. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan); Miyata, Y.; Shirai, T.; Arai, M.; Kawachi, T.; Ito, N. *Toxicol Lett* 3(3): 157-161; 1979.

The acute and subacute toxic effects of the comutagen norharman (NH) were determined in male Fischer rats. Animals were fed diets containing 1,500 ppm NH for 28 days, and 3 or 6 animals were killed for examination on days 0, 1, 3, 7, 14, 21, and 28. During NH administration, the body wt and food and water intakes of the rats decreased gradually, but kidney wt and blood urea nitrogen levels increased significantly (the latter was 107.3 mg/dl on day 28). NH caused a segmental coagulative necrosis of the tubular epithelium of the kidney that increased in severity and extent with the duration of treatment, but glomerular changes were negligible. In the testis, the germinal epithelium showed decreased spermatogenic activity after day 7. NH had no detectable effects on other organs. (9 refs)

- 79-2643 Effects of Cannabinoids on Host Resistance to *Listeria monocytogenes* and Herpes Simplex**

Virus. (Eng) Morahan, P. S. (Dept. Microbiology, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA, 23298); Klykken, P. C.; Smith, S. H.; Harris, L. S.; Munson, A. E. *Infect Immun* 23(3): 670-674; 1979.

An attempt was made to determine whether cannabinoids decrease host resistance to infections with *Listeria monocytogenes* and herpes simplex virus type 2 (HSV-2). Host resistance was measured by changes in the 50% lethal dose of the pathogen in cannabinoid-treated and control mice. The effect of cannabinoids on resistance to *L. monocytogenes* was dose-dependent. Δ^9 -Tetrahydrocannabinol, at doses of 38, 75, and 150 mg/kg, suppressed resistance to infection by 10-, 17-, and 657-fold, respectively. Marijuana extract was less active, but it significantly reduced resistance to *L. monocytogenes* at all tested doses. Resistance to systemic HSV-2 infection was decreased 96-fold by Δ^9 -tetrahydrocannabinol, although marijuana extract was inactive. The doses and regimen of treatment with cannabinoids that produced significant decreases in host resistance were similar to those that caused suppression of delayed-type hypersensitivity to sheep RBC. The possible mechanisms and public health aspects of the decreased host resistance produced by marijuana extract and its cannabinoids are presented. (22 refs)

- 79-2644 Comparative Study of Liver Enlargement Induced by Various Chemicals.** (Eng) Fukuhara, M. (Inst. Public Health, 6-1 Shirokanedai 4-chome, Minato-ku, Tokyo 108, Japan); Takabatake, E. *J Pharmacobiodyn* 1(3): 153-159; 1978.

The ability of several chemicals to induce hepatomegaly was compared after short-term ip or dietary treatment of male Wistar-SLC rats. Polychlorinated biphenyls were the most effective, followed by 3-methylcholanthrene, furylfuramide, phenobarbital, and di-2-ethylhexyl phthalate. The chemicals had different effects on hepatic cytochrome P-450 concentration, aminopyrine N-demethylase activity, lipid and glycogen levels, and total amounts of DNA or total cell number. (23 refs)

- 79-2645 Induction of Rat Hepatic Epoxide Hydratase by Dietary Antioxidants.** (Eng) Kahl, R. (Dept. Pharmacology, Univ. Mainz, 6500 Mainz, W. Germany); Wulff, U. *Toxicol Appl Pharmacol* 47(2): 217-227; 1979.

The effects of supplementation of the diet with the antioxidants butylated hydroxytoluene (BHT), butylated hydroxyanisole, or ethoxyquin (each, 0.005%-0.5% of the diet for 14 days) on rat hepatic epoxide hydratase (EH) activity were studied in male Sprague-Dawley rats. All three compounds increased liver EH activity. The increase was obvious at the 0.1% dietary level and amounted to 200%-400% at 0.5%. Increased activity was accompanied by an increase of the EH band in sodium dodecyl sulfate-polyacrylamide gels, indicat-

ing induction of the enzyme. Ethoxycoumarin deethylase activity and cytochrome b, concentrations were moderately elevated, but cytochrome P-450 concentrations and aryl hydrocarbon hydroxylase (AHH) activity remained at control levels. Preferential inhibition of monooxygenase activity by metyrapone and not 7,8-benzoflavone, as well as the increased affinity of the reduced cytochrome P-450 for metyrapone as a ligand, indicated that the cytochrome P-450 population after BHT treatment was similar to that found after phenobarbital treatment. The antioxidants had no in vitro effect on EH activity and inhibited monooxygenase activity only in phenobarbital-stimulated microsomes but not in 3-methylcholanthrene (3-MC)-stimulated microsomes. Combined treatment with dietary antioxidants and 3-MC (3 doses of 20 mg/kg, ip) resulted in a marked induction of EH activity, but the 3-MC-mediated increase in AHH activity was partially depressed. Covalent binding of tritiated benzo(a)pyrene to calf thymus DNA was less effectively catalyzed by liver microsomes from animals fed antioxidants. The depression of covalent binding was marked after combined treatment with antioxidants and 3-MC. The shift in the microsomal enzyme pattern caused by the antioxidants may be related to their inhibitory effects on chemical carcinogenesis. (39 refs)

- 79-2646** Mutagenic Activities of 1,2,7,8-Diepoxyoctane in Chinese Hamster Lung Cells. (Eng) Huang, S. L. (Lab. Environmental Mutagenesis, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709). *J Environ Pathol Toxicol* 2(3): 587-599; 1979.

The mutagenicity of 1,2,7,8-diepoxyoctane (DEO) to Chinese hamster lung cells and its ability to form adducts with nucleotides were investigated. 6-Thioguanine resistance and chromosome aberrations were used as markers for mutagenicity. DEO was found to be genetically active and to form adducts readily with radiolabeled nucleotides, as demonstrated by thin-layer chromatography of DEO/nucleotide mixtures. The continuous disappearance of cells carrying chromosome aberrations from the cell populations was observed. Based on the expected frequency, cell death can be interpreted as a result of chromosome aberrations. The mutagenic specificity of DEO was apparently related to its ability to bind with DNA. (24 refs)

- 79-2647** Effect of Dose on Urinary Bladder Carcinogenesis Induced in F344 Rats by N-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide. (Eng) Arai, M. (First Dept. Pathology, Nagoya City Univ. Medical Sch., Nagoya 467, Japan); Cohen, S. M.; Jacobs, J. B.; Friedell, G. H. *J Natl Cancer Inst* 62(4): 1013-1016; 1979.

The effect of dose on N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT)-induced bladder carcinogenesis was evaluated in weanling male F344 rats. The rats were fed a diet

containing of 0.1%, 0.05%, 0.01%, 0.005%, 0.001%, or 0.0005% FANFT for 30 wk and then a control diet for 22 wk. A control group received only the control diet throughout the experiment. Papillary tumors were present at the higher doses, hyperplasia of various degrees of severity was present at the intermediate doses, and minimal hyperplasia was observed in 4/16 rats at the 0.005% dose; no mucosal abnormalities were observed at the two lower doses or in the control group. Bladder epithelium from selected animals was examined by scanning electron microscopy (SEM) after 10 wk and again at the end of the experiment. Hyperplastic mucosa with pleomorphic microvilli similar to that previously demonstrated for 0.2% FANFT was observed at 10 wk in rats fed 0.1% FANFT. Hyperplastic mucosa with pleomorphic microvilli was also observed at 52 wk in rats fed 0.1% and 0.05% FANFT. Hyperplastic mucosa without pleomorphic microvilli was observed in rats fed 0.01% and 0.005% FANFT. The bladder appeared normal by light microscopy and SEM at the two lower doses and in the control group at both the 10- and 52-wk intervals. A dose relationship was thus demonstrated for FANFT-induced bladder carcinogenesis in male F344 rats, and more severe surface changes were observed by SEM as the dose increased. (10 refs)

- 79-2648** Procarbazine-induced Specific-Locus Mutations in Male Mice. (Eng) Ehling, U. H. (Abteilung für Genetik, Gesellschaft Strahlen- und Umweltforschung, 8042 Neuherberg, W. Germany); Neuhauser, A. *Mutat Res* 59(2): 245-256; 1979.

The specific locus method was used to determine the ability of procarbazine (single dose of 200, 400, 600, or 800 mg/kg ip) to induce mutations in pre- and postmeiotic germ cells of hybrid (101/E1 x C3H/E1)_{F1} male mice. The lowest dose that significantly increased the mutation frequency in As spermatogonia over the control frequency was 400 mg/kg; the corresponding dose for the postspermatogonial germ-cell stages was 600 mg/kg. The responses were linear with dose up to 600 mg/kg. The variations in mutation rates among the seven loci between the lowest (*a* locus) and highest (*p* locus) was 12-fold. Only 24% of the procarbazine-induced specific locus mutations in the As spermatogonia were lethal in the homozygous condition. The mutations appeared to be due primarily to base-pair changes. The doubling dose of procarbazine in As spermatogonia (the dose needed to induce as many mutations as occur spontaneously) was 114 mg/kg. The therapeutic dose for procarbazine in humans is 215 mg/kg. If humans were as sensitive to the drug as mice, 22 mutations per million children would be expected to occur as a result of procarbazine treatment. (29 refs)

- 79-2649** *cis*-Platinum(II) Diamine Dichloride Causes Mutation, Transformation, and Sister-Chromatid Exchanges in Cultured Mammalian Cells. (Eng) Turn-

bull, D. (Biology Branch, Carcinogenesis Res. Program, NCI, NIH, Bethesda, MD, 20014); Popescu, N. C.; DiPaolo, J. A.; Myhr, B. C. *Mutat Res* 66(3): 267-275; 1979.

The ability of *cis*-platinum(II) diammine dichloride (PDD) to induce sister chromatid exchanges (SCE's), mutation, and morphologic transformation in V79 chinese hamster cells and in secondary Syrian hamster embryo cells (HEC) was investigated. PDD, at 0-10 $\mu\text{g/ml}$ and 0-0.5 $\mu\text{g/ml}$, respectively, produced a dose-dependent toxicity in both V79 cells and HEC. It also caused a significant, dose-dependent increase in SCE frequency in V79 cells relative to untreated controls. The increase was more than threefold at 1.0 $\mu\text{g/ml}$ PDD and eightfold at 5 $\mu\text{g/ml}$. Chromatid and chromosome aberrations were induced within 6 hr of treatment of V79 cells with 5 $\mu\text{g/ml}$ PDD, and thioguanine-resistant mutants were induced in a dose-dependent manner. Expression of resistance was complete within 2 days following treatment with 2.5 $\mu\text{g/ml}$ and within 5 days after $\mu\text{g/ml}$. Morphologically altered colonies were found in HEC cultures treated with PDD, an approx linear relation being observed between drug concentration and the frequency of transformed colonies per survivor for all doses tested. The data suggest that PDD is a potential carcinogen. (17 refs)

79-2650 Comparison of Level, Duration of Exposure, Housing, and Source of Animal on Diethylstilbestrol-induced Mammary Tumorigenesis in C3H Mice (Meeting Abstract). (Eng) Norvell, M. J. (Natl. Center Toxicological Res., Jefferson, AR); Farmer, J. H. *J Toxicol Environ Health* 4(2/3): 492; 1978. (no refs)

79-2651 Carcinogenic Effects of Diethylstilbestrol in Male Syrian Golden Hamsters and European Hamsters. (Eng) Reznik-Schuller, H. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD, 21501). *J Natl Cancer Inst* 62(4): 1083-1088; 1979.

The tumorigenic effects of sc-implanted diethylstilbestrol (DES) on male Syrian golden hamsters and European hamsters were compared. The latter species have been found to be highly sensitive to various chemical carcinogens. Each animal received an sc implant of a pellet containing 25 mg DES; this implant was replaced every 5 mo. The adenohypophyses, kidneys, and testes of both species showed neoplastic responses to DES treatment. European hamsters were more sensitive than Syrian hamsters, inasmuch as the European hamsters had a higher tumor incidence. The testicular tumors were all Leydig cell adenomas, and they seemed to depend on the coincident occurrence of hypophyseal neoplasms (all composed of gonadotropic cells). Of the European hamsters tested, 29% also developed liver tumors (hepatocellular adenomas, carcinomas, and cholangiocarcinomas). These lesions were not found in the Syrian hamsters. (13 refs)

79-2652 Induction of Lymphomas by Urethane in Combination with Diethylstilbestrol in CFLP Mice.

(Eng) Bojan, F. (Inst. Hygiene and Epidemiology, Univ. Medical Sch., Nagyerdei krt 98, H-4012 Debrecen, Hungary); Redai, I.; Gomba, S. *Experientia* 35(3): 378-379; 1979.

The combined carcinogenic effect of urethan (UT) and diethylstilbestrol (DES) was studied in CFLP mice. Adult CFLP mice received a single dose of 1,000 mg/kg UT ip and 50 mg/kg DES sc either simultaneously or at a 14-day interval. Animals were autopsied when they appeared ill or within 300 days. Induced lymphomas were transplanted (5×10^6 cells ip) into newborn mice of several strains. Autopsy revealed two tumor types: malignant lymphomas in some mice in groups treated with DES + UT and multiple lung tumors in all animals treated with UT, with or without DES. DES + UT after 14 days induced lymphomas in 4/22 mice; DES + UT simultaneously induced lymphomas in 8/25 mice; UT + DES after 14 days induced lymphomas in 11/25 mice. UT alone induced lymphomas in 0/48 mice and DES alone in 2/40. The induced lymphomas were successfully transplanted in 6/10 newborn CFLP mice. The passage of lymphomas from CFLP mice into newborn BALB/c, C3H/He-mg, CBA/Ca, and AKR mice was also successful. All the induced and transplanted lymphomas were lymphoblastic lymphosarcomas. Intracellular A-type and extracellular C-type virus particles were detected in the tumors by electron microscopy. These results demonstrate that the carcinogenic potential of UT and DES may be enhanced in combination. (9 refs)

79-2653 Effect of In Utero Exposure to Diethylstilbestrol on the Immune Response in Mice. (Eng)

Luster, M. I. (Natl. Inst. Environmental Health Sciences, NIH, PO Box 12233, Research Triangle Park, NC, 27709); Faith, R. E.; McLachlan, J. A.; Clark, G. C. *Toxicol Appl Pharmacol* 47(2): 279-285; 1979.

The effects of a single administration of diethylstilbestrol (DES) in the prenatal period on cellular and humoral immunity were studied in Swiss-Webster (CD-1) mice. Mice were exposed to DES through maternal dosing (0.1 mg/kg sc) on gestation day 16, and they were evaluated at age 7-9 wk. When sensitized mice were challenged with purified protein derivative (PPD), only treated females had a significant reduction in delayed hypersensitivity reactions, but males showed a similar trend. Spleen cells from male and female DES-exposed mice had a significantly diminished response to concanavalin A compared with sex-matched controls. DES treatment had no effect on serum IgG2 levels, but it decreased serum IgG1 levels in females and increased these levels in males. IgM and IgA were also decreased in treated females, but they were within the normal range in male mice. In the antibody plaque assay, DES treatment did not significantly alter the response to sheep RBC. However, *Escherichia coli* 0127 lipopolysaccharide (LPS) plaque-forming cells were significantly suppressed in treated females and increased in treated males. This enhanced LPS response was due to IgM

(19S) antibodies. These results indicate that DES slightly suppressed cell-mediated immune function in both males and females. There was a sex specificity in humoral immune function, with enhanced antibody responses to a T-independent antigen in males and either no effect or slight suppression in females. (28 refs)

- 79-2654** Transplacental Exposure to Diethylstilbestrol in Men. (Eng) Andonian, R. W. (Div. Urology, Dept. Surgery, Stanford Univ. Medical Center, Stanford, CA 94305); Kessler, R. *Urology* 13(3): 276-279; 1979.

A comparison of 24 men exposed to diethylstilbestrol in utero with 24 age-matched controls revealed no differences in psychosexual history, medical history, genital examination, or semen analysis between the two groups. (9 refs)

- 79-2655** Long-Term Estrogenization in Mammals. I. Histopathology of Kidney, Bladder, Adrenals, and Gonads; Polydipsia; Body Weight; and Serum Levels of Corticosterone and Testosterone in Estrogenized Marsh Mice. (Eng) Bryson, G. (Dept. Psychology, Univ. California at Santa Barbara, Santa Barbara, CA 93106); Bischoff, F. *Res Commun Chem Pathol Pharmacol* 23(3): 575-595; 1979.

The dipsogenic effects of single sc injections of estrogen (5 mg) and estradiol (0.34 mg as estradiol cypionate) were studied in Marsh mice. Histopathologic changes in the bladder (eg, cystitis, metaplasia, hypertrophy inflammation, ulceration) occurred following the onset of polydipsia in 14/51 mice given estrogen and 29/52 mice given estradiol. Histopathology of the kidney (eg, pyelonephritis, ischemic necrosis, interstitial nephritis) occurred less frequently, indicating that bladder pathology preceded kidney pathology. The polydipsia was more intense (up to 250% over control intake) in animals housed in metal cages with floor screens than in those housed in plastic cages. Water intake by untreated controls was unaffected by housing. Water intake by estrogenized mice was always significantly greater than that by untreated controls. Estrogen produced a body wt loss that was not recovered except in the case of the youngest females (2.5 mo at the time of estrogen injection). Estrogenization markedly increased corticosterone and decreased testosterone levels in the serum. The levels of these hormones were negatively correlated in the estrogenized mice, whereas the testosterone levels were positively correlated with testis wts. (23 refs)

- 79-2656** Pathological Effects in Mice Induced by Diets Containing Diethylstilbestrol or 17 β -Estradiol

(Meeting Abstract). (Eng) Highman, B. (Univ. Arkansas Medical Sciences, Little Rock, AR); Norvell, M. J.; Greenman, D. L.; Shellenberger, T. E. *J Toxicol Environ Health* 4(2/3): 483-484; 1978 (no refs)

- 79-2657** Rotenone Does Not Interfere with Estrogen Action in the Rat Uterus (Meeting Abstract). (Eng) Morris, M. E. (Hormone Res. Program, Natl. Center Toxicological Res., Jefferson, AR); Sheehan, D. M. *J Toxicol Environ Health* 4(2/3): 490; 1978 (no refs)

- 79-2658** Induction of Pituitary and Mammary Tumors in Male, "Fale" and Female Rats by Either DMBA, Estradiol Implant or Combined Treatment. (Eng) Kao, K. J. (Dept. Physiology and Biophysics, Univ. Illinois, Urbana, IL 61801); Ramirez, V. D. *Proc Soc Exp Biol Med* 160(3): 296-301; 1979.

The endocrinologic basis for the sex difference in the response of rats to 7,12-dimethylbenz(a)anthracene (DMBA) carcinogenesis was studied in Sprague-Dawley rats. Pellets containing 5.06 mg β -estradiol-3-benzoate (E_2) crystals were implanted sc into 45-day-old intact or neonatally castrated males and females; DMBA (5 mg/ml of a 1 ml emulsion) was injected at 50 days. The incidence of DMBA-induced mammary tumors was 50% in intact females vs 16.7% in intact males. In castrated male and female rats, the tumor incidence increased from 0% to 30% and 44.4%, respectively, after E_2 implantation. E_2 did not increase the mammary tumor incidence in intact animals. High serum prolactin levels were induced by E_2 in males and females, and pituitary tumors were found in almost all animals with E_2 implants. In females implanted with E_2 at 45 days, follicle stimulating hormone and luteinizing hormone levels were normal. The increase in serum prolactin levels in these rats was effectively reduced by sc injection of α -bromoergocriptine (400 μ g) 96 days after E_2 implantation. The results suggest that long-term, low-dose administration of estrogenic substances might increase the risk of pituitary tumor induction in humans. (38 refs)

- 79-2659** Combined Epithelial and Sarcomatous Elements in a Liver Cancer Associated with Oral Contraceptive Use. (Eng) Ladaga, L. (Medical Center Hosp., 600 Gresham Dr., Norfolk, VA 23507); Kay, S.; Melcher, M.; King, J. N. *Am J Surg Pathol* 3(2): 185-190; 1979.

The occurrence of a rare malignant tumor in a 35-yr-old woman who had used oral contraceptives (norethindrone 2 mg and mestranol 0.10 mg) continuously for 12 yr is reported. Use of the contraceptives was discontinued 3 yr

prior to admission because of hepatic enlargement. The liver spontaneously regressed in size and remained stable until 3 mo prior to admission, at which time hepatomegaly, jaundice, and pruritis were noted. Hypovolemia and clotting abnormalities were corrected and an exploratory laparotomy revealed a massive ip hemorrhage secondary to a ruptured tumor on the inferior aspect of the left lobe of the liver. The patient died 19 days after admission, and postmortem examination revealed a bimorphic tumor replacing 50% of the liver and consisting of moderately differentiated hepatocellular carcinoma and sarcoma. The absence of cellular transition and of a distinct metastatic pattern implied separate primaries with collision rather than common ancestry or dedifferentiation. A sister of the patient had a histologically proved hepatic adenoma associated with the use of oral contraceptives. (19 refs)

79-2660 Oral Contraceptive Administration and Hepatocellular Neoplasms in the Rat: Preliminary Results (Meeting Abstract). (Eng) Klein, K. M. (Dept. Pathology, Coll. Medicine and Dentistry New Jersey-New Jersey Medical Sch., Newark, NJ) *Gastroenterology* 76(5, part 2): 1288; 1979 (no refs)

79-2661 Intravaginal Estrogen Creams and Endometrial Cancer. No Causal Association Found. (Eng) Horwitz, R. I. (Robert Wood Johnson Clinical Scholar Program, Yale Univ. Sch Medicine, 333 Cedar St., New Haven, CT 06510); Feinstein, A. R. *JAMA* 241(12): 1266-1267; 1979.

In two case-control studies of estrogens and endometrial cancer, 187 women with endometrial cancer and 137 matched controls were asked about previous use of intravaginal estrogen creams. The two studies used different sampling methods to select cases and controls. In one study, 7% of the women with endometrial cancer reported using estrogen creams compared with 8% of the controls. In the second study, 9% of the cancer patients reported having used estrogen creams compared with 10% of the controls. The odds ratio in the first study was 0.90 and that in the second study was 0.82. In neither study were the differences significant. The results do not support an association between the use of intravaginal estrogen creams and endometrial cancer. (10 refs)

79-2662 Contraceptive Steroids and Breast Cancer. (Eng) Spencer, J. D. (Imperial Cancer Res. Fund Breast Cancer Unit, Guy's Hosp., London SE1 9RT, England); Millis, R. R.; Hayward, J. L. *Br Med J* 1(6119): 1024-1026; 1978.

The prognosis and pathological findings in 44 breast cancer

patients who had taken contraceptive steroids during the year before diagnosis (study group) were compared with those in 44 breast cancer patients who had never used contraceptive steroids (controls) matched for age and parity. All of the patients in the study group had been using products containing ≤ 50 μ g estrogen. No appreciable difference in tumor size, histological type, grade of malignancy, or lymphoplasmacytic infiltration was found between the study group and controls. Moreover, the degree of sinus histiocytosis in unaffected lymph nodes was similar in each group. Fewer patients in the study group had axillary lymph node infiltration, and the mean number of affected nodes in these patients was less than that of controls. In patients with axillary node disease, the recurrence rate in controls was significantly higher than that in the study group. One interesting and unexplained finding was that the mothers of nine patients in the study group had had breast cancer compared with none in the control group. This difference merits further investigation in a larger series. Overall, the findings suggest that recurrence rate and mortality are unlikely to be increased in patients who have taken contraceptive pills before breast cancer is diagnosed; rather, the opposite seems to be the case. (14 refs)

79-2663 Prostatic Androgen Receptors: Changing Homeostasis and Development of Prostatic Disease (Meeting Abstract). (Eng) Shain, S. A. (Southwest Foundation Res. and Education, San Antonio, TX); McCullough, B.; Boesel, R. W.; Lamm, D.; Radwin, H. *J Toxicol Environ Health* 4(2/3): 496-497; 1978 (no refs)

See also:

*(Rev.): 79-2401, 79-2402, 79-2403, 79-2404, 79-2405, 79-2407, 79-2408, 79-2409, 79-2410, 79-2411, 79-2412, 79-2414, 79-2415, 79-2416, 79-2417, 79-2418, 79-2419, 79-2420, 79-2421, 79-2422, 79-2423, 79-2425, 79-2426, 79-2427, 79-2428, 79-2429, 79-2430, 79-2431, 79-2432, 79-2433, 79-2434, 79-2435, 79-2436, 79-2437, 79-2438, 79-2440, 79-2445, 79-2446, 79-2467, 79-2469.

*(Phys.): 79-2669, 79-2671, 79-2672, 79-2675, 79-2678.

*(Viral): 79-2695, 79-2729, 79-2733, 79-2734.

*(Immun.): 79-2823, 79-2825.

*(Path.): 79-2835, 79-2845, 79-2846, 79-2848, 79-2851, 79-2880, 79-2891, 79-2894.

*(Epid-Biom.): 79-2903, 79-2904, 79-2910, 79-2911, 79-2916, 79-2919, 79-2921, 79-2927, 79-2928, 79-2930, 79-2931, 79-2933, 79-2934, 79-2944, 79-2947, 79-2948, 79-2949, 79-2950, 79-2953, 79-2954, 79-2955, 79-2962, 79-2963, 79-2966, 79-2967, 79-2969.

PHYSICAL CARCINOGENESIS

- 79-2664 **The Many Faces of Solar Keratoses.** (Eng) Wade, T. R. (Dept. Dermatology, New York Univ. Medical Center, New York, NY); Ackerman, A. B. *J Dermatol Surg Oncol* 4(10): 730-734; 1978.

Various histologic presentations of actinically damaged skin from elastosis to keratoses and to eventual carcinomatosis are illustrated and explained. (no refs)

- 79-2665 **γ -Ray-enhanced Reactivation of UV-irradiated Adenovirus in Normal Human Fibroblasts.** (Eng) Jeeves, W. P. (Dept. Biology, McMaster Univ., Hamilton, Ontario L8s 4J9, Canada); Rainbow, A. J. *Mutat Res* 60(1): 33-41; 1979.

The γ -ray-enhanced reactivation (γ RER) of UV-irradiated human adenovirus type 2 (Ad2) in several normal human fibroblast lines was examined. The frequency of cells positive for Ad2 viral structural antigen (VAg) was reduced in pre- γ -irradiated cultures 48 hr after infection with unirradiated virus, whereas VAg was reduced to a significantly lesser extent or even increased in preirradiated cells 48 hr after infection with UV-irradiated virus. Preirradiation of cells immediately prior to infection with UV-irradiated virus markedly enhanced the survival of VAg production. The av value for the γ RER factor rose with increasing UV dose to the virus and with increasing γ -ray dose to the cells. Examination of VAg expression at various times after infection indicated that preirradiation of the cells with γ -rays prior to infection with UV-irradiated virus led to an earlier onset and/or increased rate of VAg synthesis. This enhancement of VAg production from a UV-damaged template may have resulted from an inducible DNA-repair mechanism in human fibroblasts that may or may not be error-prone. (18 refs)

- 79-2666 **Photosensitivity and DNA Repair. Possible Relationship Between Xeroderma Pigmentosum and Cockayne's Syndrome.** (Fre) Lafforet, D. (Unite de Recherche d'Hepatologie Infantile I.N.S.E.R.M., U. 56, 94270 Bicetre, France); Dupuy, J. M. *Arch Fr Pediatr* 35(10, Suppl): 65-74; 1978.

DNA repair following UV irradiation and phytohemagglutinin (PHA) transformation of lymphocytes were studied in 12 children (7 boys, 5 girls aged 3-12) with photosensitivity diseases [xeroderma pigmentosum (XP:6), Cockayne's syndrome (CS:2), XP-CS (2), and Bloom's syndrome (2)]. These diseases are rare and are transmitted genetically by autosomal recessive genes. DNA

repair was studied in lymphocytes irradiated with 30, 90, or 300 ergs/mm². After 30 ergs/mm², DNA repair, as measured by ³H-thymidine uptake, was normal. After 300 ergs/mm², however, the lymphocytes from all but two of the patients (1 with XP and 1 with XP-CS) showed diminished DNA repair. Reaction to stimulation by PHA was diminished in lymphocytes in 5/6 XP patients, 4/4 CS and XP-CS patients, and 1/2 patients with Bloom's syndrome. The immune deficit observed in the XP patients and in those with CS or XP-CS suggests that these diseases have a common origin. (19 refs)

- 79-2667 **Detection of Cyclobutyl Pyrimidine Dimers in Human Skin after In Vivo Irradiation with Ultraviolet Light (Meeting Abstract).** (Eng) Sutherland, B. M. (Dept. Biology, Brookhaven Natl. Lab., Upton, NY); Kochevar, I. E. *J Invest Dermatol* 72(4): 197; 1979 (no refs)

- 79-2668 **Induction of Sister Chromatid Exchanges in Human Cells by Fluorescent Light.** (Eng) Monticone, R. E. (Section Cellular Aging and Genetics, Lab. Cellular and Molecular Biology, Gerontology Res. Center, Natl. Inst. Aging, NIH, Baltimore, MD 21224); Schneider, E. L. *Mutat Res* 59(2): 215-221; 1979.

The bromodeoxyuridine-differential staining technique was used to examine the induction of sister chromatid exchanges (SCE's) by fluorescent light (FL) in human fetal lung fibroblasts (IMR-90). Exposure of these cells in media to FL increased the SCE frequency from a background level of 8.5 to 20.5 SCE's/cell. Cellular replication kinetics were also inhibited by FL exposure. Exposure of cells to FL in phosphate-buffered saline resulted in a twofold increase in SCE levels and an increased inhibition of cell replication, indicating that culture media may have a protective effect. Determinations of SCE frequencies with blocking filters indicated that the FL wavelengths responsible for SCE induction were in the near-UV spectrum between 300 and 390 nanometers. Culturing cells in media that had been exposed to FL resulted in a significant increase in SCE levels, 14.5 vs 7.5, demonstrating the contribution of media photoproducts to SCE induction. The role of media photoproducts was further reinforced by the significant decline in FL-induced SCE found in cells cultured in medium deficient in three known photosensitizers (phenol red, tetracycline, and riboflavin) for 2-3 wk prior to exposure. Since SCE's have been shown to be a sensitive indicator of DNA damage, these results indicate that FL can

induce genetic damage in human cells. These findings are of importance to investigators culturing cells in laboratories illuminated with FL. (30 refs)

- 79-2669 Single-Strand Breaks in DNA During Repair of UV-induced Damage in Normal Human and Xeroderma Pigmentosum Cells as Determined by Alkaline DNA Unwinding and Hydroxyapatite Chromatography. Effects of Hydroxyurea, 5-Fluorodeoxyuridine and 1- β -D-Arabinofuranosylcytosine on the Kinetics of Repair. (Eng) Erixon, K. (Dept. Radiation Chemistry, Wallenberg Lab., Univ. Stockholm, S-10691 Stockholm, Sweden); Ahnstrom, G. *Mutat Res* 59(2): 257-271; 1979.

UV light-induced excision repair was studied in normal human fibroblasts and in fibroblasts from patients with xeroderma pigmentosum (XP). At 0 C, UV light (20 joules/m²) produced a very low frequency of strand breaks in normal cells; on subsequent incubation at 37 C, breaks formed and then disappeared in a dose-dependent fashion. Hydroxyurea (HU: 10⁻² M), 5-fluorodeoxyuridine (FUDR: 2 x 10⁻⁶ M), and 1- β -D-arabinofuranosylcytosine (araC: 10⁻⁴ M) dramatically increased the number of breaks. The effects of FUDR were reversed by subsequent incubation with 50 μ M thymidine. Essentially no breaks were induced by UV light in XP cells belonging to complementation group A (XP-A), and there was no enhancement by HU. Cells from XP variants with no reduction in pyrimidine dimer excision or unscheduled DNA synthesis reacted in a manner similar to that of normal cells. When XP variant and normal cells were incubated at 37 C for 10 min following irradiation, the number of breaks increased with UV dose and a saturation level was reached. HU or araC enhanced this response considerably. For XP-A cells, there was essentially no dose effect, either in the absence or presence of HU. (58 refs)

- 79-2670 Relationship Between Actinic Damage and Chronologic Aging in Keratinocyte Cultures of Human Skin (Meeting Abstract). (Eng) Gilchrist, B. A. (Dept. Dermatology, Beth Israel Hosp., Boston, MA) *Clin Res* 27(2): 527A; 1979 (no refs)

- 79-2671 Cell Cycle Analysis after Exposure to Psoralen and UV Light (Meeting Abstract). (Eng) Burkholder, D. E. (Dept. Dermatology, Yale Univ., New Haven, CT); Cohen, S. R.; Varga, J. M.; Carter, D. M.; Grais, L. S. *J Invest Dermatol* 72(4): 197; 1979 (no refs)

- 79-2672 The Effect of 8-Methoxypsoralen-Plus Ultraviolet Light on Cell-Virus Interaction:

The Transforming Infection: Effect of PUVA on the Transformation of Baby Hamster Kidney Cells by Polyoma Virus. (Eng) Morhenn, V. B. (Dept. Dermatology, Stanford Univ. Medical Center, Stanford, CA); Kaye, J. A. *J Invest Dermatol* 72(3): 138-142; 1979.

The effects of pretreatment with 8-methoxypsoralen (8-MOP) + UV light (PUVA) on the transformation of baby hamster kidney (BHK) cells by polyoma virus (Py) were studied. PUVA enhanced viral transformation, the enhancement increasing with decreasing cell survival and being max at 0.5 μ g/ml 8-MOP + 0.3 joule (J)/cm² UV light. The number of transformed colonies increased with virus concentration, and the enhancement factor due to PUVA was independent of the multiplicity of infection. The drug and light acted synergistically in enhancing viral transformation. The absolute number of transformed colonies in the PUVA-pretreated Py-infected cultures was smaller than that in control infected cultures; therefore, the enhancement of transformation was dependent on correction for the number of cells capable of growing after PUVA treatment. PUVA had no transforming effects on uninfected BHK cultures. The PUVA-pretreated, Py-infected cells were tumorigenic following sc injection (10⁶ cells) into hamsters (9/9 and 5/6 injected animals had tumors). Viral transformation was enhanced sixfold at a PUVA concentration (0.01 μ g/ml 8-MOP + 1.2 J/cm² UV) that inhibited DNA synthesis to 75% of control levels at 38 hr after treatment. (42 refs)

- 79-2673 Quantitative Studies of Radiation Transformation with the A31-11 Mouse BALB/3T3 Cell Line. (Eng) Little, J. B. (Lab. Radiobiology, Dept. Physiology, Harvard Sch. Public Health, Boston, MA 02115) *Cancer Res* 39(5): 1474-1480; 1979.

A cloned BALB/3T3 mouse embryo-derived fibroblast cell line (A31-11) was used to study morphological transformation induced by x-rays and 254-nanometer UV light. The transformation frequency increased exponentially with increasing dose from 10 to 400 rads for x-rays and 1.0 to 7.5 joules/m² for UV exposure. Split-dose x-ray exposures led to an enhancement in transformation at total doses <100 rads and a reduction at doses of 300-400 rads. The induced transformation frequency varied among serum lots and was very dependent upon the initial cell density. Spontaneous transformants were observed in 10/22 consecutive experiments; the spontaneous transformation frequency was generally about 1 to 2 x 10⁻⁵, compared with induced frequencies that ranged up to 3 x 10⁻³ for x-rays and 7.5 x 10⁻⁴ for UV exposure. Further results indicated that this cell line has several potential advantages over the mouse 10T1/2 line for studies with relatively weak in vitro carcinogens such as radiation. These include (1) a reduced overall expression time for the appearance of transformed foci (4 wk); (2) a high cloning efficiency (50%-60%); and (3) the fact that about 20 times as many viable cells may be

plated per dish for optimal results, allowing transformation frequencies as low as 10^{-5} to be measured easily. On the other hand, there was more variability in the result of experiments with the 3T3 cell line. (26 refs)

79-2674 The Distribution of Ultraviolet and Ionizing Radiation Induced Damage and Repair in Staphylococcal Nuclease Sensitive and Resistant DNA's of Mammalian Chromatin (Meeting Abstract). (Eng) McConlogue, L. C. (Univ. California, Los Angeles, CA) *Diss Abstr Int [B]* 39(10): 4761-4762; 1979 (no refs)

79-2675 Potentiation of Lethality by Caffeine and Other Aspects of Survival-related Repair in Various Mammalian Cells Irradiated with Ultraviolet Light, Gamma-Rays, and Fast Neutrons (Meeting Abstract). (Eng) Schroy, C. B. (Pennsylvania State Univ., University Park, PA) *Diss Abstr Int [B]* 39(10): 4762-4763; 1979 (no refs)

79-2676 In Vitro Radiosensitivity of Fibroblasts from Patients with Familial and Sporadic Retinoblastoma: A Naturally Occurring Model for the Study of Mutagenesis and Carcinogenesis (Meeting Abstract). (Eng) Little, J. B. (Joint Center for Radiation Therapy, Harvard Medical Sch., Boston, MA 02115); Weichselbaum, R. R. *Int J Radiat Oncol Biol Phys* 4(Suppl 2): 88; 1978 (no refs)

79-2677 The Need for a Radiation History in Patients with Gastrointestinal Diseases--Case Reports (Meeting Abstract). (Eng) Rogers, A. G. (Dept. Medicine, Univ. Manitoba Medical Sch., Winnipeg, Manitoba, Canada); Kirkpatrick, J. R. *Gastroenterology* 76(5, part 2): 1228; 1979 (no refs)

79-2678 Effects of X-Irradiation, TPA and Protease Inhibitors on Malignant Transformation in Mouse 10T1/2 Cells (Meeting Abstract). (Eng) Kennedy, A. R. (Harvard Sch. Public Health, Boston, MA 02115); Nagasawa, H.; Little, J. B. *In Vitro* 15(3): 208; 1979 (1 ref)

79-2679 Radiation Exposure and the Simultaneous Occurrence of Primary Hyperparathyroidism and Thyroid Nodules. (Eng) Nishiyama, R. H. (Dept. Pathology, Univ. Michigan Medical Center, Ann Arbor,

MI); Farhi, D.; Thompson, N. W. *Surg Clin North Am* 59(1): 65-75; 1979.

The role of ionizing radiation in the pathogenesis of thyroid carcinoma and hyperparathyroidism (HPT) was studied among 42 women and 11 men with concomitant thyroid disease and primary HPT. The presenting symptoms were related to HPT in 30 patients and to thyroid disease in 3; 20 were asymptomatic. The pathologic changes in the thyroid glands included nodular goiters in 33 patients, follicular carcinomas in 3, papillary carcinomas in 10, and Hashimoto's disease in 7. Thirty-four percent of the patients had been exposed to head and neck radiation an av of 29 yr prior to the detection of thyroid or parathyroid disease; radiation histories were incomplete in many cases. Of the 13 patients with thyroid carcinoma, 9 had been exposed to radiation and 4 had not. Seven of the nine irradiated thyroid carcinoma patients also had parathyroid adenomas, as did all nonirradiated patients with thyroid carcinomas. Only one patient with follicular carcinoma had a history of head and neck irradiation. The data indicate that undetermined etiologic factors other than radiation may be involved in the simultaneous appearance of HPT and thyroid carcinomas in some patients. (46 refs)

79-2680 Association of Malignancy with Rapid Growth in Early Lesions Induced by Irradiation of Rat Skin. (Eng) McGregor, J. F. (Biology and Health Physics Div., Atomic Energy Canada, Ltd., Chalk River, Ontario, Canada K0J 1J0) *J Natl Cancer Inst* 62(4): 1043-1049; 1979.

Cancerous and noncancerous epithelial lesions induced by irradiation of rat skin were studied to determine (1) the relationship of malignancy to dose, (2) the types of lesions and circumstances leading to overt malignancy, and (3) the growth rates of lesions progressing to malignancy vs those of lesions remaining benign. High doses of radiation were associated with the production of epidermal cancers, the max yield being obtained at 6,400 rads. Conversely, a peak yield of noncancerous lesions was obtained at 1,600 rads. This association between malignancy and high dose was consistent for cancers evolving from warts, cysts, and chronic ulcers. Although the proportion of warts among the induced lesions was much higher than that of the cysts or chronic ulcers (76%, 14%, and 10%, respectively), the likelihood of warts becoming cancerous was substantially lower (14%, 23%, and 21%). The combined data for all doses showed that the latency period of the epidermal cancers was significantly ($p = 0.015$) shorter than that of the benign tumors. Rapid growth rates were observed for warts, cysts, and chronic ulcers progressing to overt cancer, and these did not overlap at any point on the growth scale with rates for benign tumors. This finding suggests that the potential for malignant development had been established early in the carcinogenic process, very likely at induction. (15 refs)

- 79-2681 **Assessment of Biological Damage by Personal Neutron Dosimeters.** (Ger) Dorschel, B. (Sektion Physik, Technische Universität Dresden, Mommsenstrasse 13, DDR-8027 Dresden, E. Germany); Herforth, L. *Kernenergie* 21(12): 377-382; 1978.

The feasibility of assessing biological damage due to neutron irradiation by personal neutron dosimeters was determined. The direct calibration of detectors used as personal dosimeters is possible only if the neutron field is known. (10 refs)

- 79-2682 **Comments on Leukemia Risk from Neutrons (2 Letters to Editor).** (Eng) Jablon, S. (6813 Persimmon Tree Road, Bethesda, MD 20034); Rossi, H. H.; Mays, C. W. *Health Phys* 36(2): 205-206; 1979.

A previous estimation that a worker who received the max permissible dose of neutrons for 50 yr would have five times the naturally occurring risk of death from leukemia is stated to be an overestimation by a factor of about 5. Also, there is no convincing evidence that the limits of low-LET (linear energy transfer) radiations are conservative. In a reply, it is agreed that the estimate may be high, but that the error is <30%. Facts are also presented indicating that the notion of a dose-independent relative biological effectiveness of neutrons relative to gamma radiation must be rejected. (3 refs)

- 79-2683 **Morphological Study of the Bone Marrow in Patients Injected with ThO₂.** (Eng) Parreira, F. (Hematology Lab., Universidades de Lisboa, Hospital de Santa Maria, Lisbon 4, Portugal); Carneiro de Moura, M. *Environ Res* 18(1): 61-64; 1979.

The bone marrow morphology of 12 patients who had been injected with thorium dioxide colloid (Thorotrast) 21-39 yr earlier was studied. Eight patients had anemia, most with normal or slightly decreased mean cell Hb values. One patient had acute myeloblastic leukemia (AML), one cholangiocarcinoma. Cellularity was normal in only four patients; the degree of hypoplasia resulted in aplasia in some patients. Excluding the AML patient, all patients had an abnormal myeloid:erythroid ratio; in these patients, including those with an apparently normal cellularity, granulocytopenia was depressed. It is concluded that the pathogenesis of anemia can be explained by proliferative asthenia. There is no explanation for the lack of correlation between the myeloid:erythroid ratio and the WBC count in the peripheral blood. There was also no correlation between the degree of bone marrow hypoplasia and the duration of radiation. (no refs)

- 79-2684 **Pathogenesis of Radiation Induced Lung Cancer Following ¹⁴⁴CeCl₃ Instillation**

(Meeting Abstract). (Eng) Levine, G. (Northwestern Univ., Evanston, IL) *Diss Abstr Int [B]* 39(10): 4761; 1979 (no refs)

- 79-2685 **Coefficients of Conversion and Diffusion of Plutonium Through the Skin.** (Rus) Filatov, V. V. (No affiliation given); Osanov, D. P. *Biofizika* 24(1): 171; 1979.

An attempt was made to describe the transfer of radionuclides through the skin by means of a diffusion equation. Approximation of experimental data on the accumulation of plutonium nitrate (2 µCi/ml) in 2-mo-old pigs after topical application revealed a good fit between observed and expected values. (2 refs)

- 79-2686 **Characterization of Cell Cultures Derived From ²³⁹Pu-induced Tumors in Beagles (Meeting Abstract).** (Eng) Andrews, T. K. (Northwest Lab., Richland, WA 99352); Frazier, M. E. *In Vitro* 15(3): 196; 1979 (no refs)

- 79-2687 **The Gastrointestinal Absorption of Plutonium and Americium in the Hamster.** (Eng) Stather, J. W. (Natl. Radiological Protection Board, Harwell, Didcot, Oxfordshire, OX11 0RQ, England); Harrison, J. D.; Rodwell, P.; David, A. J. *Phys Med Biol* 24(2): 396-407; 1979.

Plutonium-239 and americium-241 were given to adult Syrian golden hamsters in various chemical forms and by various routes, and their absorption from the gut was measured. One month after iv injection of the citrate complexes of ²³⁹Pu [400 Becquerels (Bq)] or ²⁴¹Am (100 Bq), the skeleton and liver contained >90% of the total body content of both actinides. The absorption of ²³⁹Pu from the gastrointestinal tract varied from 0.01% to 0.00003%, depending on the chemical form administered. The highest values were obtained after protracted administration of the nitrate (50 doses of 0.74 kBq, po), administration of a single dose as the citrate complex (12 kBq), or administration of ²³⁹Pu in a biologically incorporated form in hamster liver (44 kBq). Intermediate values were obtained with single doses of the mixed oxide (36 kBq) and the nitrate (9.3 kBq), the lowest value with the dioxide (75 kBq). The absorption of ²⁴¹Am from the gastrointestinal tract varied from 0.06% to 0.004%. The highest value was obtained after administration of the nitrate (35 kBq). A single dose of the citrate (33 kBq) gave intermediate values, and the dioxide (237 kBq) and liver-incorporated (37 kBq) forms gave the lowest absorption values. ²⁴¹Am was absorbed more readily than ²³⁹Pu. When these values were compared with those for other species, no large variations were observed that

could be attributed to species differences, providing some justification for extrapolation of the results to humans. On the basis of current best knowledge, the best values for the estimated absorption in human adults appear to be 0.05% for Am, 0.01% for Pu ingested in soluble form, and 0.0001% for Pu ingested as the dioxide. (44 refs)

- 79-2688 Ingested Mineral Fibers: Elimination in Human Urine.** (Eng) Cook, P. M. (U.S. Environmental Protection Agency, Environmental Res. Lab., 6201 Congdon Blvd., Duluth, MN 55804); Olson, G. F. *Science* 204(4389): 195-198; 1979.

Sediment filtered from human urine samples was examined by transmission electron microscopy in order to determine whether asbestos or other mineral fibers might be present. The urine samples were collected from male and female Minnesota residents, all in good health. Samples from individuals who drank only unfiltered water (from Western Lake Superior) contained 310-1,170 amphibole fibers/ml, whereas those from individuals who drank only filtered water contained 13-40 fibers/ml. The urine of two men, after months of ingesting only filtered water, contained less than one-tenth the amphibole fiber concentrations found in the urine when these same men drank only unfiltered water. This indicates that the amphibole fiber concentrations are associated with the type of drinking water. The results also indicate that mineral fibers can pass through the human gastrointestinal mucosa under normal conditions of the alimentary canal. Concentrations of amphibole fibers eliminated in urine represented approx 10^{-3} of the number of fibers ingested with drinking water. This ratio seems remarkably large and may be modified by further measurements. Diurnal variations in kidney function were not accounted for, and the magnitude for intersubject variability in gastrointestinal mucosa, kidney, and other tissue permeability to the particles is unknown. To the extent that some fibers are permanently retained by the body or are eliminated by other routes, however, the urine concentrations are an underestimate of the ingested fiber absorption. (49 refs)

- 79-2689 Comparative Cytotoxicity of Chrysotile and Crocidolite Asbestos in Hamster Tracheal Epithelial Cells (Meeting Abstract).** (Eng) Mossman, B. T. (Univ. Vermont Coll. Medicine, Burlington, VT 05405); Bradley, B. J.; Craighead, J. E. *Fed Proc* 38(3, part 2): 1352; 1979 (no refs)

- 79-2690 Pulmonary Response to Glass Fiber by Inhalation Exposure.** (Eng) Lee, K. P. (Haskell Lab. Toxicology and Industrial Medicine, E. I. du Pont de Nemours and Co., Inc., Wilmington, DE 19898); Barras,

C. E.; Griffith, F. D.; Waritz, R. S. *Lab Invest* 40(2): 123-133; 1979.

The ultrastructural details of the pulmonary response of several animal species to fiberglass exposure are described, with emphasis on a alveolar proteinosis. Male Charles River, C-D Sprague-Dawley-derived rats, male Syrian hamsters, and male albino guinea pigs were exposed to airborne glass fibers at a gravimetric concentration of 0.42 mg/liter for 6 hr/day, 5 days/wk, for 90 days. The number of dust particles $>5 \mu\text{m}$ long was $0.73 \times 10^6/\text{liter}$; the av diameter of the particles was approx $1.2 \mu\text{m}$. Most particles were approx $0.2\text{-}3.5 \mu\text{m}$ in diameter, and only 15% had a fibrous shape. Few fibers were longer than $10 \mu\text{m}$. The pulmonary response was characterized by macrophage reaction with proliferation of granular pneumocytes and alveolar proteinosis at 90 days of inhalation exposure. The light and ultrastructural alterations were similar to those of other experimental alveolar proteinosis or human alveolar proteinosis. The alveolar proteinosis disappeared at 1 yr postexposure, but focal dust cell accumulation with proliferating granular pneumocytes persisted throughout the 2-yr recovery period. No significant fibrosis or stromal changes were found in the dust-deposited areas. In hamsters and guinea pigs, most ferruginous bodies were developed from fibrous fibers but not from tiny dust particles. The tracheobronchial lymph nodes were markedly swollen and laden with dust cells. (35 refs)

- 79-2691 Glass Fiber-induced Foreign Body Granuloma.** (Ger) Lechner, W. (Dermatologische Klinik, Universität Würzburg, Josef-Schneider-Strasse 2, D-8700 Würzburg, W. Germany); Hartmann, A. A. *Hautarzt* 30(2): 100-101; 1979.

A 6-yr-old girl developed foreign body granulomas in the gluteal region, on the flexor sides of the lower legs, and in the dorsal parts of the feet. Microscopic examination of biopsy specimens revealed phagocytized glass fiber particles. The girl used to play in a shed in which fiberglass was stored. The foreign body granulomas regressed spontaneously without treatment. (5 refs)

- 79-2692 "Spontaneous" Neoplastic Transformation In Vitro: A Form of Foreign Body (Smooth Surface) Tumorigenesis.** (Eng) Boone, C. W. (Cell Biology Section, Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014); Takeichi, N.; del Ande Eaton, S.; Paranjpe, M. *Science* 204(4389): 177-179; 1979.

An attempt was made to determine whether the association in vitro between the smooth surface of a tissue culture vessel and explanted sc connective tissue cells could give rise to tumors. Explants of sc connective tissue from adult BALB/c mice in plastic petri dishes were serially sub-

cultured and tested for tumorigenicity by (1) sc implantation of cells attached to plastic plates ($1 \times 5 \times 10$ mm) and (2) sc injection of cells suspended in saline. Cells grown in vitro for ≥ 18 days before being implanted while attached to a plastic plate (2.4×10^4 to 3.4×10^5 cells/plate) formed tumors in the mice after 24-79 wk. The latent period before tumor appearance correlated inversely with the time spent by the cells in tissue culture. Cells injected in saline suspension (10-100 times the above number/plate) did not form tumors until >84 days in vitro; plates alone did not induce tumor formation within >1.5 yr of implantation. The tumors arising from the plate-attached cells were transplantable without plates and, histologically, appeared to be undifferentiated sarcomas. It is well-established that smooth-surfaced foreign bodies, regardless of their chemical composition, will produce sarcomas when transplanted sc in rodents. The data from these studies, particularly the decrease in tumor latent period with time spent in tissue culture, are interpreted as indicating that a smooth surface was acting as a carcinogen first in vitro (the surface of the tissue culture dish) and then in vivo (the surface of the plastic plate). (14 refs)

79-2693 Hyperplastic Polyps of the Gastric Mucosa Adjacent to Gastroenterostomy Stomas. (Eng)
Stemmermann, G. N. (Dept. Pathology, Kuakini Medical Center, 347 N. Kuakini St., Honolulu, Hawaii 96817); Hayashi, T. *Am J Clin Pathol* 71(3): 341-345; 1979.

In two men (aged 58 and 80 yr) with functionally active, nonatrophic oxyntic gastric mucosa, hyperplastic polyps developed on the gastric side of Billroth II and Billroth I anastomoses. The polyp associated with the Billroth II procedure was almost circumferential, sparing only the lesser curvature. That associated with the Billroth I operation was on the anterior wall from the lesser to the greater curvature. The operations had been performed 11 and 10 yr earlier for peptic ulcers of the gastroduodenal junction. The sites of polyp formation resembled those of experimental cancers in rats with Billroth I and Billroth II anastomoses and those of human cancers occurring ≥ 20 yr after gastrojejunostomy for benign disease. This suggests that stomal carcinomas and hyperplastic polyps each result from the reflux of enteric contents into the stomach remnant. (12 refs)

See also:

*(Rev.): 79-2401, 79-2456, 79-2457, 79-2465, 79-2466, 79-2467, 79-2472.

*(Chem.): 79-2492, 79-2538, 79-2629.

*(Immun.): 79-2812, 79-2823, 79-2826.

*(Path.): 79-2835, 79-2849, 79-2858, 79-2866, 79-2876.

*(Epid-Biom.): 79-2905, 79-2920, 79-2922, 79-2927, 79-2960, 79-2961, 79-2962, 79-2964, 79-2967, 79-2970, 79-2971.

VIRAL CARCINOGENESIS

- 79-2694 **tRNA-Trp as Primer for RNA-Directed DNA Polymerase Structural Determinants of Function.** (Eng) Cordell, B. (Dept. Biochemistry and Biophysics, Univ. California, San Francisco, CA 94143); Swanstrom, R.; Goodman, H. M.; Bishop, J. M. *J Biol Chem* 254(6): 1866-1874; 1979.

The specific interactions between the RNA-directed DNA polymerase of avian oncornavirus and the transfer RNA-Trp (tRNA-Trp) primer required for initiation of viral DNA synthesis in vitro were examined. Two distinct interactions, stable binding of the tRNA-Trp to the enzyme and initiation of viral DNA synthesis by the enzyme with tRNA-Trp as primer, were characterized as to the structure of tRNA-Trp required. Different structural features of the tRNA-Trp were necessary for each type of interaction. The entire primary structure and native conformation of tRNA-Trp were both required for binding to reverse transcriptase. Fragments of tRNA-Trp and intact tRNA-Trp in an altered conformation could not be bound by the enzyme using an assay that detects high-affinity binding between reverse transcriptase and native tRNA-Trp. In contrast, fragments of the tRNA-Trp molecule were able to serve as primers for viral DNA synthesis with normal efficiency, compared with intact tRNA-Trp. The fragments that initiate transcription must contain a minimum specific nucleotide sequence that extends from the 3' terminus of tRNA-Trp through 27 residues of the molecule. This portion of the tRNA-Trp molecule may be a major structural determinant of specificity in initiation. (32 refs)

- 79-2695 **Transformation-associated Cell Surface Antigens in Virus and Chemically Transformed Avian Cells.** (Eng) Bauer, H. (Fachbereich Humanmedizin, Institut für Virologie der Justus-Liebig-Universität Giessen, Frankfurter Strasse 107, D-6300 Giessen-Lahn, W. Germany); Ignjatovic, J.; Rubsamen, H.; Hayami, M. *Med Microbiol Immunol (Berl)* 164(1/3): 197-205; 1977.

The expression of cell-surface antigens after avian sarcoma virus (ASV) and avian leukosis virus (ALV) infection was reexamined using Japanese quail as effector and target cell donors. Cell-surface antigens were assayed by a cell-mediated cytotoxicity (CMC) test in which spleen cells from tumor-bearing or virus-immunized animals were reacted with in vitro grown target cells. Untransformed ALV-infected quail embryo cells (QEC) were killed according to a subgroup-specific antigenic pattern that was probably due to the subgroup (type)-specific determinant of gp85 expressed at the cell surface. ASV-transformed cells contained an additional antigen that did not seem to

be related to virus envelope glycoproteins because it was also demonstrable in QR(-) cells, an established quail cell line transformed by the Bryan high-titer strain of ASV that produces noninfectious (gp85-negative) particles. Virus-transformed cells shared surface antigens with chemically transformed cells from the same species. A weak cross-reaction between virus-transformed and primary embryonic cells was also found. Two-thirds of the spleens from tumor-bearing animals exerted a measurable cytotoxic effect, and the degree of cytotoxicity in some tests reached 80%. Preliminary data suggested that antibody-dependent (Fc receptor-bearing) cells are involved in this reaction. When virus-free cell extracts were prepared by KCl or nonionic detergent extraction and tested for their capacity to block CMC against individual target cells, four different antigens could be distinguished. One of these antigens was a subgroup-specific determinant of gp85 that was expressed in all productively infected or transformed cells. In the CMC test, the group-specific determinant was accessible only in cells transformed by nondefective ASV. An antigen, probably embryonic, was found in both chemically and virus-transformed cells and also in some untransformed embryo cells at low passage. Virus-transformed cells also expressed an antigen (transformation-specific antigen) that was found at the surface of or in extracts of chemically transformed or productively infected, untransformed cells. (13 refs)

- 79-2696 **Virally Induced Autochthonous Astrocytomas in the Mongolian Gerbil (*Meriones unguiculatus*): A System for Intracarotid (IC) Chemotherapy (Meeting Abstract).** (Eng) Serano, R. D. (Duke Univ., Durham, NC 27710); Bigner, D. D. *J Neuropathol Exp Neurol* 38(3): 340; 1979 (no refs)

- 79-2697 **Genome of Avian Myelocytomatosis Virus MC29: Analysis by Heteroduplex Mapping.** (Eng) Hu, S. S. (Dept. Microbiology, Univ. Southern California, 2011 Zonal Ave., Los Angeles, CA 90033); Lai, M. M.; Vogt, P. K. *Proc Natl Acad Sci USA* 76(3): 1265-1268; 1979.

The genome of avian myelocytomatosis virus MC29 was compared with that of the Prague strain of Rous sarcoma virus subgroup C (RSV PR-C) by heteroduplex mapping analysis using an electron microscope. The virion RNA of MC29 was hybridized to full-length DNA of RSV PR-C. The results showed that MC29-specific sequences for which there are no homologous counterparts in the RSV genome make up a contiguous stretch of RNA about 1.8 kilobases

long. These sequences are located approx in the middle of the genome, replacing the 3' half of the *gag* gene, the entire *pol* gene, and the 5' portion of the *env* gene, which are absent from MC29. This MC29-specific genetic substitution may contain information responsible for the leukemogenic transformation of the host cell. (26 refs)

- 79-2698 **Response of Hemopoietic Cells to Avian Acute Leukemia Viruses: Effects on the Differentiation of the Target Cells.** (Eng) Gazzolo, L. (Unite de Virologie, Place Joseph Renaut, 69008 Lyon, France); Moscovici, C.; Moscovici, M. G.; Samarut, J. *Cell* 16(3): 627-638; 1979.

An attempt was made to determine whether avian leukemia viruses (ALV's) interact with myeloid cells at one specific stage or at several stages of differentiation. Chicken bone marrow cells were infected with three ALV's, avian myeloblastosis virus (AMV), myelocytomatosis virus strain MC29, and Mill Hill 2 virus (MH2), and then cultured in agar in the presence of conditioned medium. Under these conditions, very few cells served as target cells for the three ALV's. Density gradient separation showed that ALV target cells were found primarily in the light density fractions and might be represented by cells committed to the mononuclear phagocyte pathway. Separation of bone marrow cells on the basis of their sedimentation velocity at unit gravity suggested that MC29 and AMV did not share the same target cells. In addition, analysis of surface receptors and functional markers characteristic of macrophages (Fc and complement receptors, phagocytosis, and immune phagocytosis) indicated that the ALV-transformed cells were blocked during differentiation. These results indicate that the transforming ability of ALV's interferes with the differentiation of their target cells. (35 refs)

- 79-2699 **Fractionation of Two Protein Kinases from Avian Myeloblastosis Virus and Characterization of the Protein Kinase Activity Preferring Basic Phosphoacceptor Proteins.** (Eng) Rosok, M. J. (Dept. Chemistry, Univ. Montana, Missoula, MT 59812); Watson, K. F. *J Virol* 29(3): 872-880; 1979.

Two protein kinase (PK) activities were fractionated from purified virions of avian myeloblastosis virus. The distinguishing characteristics of these two PK's included (1) their binding properties during purification by ion-exchange chromatography, (2) their estimated mol wt, and (3) their phosphoacceptor protein specificities. The PK that bound to the anion exchanger 2-(diethylamino)ethanol-cellulose (pH 7.2) had an estimated mol wt of 60,000-64,000 and preferred basic phosphoacceptor proteins. The PK that bound to the cation exchanger phosphocellulose (pH 7.2) had an estimated mol wt of 42,000 to 46,000 and preferred acidic phosphoacceptor proteins. The PK prefer-

ring basic phosphoacceptor proteins was further purified and characterized. Optimal transfer of phosphate catalyzed by this enzyme required a divalent metal ion, a sulfhydryl-reducing agent, and ATP as phosphate donor. Guanosine triphosphate was not an effective phosphate donor at concentrations comparable to ATP; and the cyclic nucleotides cyclic AMP and cyclic guanosine monophosphate neither stimulated nor inhibited protein phosphorylation by the PK. The specificity of the enzyme for basic phosphoacceptor proteins extended to proteins from avian myeloblastosis virus, in that the neutral to basic virion proteins p12, p19, and p27 served as phosphate acceptors. In addition, the PK also appeared to phosphorylate itself. The possibility that this virion-associated PK could disrupt normal cellular function is discussed. (30 refs)

- 79-2700 **Restitution of Fibroblast-transforming Ability in *src* Deletion Mutants of Avian Sarcoma Virus During Animal Passage.** (Eng) Vigne, R. (Dept. Microbiology, Univ. Southern California Sch. Medicine, 2011 Zonal Ave., Los Angeles, CA 90033); Breitman, M. L.; Moscovici, C.; Vogt, P. K. *Virology* 93(2): 413-426; 1979.

Evidence for the hypothesis that partial *src* deletion mutants can reacquire sarcomagenic properties during animal passage, probably by recombining with the *src*-related sequences of normal cells, is provided. The Schmidt-Ruppin strain of Rous sarcoma virus subgroup D (SR-D) gives rise to transformation-defective (*td*) mutants that have lost either all or almost all of the *src* gene (standard *td* or *std* viruses) or have only a partial deletion of *src*. These partial deletion mutants, designated *ptd* viruses, contain genomic RNA that is slightly larger than that of the *std* isolates, and heteroduplex analyses suggest that *ptd* viruses retain approx 25% of *src* from the 5' end of that gene. Several *ptd* isolates of SR-D were injected into newly hatched chickens and, after prolonged latent periods, caused sarcomas in about 30% of the birds. The tumors occurred in internal organs away from the site of injection. Infectious sarcoma viruses isolated from these growths have the envelope markers of subgroup D, are nondefective for replication, and induce a transformation in vitro that is morphologically distinct from that of SR-D. Electrophoresis of 35S genomic RNA from these recovered sarcoma viruses showed it to be of the size characteristic for nondefective sarcoma viruses. Fingerprint analysis of ³²P-labeled RNA from one of the new sarcoma viruses detected all oligonucleotides present in *ptd* viruses, the *src*-specific oligonucleotides of SR-D, and one new oligonucleotide not present in SR-D. This new RNase T₁-resistant oligonucleotide and the *src*-specific oligonucleotides identical to those of SR-D map close to the 3' end in the genome of the recovered sarcoma virus, which is the position expected for the *src* gene. These studies suggest that recovered avian sarcoma viruses have

acquired cellular sequences that are closely related in structure and function to the viral *src* gene. (35 refs)

- 79-2701 Relationship Between Changes in the Calcium Dependent Regulatory Protein and Adenylate Cyclase During Viral Transformation. (Eng) LaPorte, D. C. (Dept. Pharmacology, Univ. Washington, Seattle, WA 98195); Gidwitz, S.; Weber, M. J.; Storm, D. R. *Biochem Biophys Res Commun* 86(4): 1169-1177; 1979.

Because of previous observations that the levels of calcium-dependent regulatory protein (CDR) in transformed chicken embryo fibroblasts (CEF) are higher in soluble and membrane fractions compared with untransformed cells, the kinetics for changes in CDR levels, hexose transport, and adenylate cyclase (AC) activity during transformation were examined using a temperature-sensitive mutant of Rous sarcoma virus (tsNY68). Decreases in AC activity and increased hexose transport accompanying transformation occurred with half-lives of approx 7-8 hr. Increases in CDR occurred much slower, with a half-life of 17 hr. Thus, the increase in CDR levels accompanying the transformation of CEF by RSV is a late event that occurs after the decrease in AC activity and increased hexose transport. Therefore, the decline in AC activity cannot be due to changes in the amount of CDR. In addition, differences in AC calcium sensitivity that were found in normal and transformed membranes were not due to changes in the amount of CDR, since these membrane preparations contained comparable levels of CDR. It seems likely that Ca^{2+} (CDR) cannot modulate AC activity in transformed membranes because of alterations in the AC system or membrane environment. (11 refs)

- 79-2702 Detection of the Viral Sarcoma Gene Product in Cells Infected with Various Strains of Avian Sarcoma Virus and of a Related Protein in Uninfected Chicken Cells. (Eng) Brugge, J. S. (Dept. Pathology, Univ. Colorado Medical Center, Denver, CO 80262); Collett, M. S.; Siddiqui, A.; Marczyńska, B.; Deinhardt, F.; Erikson, R. L. *J Virol* 29(3): 1196-1203; 1979.

Sera from marmosets bearing tumors induced by the Bryan or Schmidt-Ruppin (SR) strains of avian sarcoma virus (ASV), TBM sera, were used to screen cells transformed by four ASV strains for the presence of the polypeptide product of the *src* gene of SR-ASV, a 60,000-dalton phosphoprotein designated pp60src. TBM sera contained antibody that precipitated the transforming gene product from cells transformed by the SR, Bryan, Prague, or Bratislava strains of ASV. In contrast, rabbits bearing tumors induced by the Bratislava or Bryan strains or hamsters with SR-ASV-induced tumors did not produce antibody to pp60src from any ASV strain. The immune response to pp60src was found to be dependent on the

species of tumor-bearing host and, in the case of rabbits, also on the virus strain used for tumor induction. The immunological basis for these differences in response to pp60src is not known. The 60,000-dalton polypeptides immunoprecipitated with TBM serum from cells transformed by each of the four virus strains were phosphoproteins. One-dimensional peptide mapping by limited proteolysis revealed that the pp60src proteins were structurally very similar, but not identical. Furthermore, all the viral pp60src proteins had an associated phosphotransferase activity. In addition to detecting the viral *src* proteins, TBM serum was able to immunoprecipitate an antigenically related protein from normal uninfected avian cells. These data suggest that the mechanism of ASV-induced sarcomagenesis by all four virus strains may be mediated by protein phosphorylation. However, differences in the nature of cellular transformation by the various ASV strains may be reflected in the slight variations in the primary structure or secondary protein modification of the respective pp60src proteins. These variations may result in slight changes in the specificity of phosphorylation of cellular target polypeptides phosphorylated by the transforming proteins. (16 refs)

- 79-2703 An Avian Oncovirus Mutant Deficient in Genomic RNA: Characterization of the Packaged RNA as Cellular Messenger RNA. (Eng) Gallis, B. (Dept. Biochemistry, Univ. Washington, Seattle, WA 98195); Linial, M.; Eisenman, R. *Virology* 94(1): 146-161; 1979.

The RNA from SE 21Q1B virions was analyzed by in vitro translation and by characterization of the in vitro-synthesized proteins by physical, chemical, and immunological techniques. SE 21Q1b is a noninfectious recombinant avian oncovirus [Prague strain of Rous sarcoma virus subgroup C (PR-RSV-C) x Rous-associated virus type O] continuously produced by a line of transformed quail cells. Virions of SE 21Q1b contain 1%-2% of the normal amounts of RSV-specific RNA found in PR-RSV-C virions and substantial quantities of heterogeneously sedimenting nonviral RNA. RNA extracted from purified virions of SE 21Q1b is as efficient a template for amino acid incorporation in a messenger RNA (mRNA)-dependent reticulocyte lysate as oligo(dT)-cellulose-purified cellular mRNA. Virion RNA from SE 21Q1b serves as a template in vitro for a small amount of the precursor to the internal structural proteins (*gag* proteins). Unlike RNA from other avian oncoviruses, SE 21Q1b RNA makes, in addition, several proteins whose sizes on sodium dodecyl sulfate-polyacrylamide gels resemble those of a number of uninfected quail fibroblast proteins. The proteins range in size from 15,000 to 22,000 daltons. Immunoprecipitation of proteins synthesized in vitro from SE 21Q1b virion RNA with antisera against the *gag*, *pol*, and *env* gene products shows that this RNA serves as a template for one viral gene product, the recombinant

gag gene precursor of 72,000 daltons (Δ Pr76). Addition of lysed virus to proteins made in vitro from PR-C RNA or RNA from PR-E-95c, a recombinant whose *gag* gene structure is similar to that of PR-E-21Q1b, results in the cleavage of *gag* protein precursors into several of the internal structural proteins. On the other hand, products of translation of PR-E-21Q1b RNA are not detectably cleaved into the internal structural proteins, suggesting that a very low amount of PR-E-21Q1b translation products are *gag*-related. A 37,000-dalton protein constitutes about 15% of the total protein synthesized from SE21Q1b RNA. Data from serological and peptide mapping studies indicate that this protein is unrelated to the virion *gag*, *pol*, or *env* proteins. However, the major tryptic peptide of this protein appears to be identical to the major peptide of a prominent quail cell protein having the same apparent mol wt as the in vitro translation product. Thus, several lines of evidence suggest that SE21Q1b virions contain substantial amounts of functional cellular mRNA's. (59 refs)

- 79-2704 **Recombinant Avian Oncoviruses. II. Alterations in the *gag* Proteins and Evidence for Intragenic Recombination.** (Eng) Shaikh, R. (Dept. Pathology, Harvard Medical Sch., Boston, MA 02115); Linial, M.; Brown, S.; Sen, A.; Eisenman, R. *Virology* 92(2): 463-481; 1979.

The internal structural (*gag*) proteins of recombinant avian oncoviruses selected for the *env* gene of Rous-associated virus-O [(RAV-O) an endogenous chicken virus] and the *src* gene for the Prague C strain of Rous sarcoma virus (PR-RSV-C) were examined. Eight of ten clones of recombinant viruses synthesized altered *gag* proteins. Therefore the proteins of a representative recombinant clone, PR-E-95c, were examined in detail by gel electrophoresis and tryptic peptide mapping, to distinguish between the *gag* proteins of the two parental viruses and to determine from which virus the proteins of the recombinant virus were derived. PR-E95c virions contained p27, an electrophoretically distinguishable variant of p27 that is found in isolates of RAV-O. This recombinant virus also contained p12/15, which is electrophoretically indistinguishable from the p12/15 of both of the parental viruses. However, tryptic peptide analysis of p15 indicated that PR-E-95c had inherited PR-RSV-C-specific p15 sequences. These observations suggested that at least one cross-over had occurred between p15 and p27 in PR-E-95c. The PR-E-95c proteins and those of the parental viruses differed markedly in that the recombinant lacked polypeptides migrating in the position of p19 and contained two novel polypeptides termed p19 α (mol wt 20,000) and p19 β (mol wt 15,000). Both polypeptides were phosphorylated and shared antigenic determinants and some tryptic peptides with parental p19. As determined by peptide analysis and radioimmunoassay, these p19-related proteins contained information from both parental viruses, suggesting that PR-E-95c had another cross-over within p19. The altered p19 proteins

bound to viral RNA specifically and were associated with genomic RNA in the virion. Neither the stability nor the specific infectivity of the recombinant viruses appeared to be significantly affected by the altered proteins. (43 refs)

- 79-2705 **Comparison of the Small RNAs of Polymerase-deficient and Polymerase-positive Rous Sarcoma Virus and Another Species of Avian Retrovirus.** (Eng) Sawyer, R. C. (Cancer Res. Labs., Woodhaven, NY 11421); Hanafusa, H. *J Virol* 29(3): 863-971; 1979.

The role of the polymerase reverse transcriptase molecule in the selection of small RNA's for packaging was studied using chicken cells transformed by the polymerase-positive Bryan strain of Rous sarcoma virus (BH-RSV) and the polymerase-negative strain (BH-RSV α). The cultures were labeled with phosphorus-32, and the viral RNA's were purified and separated by two-dimensional polyacrylamide gel electrophoresis. The gel pattern of small RNA's of BH-RSV represented a specific subset of host cell small RNA's and was indistinguishable from those of the nondefective Schmidt-Ruppin strain of RSV and two different leukemia viruses, Rous-associated virus 1 (RAV-1) and RAV-2. Virions of BH-RSV α incorporated an unselected population of small RNA's identical to total chicken cell small RNA. Virions of reticuloendotheliosis virus, which contains a reverse transcriptase unrelated to that of the avian leukemia and sarcoma viruses, contained a distinctly different population of small RNA's although both the avian leukemia and sarcoma and the reticuloendotheliosis viruses were grown in chicken cells. Since the primer for avian leukemia and sarcoma virus RNA-dependent DNA synthesis is a host cell transfer RNA, the differences in reverse transcriptase small RNA selection may help explain the failure of different species of retroviruses to complement for the reverse transcriptase. (43 refs)

- 79-2706 **Purification and Partial Characterization of a Protein Kinase from the Prague-C Strain of Rous Sarcoma Virus.** (Eng) Hizi, A. (Dept. Cell Biology and Histology, Tel Aviv Univ. Sch. Medicine, Tel Aviv, Israel); Wunderli, W.; Joklik, W. K. *Virology* 93(1): 146-158; 1979.

A protein kinase was purified from Rous sarcoma virus strain Prague-C by chromatography on 2-(diethylamino)ethanol-cellulose and phosphocellulose, followed by glycerol density gradient centrifugation. The purified enzyme had a sedimentation coefficient of about 2.5S, corresponding to a mol wt of about 11,000. The sedimentation coefficient of the enzyme in virus lysates was 3.9S, corresponding to a mol wt of about 45,000. Comparison of the enzyme's chymotryptic and CNBr cleavage products with those of p15, p12, and p10 showed that it

was not identical with any of these polypeptides. The enzyme required divalent metal ions for activity, Mg^{2+} being preferred to Mn^{2+} . It did not depend on cyclic nucleotides for activity. Its K_m for ATP and guanosine triphosphate (GTP) were 40 and 870 μM respectively, but the V_{max} at saturating triphosphate concentrations was 5 x higher with GTP than with ATP. The most efficient exogenous phosphate acceptor was casein, followed by phosphatidylserine and arginine-rich histone. The most efficient phosphate acceptors in virus lysates were two small polypeptides that were probably the enzyme subunit itself and p12, followed by p19 and unidentified >50,000-mol-wt proteins that were probably traces of host cell polypeptides in the virus envelope. In virus lysates that had been heated for 2 min at 90 C and supplemented with purified kinase, the most active phosphate acceptor was an unidentified protein with a mol wt of about 38,000. (28 refs)

- 79-2707 Infectivity of Proviral DNA from Avian Sarcoma Virus-transformed Mammalian Cells.** (Eng) Catala, F. (Institut Curie, Faculte des Sciences, Orsay, France); Vigier, P. *J Virol* 29(3): 833-839; 1979.

The number of Rous viral genomes in the cellular DNA of two subclones (RS2/3, RS2/6) derived from the same clone of hamster BHK-21 cells transformed by Rous sarcoma virus (RSV) was determined by hybridization with viral complementary DNA, and the capacity of the cellular DNA to infect (transfect) chicken embryo fibroblasts (CEF) was compared before and after shearing this DNA to the proviral genome size (6×10^6 to 7×10^6 daltons). The two subclones differed widely in their capacity to give rise to virus (inducibility) after fusion with CEF and in their level of viral protein expression. Subclone RS2/3 was the most inducible and expressed appreciable levels of viral proteins; no proteins were detected in subclone RS2/6. Cells of both subclones contained a single copy of RSV DNA and yielded infectious DNA. However, whereas transfection of CEF was successful with both unsheared ($\geq 18 \times 10^6$ daltons) and sheared DNA from subclone RS2/3, transfection with DNA from subclone RS2/6 succeeded only with sheared DNA. The infectivity of the sheared DNA approximated that of sheared or unsheared RS2/3 DNA. The lack of infectivity of unsheared RS2/6 DNA may be explained by a hypothesis previously proposed to account for the lack of infectivity of DNA from certain chicken cells producing spontaneously low amounts of Rous-associated virus-O (RAV-O) and resistant to exogenous RAV-O infection; that is, that the viral genome (proviral DNA) is linked to a *cis*-acting control element that blocks its expression. In RS2/6 cells, this linkage might originate from translocation of cellular DNA containing the single proviral copy. (33 refs)

- 79-2708 Formation of Rous Associated Virus-60: Origin of the Polymerase Gene.** (Eng) Sawyer,

R. C. (Cancer Res. Labs., Woodhaven, NY 11421); Rettenmier, C. W.; Hanafusa, H. *J Virol* 29(3): 856-862; 1979.

The DNA of normal chicken embryos contains sequences related to the avian leukosis-sarcoma viruses whose RNA-dependent DNA polymerase is encoded by as the *pol* gene. The nature of the endogenous virus *pol* gene in chicken cells was investigated by testing its ability to participate in genetic recombination. Rous-associated virus-60 (RAV-60) recombinant viruses isolated after infection of chicken cells with strains tsLA33PR-B or tsNY21SR-A, both of which produce a temperature-sensitive DNA polymerase, also possessed the temperature-sensitive lesion. These results are consistent with the hypothesis that the endogenous viral information used for the generation of RAV-60 is deficient in at least part of the *pol* gene and that the defect includes that portion represented by the lesions in NY21 and LA337. The frequency of polymerase-negative BH-Rous sarcoma virus α formation was not affected by the levels of endogenous viral expression, which suggests that the α defect is not derived from the endogenous *pol* gene. (47 refs)

- 79-2709 Messenger Activity of Virion RNA for Avian Leukosis Viral Envelope Glycoprotein.** (Eng) Stacey, D. W. (Rockefeller Univ., New York, NY 10021) *J Virol* 29(3): 949-956; 1979.

The RNA from particles of Rous-associated virus type 2 (RAV-2) was analyzed by an intracellular assay for viral envelope glycoprotein (*env*) messenger. RAV-2 RNA was microinjected into cells infected by the *env*-deficient Bryan strain of Rous sarcoma virus [(RSV(-) cells)]. Only when the injected RNA could be translated by the recipient cells to produce viral envelope glycoprotein was the *env* deficiency of the RSV(-) cells complemented, enabling them to release focus-forming units (FFU). The 21S RNA from the RAV-2 virus particle promoted the release of numerous FFU from RSV(-) cells, but the major 35S virion RNA species was inactive. The *env* messenger activity sedimented as a sharp peak with high specific activity. RNase T1-generated fragments of virion 35S RNA were unable to promote the release of infectious virus from RSV(-) cells, which suggested that the active molecule was *env* messenger encapsulated by the virus particle directly from the cytoplasm of infected cells. Approx 95% of the *env* messenger was associated with the virion high-mol-wt RNA complex. The temperature required to dissociate *env* messenger from this complex was the same as that required to disrupt the complex itself. Virion high-mol-wt RNA that was associated with *env* messenger sedimented slightly more rapidly than the bulk virion RNA; this was the strongest evidence that the 21S messenger had been encapsulated directly from the infected cells. These data, along with the prolonged expression of *env* messenger after injection into RSV(-) cells, raise the possibility that virus-encapsulated *env* messenger can become expressed within subsequently infected cells. (33 refs)

- 79-2710 Production of Avian Oncoviral Subgroups after Multiple Infection.** (Eng) Khoury, A. T. (Wistar Inst. Anatomy and Biology, Philadelphia, PA 19104); Hanafusa, H.; Namy, C. A. *J Virol* 29(3): 926-937; 1979.

An attempt was made to determine if there is a limit to the number of different proviral DNA's that can be expressed by a single chicken embryo fibroblast. Fibroblasts were infected with one to three subgroups of Rous-associated virus, which is a nontransforming avian oncovirus, then superinfected with a transforming virus, Rous sarcoma virus, of a different subgroup. The subgroups of viruses released by the resulting clones were analyzed. When two viral subgroups were used for preinfection, all the resulting clones produced transforming virus particles having the subgroup of the superinfecting virus, and most clones produced transforming virus particles of all the infecting viral subgroups. However, when cells were preinfected with three viral subgroups, many of the resulting clones did not produce transforming virus particles having the subgroup of the superinfecting virus, and only 1/23 clones produced transforming particles of all the infecting viral subgroups. DNA annealing experiments showed that cells infected with three or four viral subgroups had an additional 8-20 copies of proviral DNA per cell. Finally, most clones resulting from cells simultaneously infected with three or four viral subgroups were able to produce virus of all infecting subgroups. It appears that the number of exogenous oncoviral *env* genes that can be expressed by a single cell is limited and in the range of 4 to 8-20 per cell. (24 refs)

- 79-2711 RNA-directed DNA Polymerase from Particles Released by Normal Goose Cells.** (Eng) Bauer, G. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706); Temin, H. M. *J Virol* 29(3): 1006-1013; 1979.

Cultured goose embryo cells released particle-associated RNA-directed DNA polymerase and RNase H activities that required the presence of Nonidet P-40 for detection. The particles were not infectious and did not have endogenous DNA synthesis. The goose particle DNA polymerase was related to the DNA polymerase of spleen necrosis virus with respect to size, and it was inhibited by IgG's directed against spleen necrosis virus DNA polymerase. However, goose cell DNA did not contain endogenous reticuloendotheliosis virus nucleotide sequences. (16 refs)

- 79-2712 Transformation by Reticuloendotheliosis Virus: Development of a Focus Assay and Isolation of a Nontransforming Virus.** (Eng) Hoelzer, J. D. (Dept. Microbiology, Univ. Texas at Austin, Austin, TX 78712); Franklin, R. B.; Bose, H. R. *Virology* 93(1): 20-30; 1979.

A focus assay for quantitating in vitro transformation by oncogenic reticuloendotheliosis virus (REV) was developed in Japanese quail embryo fibroblast cultures. The titer of the transforming virus detected in the in vitro focus-forming (FF) assay correlated with the development of reticuloendotheliosis in chickens. The titration pattern of REV FF appeared to follow two-hit kinetics. A non-transforming virus, designated REV-associated virus (REV-A), was present in REV stocks in 100- to 1000-fold excess over the transforming virus. REV-A induced an initial cytopathic effect in chick and quail embryo fibroblast cultures; the surviving cells continue to divide, leading to the development of a persistently infected culture. The persistently infected cultures were morphologically indistinguishable from uninfected avian fibroblast cultures. REV-A interfered with superinfection by the oncogenic virus. The addition of REV-A to oncogenic preparations of REV increased FF and changed the titration pattern from two-hit to one-hit kinetics, indicating that REV is defective. The identification of this defect may help in determining the specific viral products responsible for leukemogenic transformation. (42 refs)

- 79-2713 Transcription of the Marek's Disease Virus Genome in a Nonproductive Chicken Lymphoblastoid Cell Line.** (Eng) Silver, S. (Lab. Molecular Virology, Life Sciences, Inc., St. Petersburg, FL 33710); Tanaka, A.; Nonoyama, M. *Virology* 93(1): 127-133; 1979.

Transcription of the Marek's disease virus (MDV) genome in virus-nonproducing MKT-1 cells, a lymphoblastoid cell line derived from a Marek's disease tumor, was studied by analyzing the hybridization kinetics of ³H-labeled MDV DNA with unlabeled RNA extracted from whole cells or from polyribosomes. From 12% to 14% of the viral DNA template was transcribed, and only a portion (60%-70%) of the viral-specific RNA sequences found in whole cells could be detected in the polyribosomal fraction. Treatment of MKT-1 cells with 5'-iododeoxyuridine (IUdR) induced transcription of the viral genome, so that 42% of the MDV RNA template was transcribed. This represents nearly the same degree of transcription as in chicken embryo fibroblasts productively infected with MDV. Viral antigens were also produced following IUdR induction, but viral DNA replication remained restricted. Only 60%-70% of the viral-specific RNA sequences in whole cells of IUdR-treated MKT-1 cells were found in the polyribosomal fraction. The data suggest the existence within MKT-1 cells of a posttranscriptional control mechanism that selectively excludes certain RNA transcripts from stable association with the polyribosomes and that may be responsible, in part, for the repressed expression of viral genetic information in the cells. (20 refs)

- 79-2714 Integration and Expression of Mouse Mammary Tumor Virus Genes in Mice Strains Ex-**

hibiting High and Low Tumor Incidence (Meeting Abstract). (Eng) Groner, B. (Swiss Inst. Experimental Cancer Res., Chemin des Boveresses, CH-1066 Epalinges/Lausanne, Switzerland); Hynes, N. E. *Hoppe Seylers Z Physiol Chem* 360(3): 271; 1979 (no refs)

79-2715 Autogenous Antibodies Against the Murine Mammary Tumor Virus in Strains of Mice with Low Incidences of Mammary Tumors. (Eng) Michalides, R. (Div. Virology, Antoni van Leeuwenhoekhuis, Netherlands Cancer Inst., Plesmanlaan 121, Amsterdam, Netherlands); Wagenaar, E.; Nüsse, R. *J Natl Cancer Inst* 62(4): 935-941; 1979.

A radioimmunoprecipitation assay for murine mammary tumor virus (MuMTV) was used to detect naturally occurring antibodies against MuMTV in three groups of highly inbred mouse strains. (1) Strains GR and C3H have high incidences of mammary tumors. Antibodies against MuMTV were detected in the sera of females of these strains at early ages. (2) Strains C3Hf, RIII, and BALB/c have low incidences of mammary tumor with an intermediate MuMTV expression. Some females of these strains developed antibodies against MuMTV. Hormone treatment (estrogen, progesterone, and prolactin) increased the proportion of mice carrying antibodies against MuMTV. (3) Strains O20, C57BL, and GR-*Mtv*⁻ are MuMTV-free. No antibodies against MuMTV were detected in the sera of these mice. Antibodies were detected, however, after hormone treatment. The presence of a natural humoral immunity toward MuMTV appeared to be related to the expression of MuMTV in the various mouse strains. (27 refs)

79-2716 In Vitro Selection of High-infectious, Leukemogenic Virus from Low-infectious, Non-leukemogenic Type C Virus from a Malignant ST/a Mouse Cell Line. (Eng) Willumsen, B. M. (Sidney Farber Cancer Inst., Boston, MA 02115) *J Virol* 29(3): 1213-1220; 1979.

Low-infectious, nontransforming, C-type virus was isolated from an in vitro spontaneously transformed ST/a mouse cell line, ST-L1. The virus released by the ST-L1 cells was NB-tropic and XC-. It gave rise to very small peroxidase antibody plaques (PAP) in cultures that initially were nonproducing. Sodium dodecyl sulfate (SDS)-polyacrylamide gels of the structural proteins of the ST-L1 virus showed an envelope glycoprotein with an apparent mass of 65,000 daltons (65K). SC-1, BALB/3T3, and NIH/3T3 mouse cells could be productively infected with cell-free supernatants from the ST-L1 cell line; however, virus was detected in supernatant fluids only after two to four subcultures of the infected cells. The virus thus produced was XC+ and a large plaque former. The virus released from infected SC-1 cells was N-tropic, whereas the

viruses from infected NIH/3T3 and BALB/3T3 cells were NB-tropic. The structural proteins of the N- and NB-tropic viruses could be distinguished on SDS polyacrylamide gels, the major dissimilarity being a difference in the mobility of the p30. All these viruses had an envelope glycoprotein with an apparent mol wt of 70K. The infectivity of the viruses, measured as PAP per nanogram of p30, was 30- to 60-fold lower for the virus released from the ST-L1 cell line than that of the viruses released after passage in SC-1, NIH/3T3, and BALB/3T3 cells. None of the viruses could infect rabbit or mink cells. Inoculation of the viruses into newborn mice showed that the ST-L1 virus was nonleukemogenic, whereas the NB-tropic virus selected from this virus after passage in BALB/3T3 or NIH/3T3 cells was highly leukemogenic. Viruses isolated from leukemic animals were indistinguishable with respect to host range and protein mobilities in SDS gels from the infecting viruses. Although the SC-1-selected virus was highly infectious in vitro, it was only weakly, if at all, leukemogenic. (33 refs)

79-2717 Differential Distribution of Mouse Mammary Tumor Virus-related Sequences in the DNA's of Rats. (Eng) Drohan, W. (Lab. Viral Carcinogenesis, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014); Schlom, J. *J Natl Cancer Inst* 62(5): 1279-1286; 1979.

An attempt was made to determine if mouse mammary tumor virus (MuMTV)-related proviral sequences are present in the DNA of the laboratory rat (*Rattus norvegicus*). Radioactively labeled MuMTV 60S-70S RNA, obtained from virions grown in both murine and feline cells, was employed in molecular hybridization experiments to detect the MuMTV-related sequences. With the use of relaxed conditions of hybridization and assay for RNA-DNA duplexes, all strains of laboratory rats and feral rats examined were shown to possess endogenous MuMTV-related DNA sequences in the low repetitive range. These sequences were related to approx 20% of the MuMTV genome and exhibited a melting temperature (T_m) approx 5 C lower than MuMTV-specific proviral sequences in murine (*Mus musculus*) DNA's. Certain colonies of F344 (Fischer) rats contained animals whose DNA's possessed additional MuMTV-related sequences. These sequences were related to the non-germline-transmitted, tumor-associated (TA) sequences of the highly oncogenic MuMTV(C3H). They were found in the DNA of some F344 rats and a cloned established F344 rat embryo cell line at a frequency of approx one copy per haploid genome, and they exhibited a T_m 9 C lower than that of hybrid duplexes formed between radioactive MuMTV TA-sequence RNA and C3H mouse mammary tumor DNA. The DNA's of rats, therefore, contained two sets of sequences that were related to sequences of the MuMTV genome: one set was germline-transmitted, whereas the

other set appeared to be transmitted in some rats via a non-germ line or infectious process. (23 refs)

- 79-2718 **Cell-Free Synthesis of Mouse Mammary Tumor Virus Pr77 from Virion and Intracellular mRNA.** (Eng) Dahl, H. H. (Section Medical Enzymology and Molecular Biology, Univ. Amsterdam, Amsterdam, Netherlands); Dickson, C. *J Virol* 29(3): 1131-1141; 1979.

Mouse mammary tumor virus (MuMTV) was purified from two cell lines (GR and Mm5MT/c1), and the genomic RNA was isolated and translated in vitro in cell-free systems derived from mouse L cells and rabbit reticulocytes. The major translation product in both systems was a protein with a mol wt of 77,000 daltons (77K). Several other products were also detected, 110K protein and small amounts of a 160K protein. All three polypeptides were specifically immunoprecipitated by antiserum raised against the major core protein of MuMTV (p27), but they were not precipitated by antiserum against the virion glycoprotein gp52. Analysis of the in vitro products by tryptic peptide mapping established their relationship to the virion nonglycosylated structural proteins. The 77K-dalton polypeptide was found to be similar, if not identical, to an analogous precursor isolated from MuMTV-producing cells. Peptide mapping of the 110K protein showed that it contains all of the methionine-labeled peptides found in the 77K protein plus some additional peptides. It is concluded that the products synthesized in vitro from genomic MuMTV RNA are related to the nonglycosylated virion structural proteins. Polyadenylic acid-containing RNA from MuMTV-producing cells also directed the synthesis of the 77K polypeptide in the L-cell system. When this RNA preparation was first fractionated by sucrose gradient centrifugation, the 77K protein appeared to be synthesized from messenger RNA with a sedimentation coefficient of 25S-35S. (56 refs)

- 79-2719 **Antigenic Properties and Molecular Weights of Murine Leukemia Virus-binding Proteins.** (Eng) Baird, S. M. (Veterans Admin., VA Medical Center, San Diego, CA) *J Immunol* 122(4): 1389-1396; 1979.

A murine T lymphoma cell line, WEHI-22, was studied for the presence of murine leukemia virus-binding proteins and for the presence of cell-surface molecules that share antigens with mouse immunoglobulins (Ig's). Surface radioiodination, detergent disruption, and immunoprecipitation techniques identified a 60,000- to 70,000-dalton polypeptide that was recognized by chicken anti-mouse Ig serum. This serum did not detect a molecule similar to the WEHI-22 molecule on normal thymocytes. In competition experiments, this molecule cross-reacted with highly purified mouse IgM myeloma proteins. A cell-

surface molecule of similar size was shown to bind to murine leukemia viruses. Precipitation of the WEHI-22 cell surface material with chicken anti-mouse Ig removed the material binding to the leukemia viruses. (25 refs)

- 79-2720 **Genetic Linkage of C3H/HeJ and BALB/c Endogenous Ecotropic C-Type Viruses to Phosphoglucomutase-1 on Chromosome 5.** (Eng) Ihle, J. N. (Cancer Biology Program, Frederick Cancer Res. Center, Frederick, MD 21701); Joseph, D. R.; Domotor, J. *J. Science* 204(4388): 71-73; 1979.

Serological assays of backcrossed mice were used to demonstrate the apparent allelism of BALB/c and C3H/HeJ endogenous ecotropic C-type viral loci on chromosome 5. In the backcross C57BL/6 x (C57BL/6 x C3H/HeJ), there is a clear segregation of the C3H/HeJ phenotype, in that individual mice are either antibody-positive early in life and remain antibody-positive or they remain antibody-negative. In 208 backcrossed mice, 51.4% were of the C3H/HeJ phenotype, which suggests that a single gene locus controls the serological phenotype. Using the same backcross, no apparent linkage of the C3H/HeJ phenotype with a variety of isoenzyme markers (serum esterase-1, serum esterase-3, glucose-6-phosphate dehydrogenase-1, the agouti locus, the locus for the β chain of Hb, the histocompatibility locus, or malate dehydrogenase) was found. However, linkage was apparent with phosphoglucomutase-1 (*Pgm-1*), in that only 40/141 mice examined were of the recombinant phenotype. These results demonstrate that the C3H/HeJ viral locus (*C3v*) is on chromosome 5 and is linked to *Pgm-1*. Further experiments demonstrated that the BALB/c and C3H/HeJ loci are either allelic or closely linked on chromosome 5, in contrast to the C57BL/6 viral locus, which is not allelic and probably is not linked to the C3H/HeJ locus. (22 refs)

- 79-2721 ***Fv-1* Restriction of Xenotropic and Amphotropic Murine Leukemia Virus Genomes Phenotypically Mixed with Ecotropic Virus.** (Eng) Ishimoto, A. (Lab. Viral Diseases, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Hartley, J. W.; Rowe, W. P. *Virology* 93(1): 215-225; 1979.

Xenotropic murine leukemia virus (MuLV) genomes rendered capable of infecting mouse cells by phenotypic mixing with an ecotropic MuLV exhibited the *Fv-1* restriction pattern of that ecotropic virus. Like *Fv-1*-restricted ecotropic viruses, xenotropic genomes phenotypically mixed with N-tropic ecotropic MuLV showed two-hit dose-response kinetics when titrated on restrictive *Fv-1b* cells; the kinetics were converted to one-hit by the addition N-tropic ecotropic virus. When SC-1 cells chronically infected with N-tropic amphotropic MuLV were superinfected with B-tropic ecotropic virus, each genome tended to maintain

its homologous tropism, but with intermediate N:B infectivity ratios that changed between the acute and chronic phases of infection. In clonal lines chronically producing both viruses, the relative N- or B-tropism of the two genomes tended to vary in the same direction, and this correlated with the genome that was produced in greater abundance. The results suggest that (1) there is some recognition of amphotropic and ecotropic genomes by their homologous *Fv-1* tropism determinants, and (2) that the tropism of the virions in the mixed harvest is determined by this recognition factor and by the relative abundance of the two types of determinant. (30 refs)

- 79-2722 Defective Retrovirus-like 30S RNA Species of Rat and Mouse Cells Are Infectious if Packaged by Type C Helper Virus.** (Eng) Scolnick, E. M. (Lab. Tumor Virus Genetics, NCI, Bethesda, MD 20014); Vass, W. C.; Howk, R. S.; Duesberg, P. H. *J Virol* 29(3): 964-972; 1979.

Cell lines that express high levels of a 30S defective retroviruslike (DRV) RNA were used to form pseudotypes of the 30S RNA's with helper-independent C-type viruses. A pseudotype virus complex containing a mouse 30S subunit was transmitted to rat cells, and a pseudotype virus complex containing a rat 30S subunit was transmitted to bat cells. In addition, a rat 30S subunit was isolated in non-producer bat cells without detectable expression of the helper-independent C-type virus used to pseudotype it. The transmissibility of an endogenous rat 30S subunit to heterologous nonproducer cells extends the similarity between the 30S subunit and other replication-defective retroviruses and is consistent with the provirus hypothesis. The function of the 30S DRV RNA in mouse and rat cells is not known, and cells expressing these RNA's and releasing them as viral pseudotypes do not appear morphologically transformed in culture. Relatively low levels of rat 30S DRV RNA were detected in rat cells and bat cells, and it is possible that morphological transformation may depend on a critical threshold concentration. It is also possible that the oncogenicity in animals of certain mouse and rat cell lines considered untransformed in culture may be related to the presence and concentration of 30S DRV RNA, which may function directly in neoplastic transformation in the animal or could become active only by recombination with some other viral or cellular gene. (27 refs)

- 79-2723 Virus-like 30S RNA in Mouse Cells.** (Eng) Besmer, P. (Hadassah Medical Sch., Hebrew Univ., Jerusalem, Israel); Olshevsky, U.; Baltimore, D.; Dolberg, D.; Fan, H. *J Virol* 29(3): 1168-1176; 1979.

Particles released from JLS-V9 cells, a cell line derived from BALB/c mice, after induction with bromodeox-

yuridine (BrdU) were characterized biochemically. After heat denaturation, most of the RNA in particles released after BrdU treatment (BU-V9 virus) migrated as 30S RNA material during electrophoresis through agarose gels. This viruslike 30S RNA (VL30) could be packaged in virus particles, could polymerize to a 50S form, and could be reverse-transcribed. Annealing experiments of BU-V9 complementary DNA (cDNA) to cytoplasmic RNA's from other cells revealed that VL30 RNA sequences are expressed in other mouse cells, eg, NIH/3T3, but not in heterologous (rat, rabbit, or mink) cells. Fingerprint analysis and hybridization studies indicated that VL30 RNA does not have homology with the standard nondefective murine leukemia viruses (MuLV's). Upon superinfection with a nondefective MuLV or upon induction of endogenous virus with BrdU, VL30 RNA was rescued into virions by phenotypic mixing. When VL30 RNA was rescued by BrdU induction, it was mainly as a 50S complex, but when it was rescued by superinfection, VL30 was also found in 70S RNA. Hybridization of a cDNA probe to cellular RNA immobilized on paper revealed that no subgenomic RNA related to the VL30 RNA is present in cells expressing the VL30 sequences. VL30 RNA is apparently from a new class of endogenous defective retroviruses and is probably present in most stocks of leukemia and sarcoma viruses made in mouse cells, considerations that must be taken into account when carrying out hybridization experiments with MuLV's. (35 refs)

- 79-2724 Association of Productive Murine Leukemia Virus (MuLV) Infection with Enhanced H-2 Antigen Expression on Murine Lymphoblastoid Cells (Meeting Abstract).** (Eng) Henley, S. L. (Univ. Alabama Birmingham, Birmingham, AL 35294); Acton, R. T.; Wise, K. S. *Fed Proc* 38(3, part 2): 927; 1979 (no refs)

- 79-2725 Antiserum to Murine Leukemia Virus Recognizes Novel Cell Surface Molecules Associated with Growth Control and Transformation.** (Eng) Rieber, M. (Center Microbiology and Cell Biology, Instituto Venezolano de Investigaciones Cientificas, Apartado 1827, Caracas, Venezuela); Rieber, M.; Alonso, M. *Int J Cancer* 23(4): 547-554; 1979.

Data on the nature and surface localization of antigenically related molecular species and on their relationship to growth control are presented. Antiserum directed against murine leukemia virus (MuLV) also reacts with several external proteins present in rat cells transformed by a temperature-sensitive Rous sarcoma virus (RSV). Reaction of iodinated cell extracts with anti-MuLV serum revealed the presence of a 200,000-dalton iodinated component detectable also by metabolic labeling with glucosamine only in serum-starved cultures restricted in the expression of transformation. A similar assay with iodinated cells that

express the transformed phenotype revealed the preferential recognition of two components with an approx mol wt of 100,000 daltons as well as an additional 65,000-dalton external component. Growth of the transformed non-producer NT₃-KR cells (cloned derivative from the normal rat kidney line transformed by a temperature-sensitive derivative of the B77 strain of RSV) in the presence of inducers of C-type viruses led to increased synthesis of a 100,000-dalton glycoprotein (gp100) recognized by the anti-MuLV serum, which is also recognized by the antiserum in NRK-MSV-MuLV-transformed producer cells, in addition to a viruslike glycoprotein of 71,000 daltons (gp71). Absorption of the anti-MuLV serum with monolayers of NT₃-KR cells eliminated the ability of the serum to recognize the gp100, but not the gp71, from NRK-MSV-MuLV-transformed producer cells. The mediation of posttranslational changes in growth control is suggested by the transformation-dependent alteration in the mol wt of the nonviral surface proteins recognized by anti-MuLV serum in the rat cells. (17 refs)

79-2726 Immunoglobulin Production by Lymphosarcomas Induced by Abelson Virus in Mice.

(Eng) Potter, M. (Lab. Cell Biology, Public Health Service, U.S. Dept. Health, Education and Welfare, NCI, NIH, Bethesda, MD 20014); Premkumar-Reddy, E.; Wivel, N. A. *Natl Cancer Inst Monogr* (48): 311-320; 1978.

Electron microscope studies of undifferentiated lymphosarcomas (LS) and plasmacytic lymphosarcomas (PL) induced in BALB/c mice by Abelson murine leukemia virus showed that both tumor types may be mixed and contain undifferentiated cells or cells with a moderate amount of rough endoplasmic reticulum and polysomes. However, PL tumors are composed predominantly of the latter. In biosynthetic studies, PL tumors produced more immunoglobulin (Ig) than LS and more of the heavy chain Ig that is thought to be the murine counterpart of IgD. PL cells sensitized with rabbit antisera to mouse kappa chains formed rosettes with formalinized protein-A producing *Staphylococcus aureus* Cowan I strain. The rabbit antisera were specific for kappa chains by absorption. The failure of lymphosarcoma cells to secrete Ig indicates that their differentiation is blocked by the transformation process. Therefore, lymphosarcoma cells appear to be derived from B lymphocytes. (no refs)

79-2727 Cell-Surface Antigens Associated with Recombinant Mink Cell Focus-inducing Murine Leukemia Viruses. (Eng) Cloyd, M. W. (Lab. Viral Diseases, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Hartley, J. W.; Rowe, W. P. *J Exp Med* 149(3): 702-712; 1979.

The purpose of this study was to characterize reagent antisera made against AKR mink cell focus-inducing (MCF) murine leukemia viruses (MuLV's) and to characterize the cell-surface antigens induced by naturally occurring mouse MCF viruses. Distinct type-specific antigens were detected on cells infected with cloned MCF MuLV's by cell-surface immunofluorescence absorption assays with rabbit antisera raised against naturally occurring AKR MCF viruses. The MCF type-specific antibodies were present in high titer and not absorbable by cells infected with ecotropic, xenotropic, or wild mouse amphotropic MuLV's or with combinations of ecotropic and xenotropic viruses. Three MCF subtype-specific reactions were identified. One subspecificity (operationally designated MCFA-1) defined antigenic determinant(s) distributed among MCF viruses in general. Another (MCFA-2) specified determinant(s) induced by all naturally occurring MCF isolates not of Friend or Moloney origin. A third subspecificity (MCFA-3) was induced by some MCF isolates, and not by others; the presence of this antigen did not correlate with the source of any presently known biological property of the viruses. In addition, type-specific antigenic determinants of ecotropic and xenotropic MuLV's were expressed on MCF virus-infected cells. The serological profile of MCF viruses thus supports the contention that they are *env* gene recombinants between ecotropic and xenotropic MuLV's. However, new, distinct MCF-specific determinants are also generated, and these could be useful markers in studying MCF viruses. (18 refs)

79-2728 Properties of Mouse Leukemia Viruses. XVI. Suppression of Spontaneous Fatal Leukemias in AKR Mice by Treatment with Broadly Reacting Antibody Against the Viral Glycoprotein gp 71. (Eng) Schwarz, H. (Max-Planck-Institut für Virusforschung, Tübingen, W. Germany); Fischinger, P. J.; Ihle, J. N.; Thiel, H. J.; Weiland, F.; Bolognesi, D. P.; Schafer, W. *Virology* 93(1): 159-174; 1979.

AKR mice were treated early in life with antibodies prepared in a goat against the major glycoprotein (gp71) of Friend murine leukemia virus (FLV) and evaluated for up to 2 yr for various parameters associated with AKR thymoma/lymphoma. When both mothers and neonates were given the antibody, AKR disease was suppressed to the degree that the 50% incidence of leukemia was delayed by about 1 yr. The treatment was less effective if initiated at age 3 days, and administration of the antibody to mothers alone or to mice at age 39 days was not successful. Thus, the critical period for treatment was between birth and the first few days of life. Postmortem examination of animals that were treated during this early period revealed a unique leukemic pattern in that the frequency of B-type leukemia (lymphoma only) nearly equaled that of A-type leukemia (thymoma + lymphoma). In addition, several mice died from nonmalignant disease and one died from a lung adenoma. Overall, successful antibody treatment resulted in suppression of virus and development of signifi-

cant levels of antiviral antibodies. Control mice exhibited the opposite pattern: high levels of virus and no detectable antibody. Moreover, the antibody suppressed the expression of mink cell focus-inducing type recombinant virus in tumors of treated mice. It is hypothesized that the heterologous antibody disturbed a key event that occurs during the early life of the AKR mouse and that is of critical importance for the development of leukemia from age 6 mo onward. (35 refs)

- 79-2729 Correlation of the Induction of Transcription of the AKR Mouse Genome by 5-Iododeoxyuridine with the Activation of an Endogenous Murine Leukemia Virus.** (Eng) Chattopadhyay, S. K. (Building 10, Room 2B-50, NIH, Bethesda, MD 20014); Jay, G.; Lander, M. R.; Levine, A. S. *Cancer Res* 39(5): 1539-1546; 1979.

The possibility that incorporated 5-iododeoxyuridine (IdUrd) qualitatively alters the transcription of cell DNA was studied with the use of the highly activatable mouse embryo cell line AKR-2B. Nucleic acid hybridization between radioactive mouse-unique DNA and RNA from AKR-2B cells indicated that the normal extent of transcription in AKR-2B cells is considerably lower than that observed in other mouse cell lines studied (SC-1, NIH 3T3, BALB/c 3T3, and NIH Swiss). Treatment of AKR-2B cells with IdUrd increased the extent of transcription of unique DNA by 60%, which corresponds to an induction of approx 2.5×10^4 gene equivalents. Included among this new set of RNA's were sequences transcribed from the DNA genome of the endogenous AKR-type murine leukemia virus present in AKR-2B cells. IdUrd treatment also markedly increased the synthesis and/or accumulation of those RNA transcripts that are normally expressed in untreated cells. These results suggest that IdUrd stimulates the overall transcription of AKR-2B cells. IdUrd-induced activation of endogenous murine leukemia virus may be a consequence of this stimulation. (39 refs)

- 79-2730 Interaction of Leukemia Viruses with Cells of the Immune Response System.** (Eng) Friedman, H. (Dept. Microbiology, Univ. South Florida Coll. Medicine, Tampa, FL 66312); Specter, S. *Transplant Proc* 11(1): 1060-1065; 1979.

The effects of the Friend leukemia virus (FLV) complex on in vivo and in vitro immune responses were studied in BALB/c and DBA mice. Spleen cells from infected animals were injected into heavily irradiated recipient mice, which were then challenged with sheep RBC and examined for their antibody response. Donor mice, infected for 7-10 days or longer, showed an 80%-90% or greater suppression of antibody responsiveness to sheep RBC. The B cell precursors in the bone marrow were more impaired than

the T lymphocytes. Spleen cells from FLV-infected animals also exhibited depressed immune responses in vitro. In addition, infected splenocytes added in relatively small numbers to normal spleen cells in vitro resulted in immunosuppression. Separation of the immunosuppressive infected spleen cells from normal cells by cell-impermeable membranes (0.45 μ m pore size) still resulted in immunosuppression, indicating that a virus or virus-associated agent crossed the membrane and depressed immunity. Anti-FLV serum neutralized this suppression. Cell-free extracts, but not unclarified extracts, from infected spleens also inhibited antibody formation when added to normal spleen cells in culture. Electron microscopy and fluorescent antibody studies indicated that the number of splenocytes with the normal surface topography of lymphoid cells was decreased in the spleens of infected animals. These changes were correlated with decreased numbers of splenocytes evincing surface immunoglobulin and theta antigen and increased numbers positive for FLV-associated surface antigen. (14 refs)

- 79-2731 Alterations in the Response to Erythropoietin and RNA-dependent DNA Polymerase Activity in Mouse Spleen Cells Infected with Friend Leukemia Virus.** (Eng) Horikoshi, A. (First Dept. Internal Medicine, Nihon Univ. Sch. Medicine, Oyaguchi, Itabashi, Tokyo 173, Japan); Sasaki, R.; Mizoguchi, H.; Miura, Y.; Takaku, F.; Amaki, I. *Cancer Res* 39(5): 1841-1846; 1979.

Changes in heme synthesis, in responsiveness to erythropoietin in vitro, and in RNA-dependent DNA polymerase activity (RDDP) of mouse spleen cells were determined after the ip inoculation of C3H/He mice with about 2×10^4 spleen focus-forming units of Friend leukemia virus (FLV). By day 6 following FLV injection, hyperbasophilic "Friend cells" resembling proerythroblasts became dominant in the spleen. The heme synthesis rate in the infected spleen cells started to increase 14 days after infection, and it reached a plateau at a level about five times higher than that of the uninfected control spleen cells after about 22 days. Although a normal response to erythropoietin was observed 4 hr after infection, no response could be detected at 24 hr and thereafter. RDDP activity in the spleen cells began to increase on day 4, attaining a peak on days 8 and 10 and decreasing markedly thereafter. Enzyme activity again increased on day 28. These results suggest that FLV affected the erythropoietin-responsive cells at a very early stage of infection, before morphological changes in the spleen cells or increases in RDDP activity could be observed. (45 refs)

- 79-2732 Erythroid Leukemia Induced by Friend Lymphatic Leukemia Virus in T-Cell-depleted Mice.** (Eng) Dawson, P. J. (Dept. Pathology, Univ. Chicago, 950 E. 59th St., Chicago, IL 60637); Dresler, S. L.; Fieldsteel, A. H. *Cancer Res* 39(5): 1611-1615; 1979.

The possibility that the production of different leukemias in intact and thymectomized mice is due to the selective expression of different components from a mixture of leukemia viruses was investigated. BALB/c mice depleted of T cells by thymectomy at 3-5 days of age and by treatment with antithymocyte serum were inoculated with Friend virus-associated lymphatic leukemia virus (LLV-F). After a long latent period, these animals developed erythroid leukemia. In contrast, intact control mice inoculated with LLV-F developed typical T-cell lymphomas. Cell-free virus prepared from leukemic T-cell-depleted animals induced lymphoid, myeloid, and erythroid leukemias in intact mice. The erythroid leukemia-inducing virus differed from the spleen focus-forming component of Friend virus in its long latent period (88-225 days) and in its inability to induce spleen foci. End-point dilution experiments suggested that a hitherto undescribed component of the Friend virus complex might be responsible for these late-appearing erythroid leukemias. (14 refs)

- 79-2733** Enhanced Expression of Viral Polypeptides and Messenger RNA in Dimethyl Sulfoxide and Bromodeoxyuridine-treated Friend Erythroleukemic Cells. (Eng) Colletta, G. (Chair Viral Oncology, Inst. General Pathology, 2nd Faculty Medicine and Surgery, Univ. Naples, I-80131 Naples, Italy); Fragomele, F.; Sandomenico, M. L.; Vecchio, G. *Exp Cell Res* 119(2): 253-264; 1979.

The intracellular virus-specific macromolecular changes induced by dimethyl sulfoxide (DMSO) and/or bromodeoxyuridine (BUdR) were analyzed in the Friend erythroleukemic cell (FLC) clone 745 A 19. These cells, which were transformed in vivo by Friend virus, are arrested in an undifferentiated state but can be induced to differentiate in vitro by DMSO. Treatment of FLC's with 2% DMSO for 4 days enhanced extracellular virus production and, concomitantly, increased by about four-fold intracellular virus-specific polyribosomal RNA. The simultaneous addition to FLC's of DMSO and BUdR (the latter inhibits DMSO-induced differentiation) brought about an even greater increase in both viral polypeptides and viral messenger RNA (mRNA). As a consequence of treatment with both drugs, new viral nucleotide sequences were expressed, as revealed by nucleic acid hybridization studies. Moreover, it appears that two p30-like virus-specific intracellular polypeptides, instead of one single p30, were expressed in FLC's treated with DMSO and BUdR. These data also support the idea that gene sequences different from those present in the original Friend virus are expressed as a consequence of the combined treatment. It is concluded that the main site of action of both drugs in this cell system is at the level of transcription of viral RNA, and it is suggested that the drugs may act upon cell differentiation by modulating the expression of integrated viral genomes differently. (38 refs)

- 79-2734** Enhancement of Erythroid Target Cells for Friend Murine Leukemia Virus by Intravenous Pyran Treatment. (Eng) Schuller, G. B. (Dept. Surgery, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA 23298); Morahan, P. S. *J Natl Cancer Inst* 62(5): 1257-1260; 1979.

The enhancement of Friend murine leukemia virus (F-MuLV) leukemogenesis by pyran, a synthetic polyanionic immunomodulator, was investigated. Male BALB/c mice that received prophylactic treatment with pyran (25 mg/kg iv) had significantly enhanced splenomegaly, an increased number of splenic foci induced by the spleen focus-forming virus (SFFV) in the F-MuLV complex, and a slightly decreased mean survival time compared with untreated controls infected with F-MuLV. A corresponding increase in the lymphatic leukemia virus component of the F-MuLV complex was not observed, which suggests that the enhancement of the disease was due primarily to a selective increase in the SFFV component of the F-MuLV complex. That the enhancement was related to an increased number of target cells for SFFV was substantiated by data concerning erythropoiesis in iv pyran-treated animals. Increases in splenic hematocrits and in uptake of ^{59}Fe in the spleens of animals treated with pyran provided quantitative evidence for the histologic finding of increased erythroid precursors in the spleens. (22 refs)

- 79-2735** Expression of *Fv-4r* Allele in Hematopoietic Cells from G Mice Resistant to Friend Leukemia Virus. (Eng) Ikeda, H. (Dept. Genetics, Inst. Medical Science, Univ. Tokyo, P.O. Takanawa, Tokyo 108, Japan); Odaka, T. *Int J Cancer* 23(4): 514-518; 1979.

Susceptibility to Friend leukemia virus (FLV) infection was studied in radiation chimeras between G mice carrying the *Fv-4*-resistant allele and *Fv-4*-susceptible DDD mice. The helper lymphatic leukemia virus (LLV) of FFV did not replicate in FFV-infected G mice, and virus growth was not enhanced by treatment with cyclophosphamide or cortisone acetate. Reciprocal bone marrow or spleen cell transplantation between G and DDD or DDD-*Fv-r* mice converted the susceptibility of the recipients to that expected from the genotypes of the donors. However, G mice reconstituted with DDD or DDD-*Fv-r* cells were not as susceptible as DDD mice reconstituted with DDD or DDD-*Fv-r* cells. Reconstituted G mice showed lower LLV and spleen focus-forming virus titers than did similarly reconstituted DDD mice, and they developed splenomegaly after a longer latent period. It is concluded that helper LLV grows mainly in the radiosensitive, bone marrow-derived cells and that the *Fv-4* gene is expressed in these cells. (18 refs)

- 79-2736** In Vitro Transformation of Mouse Bone Marrow Cells by the Polycythemic Strain of Friend

Leukemia Virus. (Eng) Revoltella, R. (Dept. Tumor Immunobiology, Lab. Cell Biology, Consiglio Nazionale delle Ricerche Rome, Rome, Italy); Bertolini, L.; Friend, C. *Proc Natl Acad Sci USA* 76(3): 1464-1468; 1979.

Strains of Friend leukemia virus (FLV) that are associated with polycythemia contain the defective spleen focus-forming virus (SFFV). The ability of these FLV strains to transform DBA/2J mouse bone marrow cells in vitro was studied in an effort to clarify the role that SFFV may play in the pathogenesis of leukemia in vivo. Criteria for transformation were the establishment of permanent lines, growth on semisolid agarose, and the production of tumors at the inoculation site in syngeneic hosts. Two lines of immature hematopoietic cells that grew in suspension originated from the infected cultures. Each had an almost diploid karyotype (38-39 chromosomes) and 3-4 metacentric chromosomes. These transformed cells expressed the gp71 viral envelope glycoprotein and p30 viral core protein antigens. Virus production was measured by the reverse transcriptase (RNA-dependent DNA polymerase) activity of the virions released into the medium. The virus, assayed in vivo for infectivity, had SFFV activity but was attenuated for leukemogenicity. The stimulation of Hb synthesis in cells grown in medium supplemented with dimethyl sulfoxide or hexamethylene bisacetamide indicated that the cells are erythroid in origin. SFFV may have a function analogous to that of erythropoietin in influencing the process of transformation by FLV. (32 refs)

79-2737 Replication-defective S+L- Moloney MSV Codes for an Envelope Glycoprotein with Moloney MuLV- and MCF-MuLV-specific Determinants. (Eng) Reynolds, F. H. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD 21701); Rapp, U. R.; Todaro, G. J.; Stephenson, J. R. *Virology* 93(2): 582-588; 1979.

The expression of a recombinant envelope glycoprotein containing mink cell focus-forming (MCF)-specific determinants in cells nonproductively transformed by the S + L-strain of Moloney murine sarcoma virus (Mo-MSV) is reported. Cells nonproductively transformed by the S + L-strain of Mo-MSV expressed three *gag* gene components, p15, p12, and p30, in the form of a 55,000-mol wt precursor polypeptide. In addition, these transformed cell lines were characterized by expression of an envelope glycoprotein containing antigenic determinants related to those of both Moloney murine leukemia virus and viruses of the previously described highly leukemogenic MCF group. These cells lacked detectable reactivity in competition immunoassays for either the carboxy-terminal *gag* gene protein, p10, or C-type viral reverse transcriptase. Cells transformed by either of two other Mo-MSV isolates lacked detectable levels of expression of any of the known *gag* or *env* gene-coded proteins. These findings indicate that although recombination within C-type virus structural

genes may be involved in the genesis of mammalian sarcoma viruses, expression of viral structural proteins is not necessary for malignant transformation. (29 refs)

79-2738 Functional Activity of Murine Lymphocytes During Viral Carcinogenesis. (Rus) Umanskii, Iu. A. (Dept. Immunology Carcinogenesis, Inst. Problems Oncology, Kiev, USSR); Evsev'eva, A. I.; Semenova-Kobzar', R. A.; Kushko, L. Ia. *Tsitologiya* 21(2): 191-195; 1979.

The functional activity of lymphocytes from BALB/c mice with a Moloney virus-induced sarcoma was evaluated. Animals were inoculated im with a 10% extract of tumor tissue and, within different time periods after inoculation, the antitumor activity of the lymphocytes and their ability to undergo phytohemagglutinin (PHA)-induced blast transformation (BT) was determined. On days 7-8 after virus inoculation (ie, during tumor progression), the BT activity of the lymphocytes was nine times lower than that of lymphocytes from control mice. On days 13-15 after virus administration (ie, during tumor regression) the lymphocytes showed a 4.2-fold increase in BT activity. (21 refs)

79-2739 Immune Responses to Weakly Immunogenic Virally Induced Tumors. III. Genetically Unrestricted Cytolysis of Allogeneic Tumor Target Cells. (Eng) Devens, B. (Lautenberg Center General and Tumor Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Naor, D. *J Immunol* 122(4): 1397-1401; 1979.

The genetically nonrestricted cytotoxicity of allogeneic tumor target cells was investigated using in vitro- or in vivo-passaged YAC, a Moloney virus-induced lymphoma of A mice (H-2a), and RBL5, a Rauscher virus-induced lymphoma of C57BL/6 mice (H-2b). These tumors cross-react serologically. Splenocytes from A mice injected with in vitro-passaged YAC-1 or with RBL5 could generate, after in vitro culture with or without stimulation, a genetically nonrestricted cytotoxic response against RBL5. The effector cells that were generated after the in vitro cultivation recognized tumor-associated antigens (TAA) on the target cells. H-2 alloantigens were not recognized by the effector cells. The effector cells that killed RBL5 tumor cells in a genetically nonrestricted manner were identified as T cells. In vivo-passaged YAC did not induce anti-RBL5 reactive cells in A mice. Instead, it induced suppressor cells that could abrogate the anti-RBL5 cytotoxic response of RBL5-primed splenocytes but not that of YAC-1 primed splenocytes. This suggests that the reactive cells generated after RBL5 injection are different from the reactive cells generated after YAC-1 injection. (18 refs)

VIRAL CARCINOGENESIS

- 79-2740 Amino Acid Sequence Homology Between Histone H5 and Murine Leukemia Virus Phosphoprotein p12.** (Eng) Henderson, L. E. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD 21701); Gilden, R. V.; Oroszlan, S. *Science* 203(4387): 1346-1348; 1979.

Amino acid sequences of the conserved regions near the amino termini of several murine leukemia virus (MuLV) p12 phosphoproteins were compared with those of the H5 histones. H5 histones are phosphorylated nuclear proteins found only in nucleated RBC. Comparison of residues 4-15 in goose H5 with residues 1-12 in Moloney MuLV showed 6 positional identities out of 12 residues without the introduction of a gap in either sequence. The number of identities increased with the introduction of gap at position 13 in the alignment of the H5 sequences. Functionally homologous amino acid residues also appeared in positions 2, 4, 8, 9, 12, 14 and 15. Apparent amino acid sequence similarities between human immunoglobulins and the p12 proteins appeared to be the result of chance and the method of comparison. Conversely, the H5 histones and viral p12 proteins appeared to be under selective pressure to conserve the highly related sequences in a coiled structure near the amino acid terminal regions. It is unlikely that the two groups of proteins evolved from a single common ancestral gene, but evolution of retrovirus structural proteins by a process of differentiation from preexisting cellular genes is likely. The amino terminal sequence homology between viral p12 proteins and the H5 histones suggests that the amino terminal regions of the p12's may also be involved in nucleic acid binding and/or phosphorylation. (25 refs)

- 79-2741 Isolation and Characterization of a Mouse Cell Line Containing a Defective Moloney Murine Leukemia Virus Genome.** (Eng) Besmer, P. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Fan, H.; Paskind, M.; Baltimore, D. *J Virol* 29(3): 1023-1034; 1979.

Mouse JLS-VII cells containing a 1,000-nucleotide deletion mutant of Moloney murine leukemia virus (M-MLV) were cultured and characterized. The deletion did not affect the size or function of the 21S messenger RNA (mRNA) that encodes the *env* gene products. Both the deleted RNA and the 21S mRNA were recovered in polyribosomes. Cells containing the deleted virus made no detectable Pr180gag-pol. Pr65gag synthesis was also absent, but a 45,000-mol-wt gag gene product was found that might be encoded by the deleted genomes. Biosynthesis of Pr80env proceeded normally in these cells; the intracellular precursor was cleaved and migrated to the cell surface as gp70. The cells could not be superinfected by homologous M-MLV, presumably because of surface restriction due to the gp70. Although the cells expressed M-MLV gp70 on their surface, they did not make pseudotypes after infection with

vesicular stomatitis virus, which suggests that PR65 gag may play a critical role in pseudotype formation. Induction of endogenous virus expression in the cells carrying the deletion mutation generated an N-tropic MLV that could fuse XC cells. This may represent a recombinant between the deletion mutant and an endogenous virus. (59 refs)

- 79-2742 Maturation of Moloney Murine Leukemia Virus.** (Eng) Lu, A. H. (Dept. Microbiology, Sch. Basic Medical Science, Univ. Illinois, Urbana, IL 61801); Soong, M. M.; Wong, P. K. *Virology* 93(1): 269-274; 1979.

Virion changes during the membrane postbudding process were studied using freshly released virions of a temperature-sensitive mutant (ts3) of Moloney murine leukemia virus. Cultured TB cells, a CFW/D mouse thymus-bone marrow line, were infected with ts3 and grown at 39 C (nonpermissive temperature) until semiconfluent; the virions were then collected from the culture supernatant beginning 30 min after the cultures were shifted to 34 C. Virions released at 34 C showed a twofold increase in infectivity and reverse transcriptase activity for the first 90-120 min after release. Upon further incubation, both infectivity and reverse transcriptase activity decreased. Furthermore, processing of the gag precursor was also observed. These postrelease changes were found to be correlated with the morphologic transformation of immature to mature virions. (15 refs)

- 79-2743 Cell-free Synthesis of Rauscher Murine Leukemia Virus "gag" and "env" Gene Products from Separate Cellular mRNA Species.** (Eng) Murphy, E. C. (Dept. Biology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Campos, D.; Arlinghaus, R. B. *Virology* 93(2): 293-302; 1979.

RNA from cells infected with Rauscher murine leukemia virus (R-MuLV) was translated in a messenger RNA (mRNA)-dependent cell-free protein synthesizing system. A cellular RNA species of about 35S in size coded for polypeptides of approx 65,000 mol wt (Pr65gag) and 200,000 mol wt (Pr200gag) that were immunoprecipitable with antisera directed against the R-MuLV gag proteins p30, p15, p12, and p10. The methionine-containing tryptic peptides of the 65,000-mol wt polypeptide translated from cellular 35S RNA were identical to those of authentic Pr65gag. Translation of RNA in the 25S-35S size class suggests that although Pr65gag can be translated by RNA throughout this size range, Pr200gag-pol translation is restricted to mRNA that sediments at 35S. Antiserum directed against the R-MuLV envelope protein gp69/71 recognized a polypeptide of 68,000 mol wt, designated Pr68env, that was coded for by RNA that sedimented at

about 22S in sucrose gradients and had a minimum size of about 1.25×10^6 daltons, as estimated by agarose gel electrophoresis. Tryptic maps of Pr68env showed that it contained all of the methionine-labeled tryptic peptides and most of the tyrosine-containing tryptic peptides characteristic of gPr90env, the authentic R-MuLV glycosylated envelope precursor. (25 refs)

- 79-2744 Identification of a Mouse Gene Required for Binding of Rauscher MuLV Envelope gp70.** (Eng) Hilkens, J. (Div. Genetics, Netherlands Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam, Netherlands); Colombatti, A.; Strand, M.; Nichols, E.; Ruddie, F. H.; Hilgers, J. *Somatic Cell Genet* 5(1): 39-49; 1979.

The genetics of the receptor for ecotropic murine leukemia virus (MuLV) infection was studied in somatic cell hybrids. Mouse chromosome-segregating somatic cell hybrids were established between a mouse thymic leukemia cell line (GRSL) and a Chinese hamster lung fibroblast line (E36). The GRSL cells specifically bound purified Rauscher MuLV envelope glycoprotein gp70, but the E36 cells exhibited no binding. The hybrids selectively bound Rauscher gp70, depending on the presence of a mouse cellular gene for the ecotropic MuLV gp70 receptor. A syntenic relationship was observed between the dipeptidase-3 chromosome marker (on chromosome 5) and the gp70 receptor in primary clones and subclones of these hybrids; this relationship was confirmed by chromosome analysis. The involvement of the major histocompatibility (H-2) antigens in the binding of Rauscher MuLV gp70 could be ruled out, because discordancies of the receptor presence/H-2 absence as well as of the receptor absence/H-2 presence type were observed. These results indicate that the *Rec-1* (replication ecotropic MuLV) gene may be the receptor gene for ecotropic MuLV. (31 refs)

- 79-2745 Tunicamycin Inhibits Glycosylation of Precursor Polyprotein Encoded by env Gene of Rauscher Murine Leukemia Virus.** (Eng) Schultz, A. M. (Frederick Cancer Res. Center, Frederick, MD 21701); Oroszlan, S. *Biochem Biophys Res Commun* 86(4): 1206-1213; 1979.

The effect of tunicamycin on the synthesis and processing of the viral envelope precursor polyprotein (gPr85env) of Rauscher murine leukemia virus (R-MuLV) was investigated in BALB/c mouse bone marrow cells. Tunicamycin specifically inhibits the synthesis of oligosaccharides that attach to glycoproteins via asparagine residues. The mol wt of this precursor polyprotein was reduced from 85,000 to 68,000 daltons when it was synthesized in the presence of tunicamycin. The unglycosylated precursor protein (Pr68env) was synthesized at a rate comparable to that of the normal carbohydrate-

containing envelope precursor (gPr85env). Pr68env was not proteolytically processed, and it remained undegraded in the cell. Thus, most, if not all, of the carbohydrate content of gPr85env is N-linked, and glycosylation appears to be necessary for normal processing of precursor proteins into viral particles. (28 refs)

- 79-2746 Properties of "Mink Cell Focus-inducing" (MCF) Virus Isolated from Spontaneous Lymphoma Lines of BALB/c Mice Carrying Moloney Leukemia Virus as an Endogenous Virus.** (Eng) Vogt, M. (Tumor Virology Lab., Salk Inst., P. O. Box 1809, San Diego, CA 92112) *Virology* 93(1): 226-236; 1979.

Mink cell focus-inducing (MCF) viruses were isolated from lymphoid cell lines derived from five spontaneous thymomas of BALB/c (Mo) mice. The Mo-MCF isolates grew both in murine and nonmurine cells, were interfered with by both ecotropic and xenotropic murine leukemia viruses (MuLV), and were NB-tropic. They also induced cytopathic changes in mouse cells. In cells producing both Mo-MCF and Mo-MuLV, Mo-MCF genomes were predominantly released as pseudotypes with Mo-MuLV envelopes. Mo-MCF was weakly lymphomagenic in NIH Swiss mice, inducing thymomas in only 3/35 mice inoculated sc with 650-4,500 focus-forming units of virus. However, each cell in a BALB/c (Mo) thymoma line and each cell in a Mo-MCF-induced thymoma produced MCF virus, which suggests that MCF viruses are required for lymphomagenesis. Mo- and AKR-MuLV might play a double role in the development of spontaneous lymphomas: (1) as parental viruses by generating MCF recombinants and (2) as helper viruses by forming pseudotypes of MCF viruses. (28 refs)

- 79-2747 Effect of Interferon on Human Cells Releasing Oncornaviruses: An Assay for Human Interferon.** (Eng) Salzberg, S. (Dept. Life Science, Bar-Ilan Univ., Ramat Gan, Israel); Heller, A.; Aboud, M.; Gurari-Rotman, D.; Revel, M. *Virology* 93(1): 209-214; 1979.

Human RD-114 cells chronically infected with a feline RNA tumor virus were treated with 20 or 60 units/ml of human interferon (IF) to determine the kinetics of development of the antiviral state in these cells. The amount of virus released was monitored by reverse transcriptase (RT) activity in the culture medium. RT activity was inhibited by 70%-80% 4-6 hr after the cells were treated with 60 units/ml IF. When RD-114 cells were treated with 20 or 60 units/ml IF for 18 hr, the antiviral state remained unaltered at 20 hr after IF removal; with the higher concentration, >70% inhibition in virus release was observed 24 hr postremoval. Human IF and mouse IF showed species specificity when tested for their effects on virus release from RD-114 and mouse NIH/3T3 (murine leukemia virus)

cells. Human IF was titrated on RD-114 cells by monitoring the RT activity in the culture medium or by determining the reduction in the number of plaques formed on cell monolayers after infection with vesicular stomatitis virus. Both assays were identical in their sensitivity. The use of the RT assay for detecting human fibroblast IF during its purification by ion-exchange chromatography is illustrated. (18 refs)

79-2748 Feline Oncornavirus-associated Cell-Membrane Antigen (FOCMA) (Meeting Abstract). (Eng) Neil, J. C. (Dept. Veterinary Pathology, Univ. Glasgow Veterinary Sch., Bearsden Road, Bearsden, Glasgow, Scotland) *Br J Cancer* 39(4): 471-472; 1979 (no refs)

79-2749 Translation of Bovine Leukemia Virus Virion RNAs in Heterologous Protein-synthesizing Systems. (Eng) Ghysdael, J. (Departement de Biologie Moleculaire, Universite Libre de Bruxelles, 1640 Rhode St. Genese, Brussels, Belgium); Kettmann, R.; Burny, A. *J Virol* 29(3): 1087-1098; 1979.

Studies were conducted to define the bovine leukemia virus (BLV) genome more precisely and to identify the genetic information encoded in it. BLV 60S-70S RNA was heat-denatured, the polyadenylic acid-containing species were separated by velocity sedimentation, and several size classes were translated in a micrococcal nuclease-treated cell-free system from rabbit reticulocytes. The major RNA species sedimented at 38S and migrated as a single component of mol wt 2.95×10^6 when analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. The predominant polypeptides of the in vitro translation of BLV virus 38S RNA were products with mol wts of 70,000 and 45,000; minor components with mol wts of 145,000 and 18,000 were also observed. Two lines of evidence indicate that the 70,000- and 45,000-mol wt polypeptides represent translation products of the *gag* gene of the BLV genome (Pr70*gag* and Pr45*gag*). First, they are specifically precipitated by a monospecific antiserum to the major internal protein, p24; second, they are synthesized and correctly processed into virion proteins p24, p15, and p10 in *Xenopus laevis* oocytes microinjected with BLV virus 38S RNA. The 145,000-mol wt polypeptide was immunoprecipitated by the anti-p24 serum and not by an antiserum to the major envelope glycoprotein, gp60. It contained all the tryptic peptides of Pr70*gag* and additional peptides unique to it and, thus, represents an elongation product of Pr70*gag* in an adjacent gene, presumably the *pol* gene. The 18,000-mol wt product was antigenically unrelated to p24 and gp60 and shared no peptides in common with Pr70*gag*, Pr45*gag*, or the 145,000-mol wt polypeptide. It was maximally synthesized on a polyadenylic acid-containing virion 16S-18S RNA, and

evidence that this RNA is a 3' end-derived subgenomic fragment of the BLV genome rather than a contaminating cellular RNA is presented. (36 refs)

79-2750 Carnivores Have Sequences in Their Cellular DNA Distantly Related to the Primate Endogenous Virus, MAC-1. (Eng) Bonner, T. I. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014); Todaro, G. J. *Virology* 94(1): 224-227; 1979.

The hybridization of the complementary DNA (cDNA) of a new endogenous primate virus (MAC-1) isolated from a *Macaca arctoides* cell line to the DNA's of several feline species and other carnivores was assayed using low-stringency-hydroxyapatite conditions. At 60 C, the amount of hybrid was 2.1%-4.3% for two primate species (human and howler monkey) and the dog, compared with 10.0%-15.7% for the feloid carnivores. In addition, the melting temperature for the hybrids that formed with the feloid carnivore DNA's ranged from 58 to 61 C, compared with 56 C for those formed with the primate DNA's. The kinetics of hybridization of MAC-1 cDNA with two representative carnivores indicated that there would be at least 50 copies of viral gene sequences per haploid genome. In summary, cDNA transcripts from the endogenous macaque virus detect related sequences in carnivores that, from the S_1 nuclease values, must be distinct from the two known cat C-type viruses, RD-114 and feline leukemia virus. These sequences appear to be present in a number of carnivores, indicating that they have been present for several million years. Similarly, MAC-1-related sequences have been previously concluded to be present in primates for at least 40 million years. (13 refs)

79-2751 Primate Retroviruses: Intracistronic Mapping of Type D Viral *gag* Gene by Use of Nonconditional Replication Mutants. (Eng) Devare, S. G. (Viral Genetics Section, Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014); Stephenson, J. R. *J Virol* 29(3): 1035-1043; 1979.

Findings that represent the first determination of the *gag* gene order of an endogenous retrovirus of primate origin and provide information regarding the evolutionary relatedness of C- and D-type mammalian retroviruses are presented. Nonconditional replication mutants of squirrel monkey retrovirus (SMRV), an endogenous D-type virus of primates, are defective in posttranslational processing of nonglycosylated virus-coded structural proteins. Utilizing these mutants, in combination with sensitive radioimmunological assays, the existence of a 72,000-dalton precursor polypeptide (Pr72*gag*) encoded by a region of the SMRV genome designated *gag* was demonstrated. Posttranslational cleavage of this precursor polypeptide gives rise to virion structural proteins with mol wts of 35,000

(p35), 16,000 (p16), 12,000 (p12), and 9,000 (p9) daltons. p35, p16, and p9 are phosphorylated. Analysis of viral antigen expression in cell lines nonproductively infected with either of two replication-defective SMRV mutants or mink cells productively infected with wild-type SMRV resulted in the detection of several SMRV Pr72gag intermediate cleavage products. Adjacent proteins within such intermediates were identified by specific competition immunoassays, and the intracistronic order of individual structural proteins with SMRV Pr72gag was tentatively deduced as NH₂-p16-p12-p35-p9-COOH. (31 refs)

79-2752 Herpesvirus saimiri: Studies In Vivo and In Vitro with Attenuated and Oncogenic Strains (Meeting Abstract). (Eng) Wright, J. J. (Univ. Illinois, Medical Center, Urbana, IL 61801) *Diss Abstr Int [B]* 39(10): 4748; 1979 (no refs)

79-2753 Structure and Origin of Defective Genomes Contained in Serially Passaged Herpes Simplex Virus Type 1 (Justin). (Eng) Locker, H. (Dept. Biology, Univ. Chicago, Chicago, IL 60637); Frenkel, N. *J Virol* 29(3): 1065-1077; 1979.

Studies concerning the evolution and structural organization of the defective herpes simplex virus type 1 (HSV-1, Justin) genomes are reported, along with the precise localization of the small (S)-component sequences from which these defective genomes arise. Restriction enzyme and hybridization analyses revealed that high-density DNA prepared from passage 15 of serially passaged HSV-1 contains three major classes of modified viral DNA molecules, each composed of distinct but closely related types of repeat units. The DNA sequences within the three types of repeat units are colinear with the DNA sequences located at the right end (between coordinates 0.94 and 1.0) of the parental HSV-1 genome. Thus, the three types of repeat units each contain the entire repeat sequence (*ac*) which brackets the unique sequences of the S component of HSV-1 DNA and differ only with respect to the amount of unique S sequences that they contain. The three classes of high-density DNA molecules were stably propagated between passages 6 and 15 of this series. (31 refs)

79-2754 Kinetics of Nuclear Changes During Herpetic Infection of Human Primary Liver Cell Cultures. (Eng) Scotto, J. M. (Unite de Recherches d'Hepatologie Infantile, U 56 INSERM, Hopital d'Enfants, F-94270 Le Kremlin-Bicetre, Paris Cedex 06, France); Sauron, B.; Dupuy-Coin, A. M.; Gautier, M. *J Submicrosc Cytol* 11(2): 229-241; 1979.

The kinetics of herpes simplex virus type 2 (HSV-2)-

induced nuclear changes in human primary liver cell cultures infected at high multiplicity (100-200 plaque-forming units/cell) was investigated electron microscopically. During the first 4 hr of infection, the nucleoli were progressively and completely destroyed. Between 5 and 8 hr, the chromatin lost its initial condensed appearance. During the course of infection, a central electron-lucent area appeared in the nuclei that, between 5 and 8 hr, was seen in almost all nuclei. The number of hairy dense bodies and plexiform structures increased during viral replication. These observations indicate that primary cultures of human liver cells behave in a manner similar to that of other cells when infected with HSV-2. (49 refs)

79-2755 Herpes Simplex Virus Types 1 and 2: Comparison of the Defective Genomes and Virus-specific Polypeptides. (Eng) Bookout, J. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Hirsch, I.; Purifoy, D. J.; Biswal, N. *Virology* 93(2): 598-604; 1979.

The defective DNA's of herpes simplex virus (HSV) types 1 and 2 were investigated with regard to their reassociation kinetics and susceptibility to cleavage by *HindIII*, the polypeptides synthesized after infection with serially passaged HSV-1 or HSV-2 were studied. Defective DNA of types 1 and 2 showed similar reassociation kinetics with a complexity of approx 12.43×10^6 daltons. This estimate suggests that the defective genomes have a complexity less than 13.4% of the standard genome. The defective DNA's appeared to be homologous to 12.1 or 8.5% of the standard genome. The percent homology between defective DNA's of types 1 and 2 was estimated at 40%. An overproduction of an early polypeptide, VP175, was observed in cells infected with defective HSV-1, but no overproduction of any polypeptide equivalent to VP175 was seen in cells infected with defective HSV-2. The results suggest that although the defective DNA's of HSV-1 and HSV-2 have some common physiochemical properties, their base sequences and genomic expression in the infected cell are different. (23 refs)

79-2756 Herpes Simplex Virus DNA Isolation from Infected Cells with a Novel Procedure. (Eng) Pignatti, P. F. (Istituto di Genetica, Laboratorio di Genetica Biochimica, Pavia, Italy); Cassal, E.; Meneguzzi, G.; Chenciner, N.; Milanesi, G. *Virology* 93(1): 260-264; 1979.

Herpes simplex virus type 1-infected Hep₂ cells were extracted in the presence of 0.25% Triton X-100-0.2 M NaCl. Viral DNA associated with proteins was found in the supernatant after low-speed centrifugation. Only viral DNA was recovered by this procedure, as shown by CsCl density analysis after deproteinization. Full-length viral DNA

molecules were observed in the electron microscope. (10 refs)

- 79-2757 Herpes Simplex Virus (HSV) in C-1300 Mouse Neuroblastoma Cells (Meeting Abstract). (Eng) Schwartz, J. (Mt. Sinai Sch. Medicine, New York, NY); Elizan, T. S. *J Neuropathol Exp Neurol* 38(3): 339; 1979 (no refs)

- 79-2758 Membrane Proteins Specified by Herpes Simplex Viruses. IV. Conformation of the Virion Glycoprotein Designated VP7(B₂). (Eng) Sarmiento, M. (Dept. Pathology, Univ. Chicago, Chicago, IL 60637); Spear, P. G. *J Virol* 29(3): 1159-1167; 1979.

The physical properties of detergent-solubilized herpes simplex virus type 1 envelope proteins were investigated, and the structure of the virion glycoprotein designated VP7(B₂) was partially characterized. VP7(B₂) was extracted from virions by a nonionic detergent in the form of an oligomer, whereas the other detergent-soluble envelope proteins were extracted as monomers. The subunits of the VP7(B₂) oligomer were resistant to dissociation by 2-mercaptoethanol or by a mixture of sodium dodecyl sulfate (SDS) and 2-mercaptoethanol except at elevated temperature. The oligomeric form of solubilized VP7(B₂) was predominantly dimeric, based on the sedimentation rates in sucrose gradients and the electrophoretic mobilities in SDS-containing acrylamide gels of the undissociated and heat-dissociated forms of VP7(B₂). It is concluded that the oligomeric conformation of solubilized VP7(B₂) is maintained at least in part by noncovalent interactions and that the oligomer is apparently composed of two polypeptides with similar electrophoretic mobility, perhaps of two identical glycopolypeptides. (35 refs)

- 79-2759 Membrane Proteins Specified by Herpes Simplex Viruses. III. Role of Glycoprotein VP7(B₂) in Virion Infectivity. (Eng) Sarmiento, M. (Dept. Pathology, Univ. Chicago, Chicago, IL 60637); Haffey, M.; Spear, P. G. *J Virol* 29(3): 1149-1158; 1979.

A temperature-sensitive (ts) mutant of herpes simplex virus type 1 (HSV-1) that has a selective defect in the accumulation of envelope glycoprotein VP7(B₂) at the nonpermissive temperature was used to investigate the role of VP7(B₂) in virion infectivity. At the nonpermissive temperature, the mutant produced virions that have very low specific infectivity. Upon sodium dodecyl sulfate (SDS)-polyacrylamide gel electrophoresis, a fraction of the VP7(B₂) from the parental wild-type HSV-1 was present as an SDS-stable dimer, whereas this form of VP7(B₂) was not detected in the mutant virions. The infectivity of ts HSV-1 virions pro-

duced at 34 C was inactivated by heat at a much higher rate than that of the parental HSV-1. Kinetics studies indicated that the ratio of the binding efficiencies of ts HSV-1 produced at 34 C and that produced at 39 C (to Vero cells) was 1.3:1 after 30 min of incubation but 2:1 after 2 hr. However, it is unlikely that this difference in adsorption could account for the large differences in specific infectivities. Treatment with polyethylene glycol, which promotes membrane fusion, enhanced the infectivity of mutant virions produced at 39 C by as much as 80-fold but had no effect on virions produced at 34 C or on parental HSV-1 infectivity. These findings indicate that the poor infectivity of the mutant virions is caused by failure to penetrate the host cell rather than by failure to absorb. It is concluded that VP7(B₂) is required for the penetration stage of the infective process following adsorption. (31 refs)

- 79-2760 Transcription of Herpes Simplex Virus Type 1 DNA by Eukaryotic and Prokaryotic RNA Polymerases: Size and Sequence Analysis of RNA. (Eng) Chenciner, N. (Centre de Biochimie, Faculte des Sciences, 06034 Nice, France); Meneguzzi, G.; Cassai, E.; Milanese, G. *Virology* 94(1): 232-236; 1979.

The transcription of herpes simplex virus type 1 (HSV-1) DNA by purified RNA polymerase was analyzed in vitro, to determine if the simple interaction of these two elements could reproduce the transcription observed immediately after HSV-1 infection. HSV-1 DNA was transcribed in vitro by *Escherichia coli* RNA polymerase and by calf thymus RNA polymerase B. The RNA's obtained were 45%-50% self-complementary and had broad size distributions. The prokaryotic enzyme failed to transcribe the inverted repeats of the small component of the molecule. Small, unique regions of the large component were preferentially transcribed with this RNA polymerase. In contrast, all regions were uniformly transcribed by the eukaryotic enzyme. An approx distribution of initiation sites for both polymerases was determined. (23 refs)

- 79-2761 Cell Surface Glycoprotein Changes in Epstein-Barr Virus-Positive and -Negative Human Hematopoietic Cell Lines. (Eng) van Beek, W. P. (Div. Cell Biology, Anton van Leeuwenhoek Huis, Netherlands Cancer Inst., Amsterdam, Netherlands); Nilsson, K.; Klein, G.; Emmelot, P. *Int J Cancer* 23(4): 464-473; 1979.

Epstein-Barr virus (EBV)-positive and -negative human hematopoietic cell lines were studied with regard to their cell-surface glycopeptides. Fast-eluting glycopeptides were demonstrated by gel filtration analysis in 2 EBV-negative and 14 EBV-positive Burkitt's lymphoma (BL) cell lines, 14/15 EBV-negative lymphoblastoid cell lines, 5 non-BL lines, and 5/6 leukemia lines. One lymphoblastoid line derived from a patient with infectious mononucleosis, B

lymphoblasts stimulated by pokeweed mitogen, one leukemia line, and two myeloma lines did not contain the fast-eluting material. The BL lines were relatively insensitive to neuraminidase digestion and mild acid treatment, as were the lymphoblastoid cell lines. The non-BL, leukemia, and myeloma cell lines were sensitive to neuraminidase digestion. In general, the glycopeptide elution profiles of the lymphoblastoid lines were strikingly similar to those of the BL lines. The data support the hypothesis that BL develops from the stepwise malignant transformation of an EBV-infected population of B cells. (63 refs)

- 79-2762** Induction of the Epstein-Barr Virus (EBV) Cycle in Latently Infected Cells by n-Butyrate. (Eng) Luka, J. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm 60, Sweden); Kallin, B.; Klein, G. *Virology* 94(1): 228-231; 1979.

A dramatic increase in the number of virus-producer cells following treatment of the prototype Epstein-Barr virus (EBV)-carrying P3HR-1 and B95-8 lines with n-butyrate is reported. This compound was tested in view of its ability to induce differentiation in the Friend erythroleukemia system and to inhibit DNA synthesis. Induction was also seen in the nonproducer Raji and the low-producer Daudi lines, but at a much lower level. The virus-containing supernatant of the butyrate-treated P3HR-1 cells induced Epstein-Barr nuclear antigen preferentially in EBV-negative Ramos target cells, whereas the spontaneously produced virus induced predominantly EA in Raji indicator cells. This suggests a possible difference in the biological properties of the butyrate-induced vs the prototype virus. In addition to providing a convenient method to obtain a high yield of viral DNA and virus antigen-producing cells in the EBV system, these studies raise interesting questions concerning the mechanism of EBV induction and its possible relationship to the known differentiation inducing ability of n-butyrate. (24 refs)

- 79-2763** Interaction Between Epstein-Barr Virus and Type-C Virus in Human Cells. (Eng) Osato, T. (Dept. Virology, Cancer Inst., Hokkaido Univ. Sch. Medicine, Sapporo 060, Japan); Yamamoto, K.; Matsuo, T.; Aya, T.; Mizuno, F.; Nonoyama, M. *IARC Sci Publ* 20: 413-420; 1978.

The interaction between Epstein-Barr virus (EBV) and C-type virus in human lymphoid FVNC cells was studied to determine the significance of possible cofactor(s) influencing EBV oncogenesis. The FVNC cell line was established by exposing EBV genome-positive human lymphoid NC-37 cells to C-type Friend murine leukemia virus. Each FVNC cell contains a few EBV genomes and a C-type viral genome in repressed form. Compared with NC-37 cells,

FVNC cells showed three to four times more early antigen (EA)-positive cells after superinfection with EBV. C-type viral induction was also observed in FVNC cells following EBV superinfection. When FVNC cells were exposed to C-type virus, a high frequency of induction of EBV-related early nuclear antigen was demonstrated. The results of human-mouse somatic cell hybridization experiments suggested that the repressed EBV and C-type viral genomes may be associated with different chromosomes in FVNC human lymphoid cells. (5 refs)

- 79-2764** Early and Late Components of Epstein-Barr Virus-associated Membrane Antigen in Superinfected Daudi Cells and Their Reactivity with Sera from Nasopharyngeal Carcinoma Patients. (Eng) Hinuma, Y. (Dept. Microbiology, Kumamoto Univ. Medical Sch., Kumamoto, Japan); Sairenji, T.; Maeda, M. *IARC Sci Publ* 20: 385-390; 1978.

An indirect membrane immunofluorescence technique was used to study the appearance of early and late membrane antigen (MA) following superinfection of Daudi cells with the P3HR-1 strain of Epstein-Barr virus (EBV). The levels of MA, early antigen (EA), and viral capsid antigen (VCA) increased by 64%, 34%, and 23%, respectively, 22 hr after infection. Puromycin (25 µg/ml) inhibited MA, EA, and VCA synthesis, and cytosine arabinoside (ara-C, 20 µg/ml) or disodium phosphoacetate (200 µg/ml) inhibited the synthesis of MA and VCA, but not EA. Differential absorption of an EBV antibody-positive human serum with ara-C-treated or -untreated infected cells revealed two antigenically different components of MA: early MA (ara-C-insensitive) and late MA (ara-C-sensitive). Three of five sera from patients with nasopharyngeal carcinoma, but none of five sera from normal adult humans, showed an apparent "prozone" phenomenon in their reactivity against late MA, but not early MA. (9 refs)

- 79-2765** Polyclonal Immunoglobulin Secretion by Human B Lymphocytes Exposed to Epstein-Barr Virus In Vitro. (Eng) Kirchner, H. (Inst. Virus Res., German Cancer Res. Center, 69 Heidelberg, W. Germany); Tosato, G.; Blaese, R. M.; Broder, S.; Magrath, I. T. *J Immunol* 122(4): 1310-1313; 1979.

The Epstein-Barr virus (EBV)-induced activation of human peripheral blood lymphocytes was studied by a reverse hemolytic plaque assay for the detection of Ig-producing cells, and the results were compared with the effects of pokeweed mitogen (PMW) on these cells. Both agents caused the development of Ig-producing cells in cultures of unseparated mononuclear cells. However, B-cell populations sufficiently depleted of T cells by a variety of techniques to be unresponsive to PMW showed a marked response to EBV. The reactivity of B cells to PWM could

be restored by irradiated T cells, whereas there was no effect of irradiated T-cells on the reactivity to EBV. These data suggest that the response to EBV, in contrast to the PWM response, is T-cell-independent. Lymphocytes secreting each class of Ig (IgG, IgA, and IgM) were found in EBV-stimulated cultures of both unseparated mononuclear cells and T-cell-depleted cultures, demonstrating that the response in each Ig class is also T-cell-independent in this system. When unseparated cell populations and B-cell populations cultured at the same cell concentration were compared, the latter showed a two- to fivefold increased reactivity to EBV. This difference appeared to be caused primarily by an enrichment of B-cells, as was suggested by experiments in which the two cell populations were compared at different cell concentrations. (21 refs)

- 79-2766 Epstein-Barr Virus-related Antibody Patterns in Ataxia-Telangiectasia.** (Eng) Berkel, A. I. (Immunology Lab., Hacettepe Children's Hosp., Ankara, Turkey); Henle, W.; Henle, G.; Klein, G.; Ersoy, F.; Sanal, O. *Clin Exp Immunol* 35(2): 196-201; 1979.

Since Epstein Barr virus (EBV) has oncogenic potential and high titers of EBV-related antibodies are found in patients with decreased cellular immunity, these titers were determined in 27 patients with ataxia-telangiectasia (AT) and twenty-two healthy members of their families, 22 patients with other diseases (including 10 with Behcet's disease and 10 with various primary immune deficiencies) and 15 healthy members of their families, and 23 unrelated healthy controls. The AT patients showed an increased incidence (55.6%) of high antibody titers ($\geq 1:320$) to viral capsid antigen (VCA) and a high incidence (48.2%) of antibodies to EBV-induced early antigens (EA: ranging in titer from 1:10 to 1:60), but low titers ($< 1:10$) of antibodies to EBV-associated nuclear antigen (EBNA) in 44.2% of the cases. The geometric means of anti-VCA were three- to fourfold higher and those of anti-EBNA sixfold lower than the values for the control groups. The values for patients with the other diseases did not differ significantly from control values except for a higher incidence of anti-EBNA titers of $< 1:10$ (38.1%, vs 4%-5%). AT patients with low anti-EBNA titers tended to have more advanced T-cell deficiencies than AT patients with moderate anti-EBNA titers, as detected by counts of total lymphocytes and erythrocyte-rosetting cells and by skin test responses. The results support the hypothesis that a functioning T-cell system is required to release EBNA from EBV genome-carrying cells for initial and maintained production of anti-EBNA. (17 refs)

- 79-2767 Identification of the Soluble HVP-associated Antigen of the Lymphoblastoid Cell Line Established from Lymphomatous Baboon (*Papio***

***hamadryas*).** (Eng) Voevodin, A. F. (Lab. Experimental Oncology, Inst. Experimental Pathology and Therapy, USSR Acad. Medical Sciences, Sukhumi, USSR); Lapin, B. A.; Agrba, V. Z.; Timanovskaya, V. V. *Acta Biol Med Ger* 37(9): 1509-1512; 1978.

A new, indirect, double-immunodiffusion technique for the detection of Epstein-Barr virus (EBV)-associated antigen and antibody is described, and it was used to identify the soluble lymphomatous baboon herpesvirus (HVP)-associated antigen. The technique involves three steps: (1) simple double immunodiffusion with extracts of Raji cells (or other EBV genome-positive cells) and human sera containing antibodies against EBV-associated soluble antigen; (2) extensive washing and treatment of the preparations with anti-human globulin; and (3) extensive washing and tannic acid treatment. With this technique, the soluble HVP-associated antigen from a lymphoblastoid cell line established from lymphomatous baboon (KMPG-1) was shown to be indistinguishable from the soluble EBV-associated antigen. (23 refs)

- 79-2768 Human Papillomavirus DNA Detected in Two Verrucous Carcinomas (Meeting Abstract).**

(Eng) Ubben, K. (Dept. Dermatology and Microbiology, Univ. Minnesota Medical Sch., Minneapolis, MN); Krzyzek, R.; Ostrow, R.; Bender, M.; Zelickson, A.; Faras, A.; Pass, F. *Clin Res* 27(2): 538A; 1979 (no refs)

- 79-2769 Increase of Nuclear RNA Polymerase Activity: An Early Effect in Primary Mouse Kidney Cells Infected with Polyoma Virus or SV40 (Meeting Abstract).** (Eng) Pockl, E. (Institut für Molekularbiologie, Universität Wien, Wargasse 9, A-1090 Wien, Germany); Wintersberger, E. *Hoppe Seylers Z Physiol Chem* 360(3): 344; 1979 (no refs)

- 79-2770 Transplantation of Polyoma Virus-infected Bovine Odontogenic Tissues (Meeting Abstract).** (Eng) Chalk, A. J. (Dept. Dental Medicine and Surgery, Univ. Melbourne, Melbourne, Australia) *J Dent Res* 58(Special C): 1208; 1979 (1 ref)

- 79-2771 Multiple Forms of Polyoma Virus Tumor Antigens from Infected and Transformed Cells.** (Eng) Simmons, D. T. (Sch. Life and Health Sciences, Univ. Delaware, Newark, DE 19711); Chang, C.; Martin, M. A. *J Virol* 29(3): 881-887; 1979.

The relationship between the multiple forms of polyoma (Py) virus tumor antigens from infected and transformed

cells was examined by analyzing their methionine-containing tryptic peptides. At least three distinct forms of Py virus tumor antigens were isolated from productively infected and transformed hamster cells by immunoprecipitation with serum directed against Py-induced tumors (anti-T serum). Their proteins had approx mol wts of 105,000 (large T antigen), 63,000 (middle T antigen), and 20,000 (small T antigen), as estimated by acrylamide gel electrophoresis. An examination of the appearance of these antigens in Py-infected mouse cells showed that all three polypeptides were synthesized maximally at approx the same time after infection. Analysis of the methionine-containing tryptic peptides of these proteins indicated that the large, middle, and small forms of polyoma T antigens contained five similar or identical peptides. In addition, the 63,000- and 20,000-dalton antigens contained two other methionine peptides absent from the large T-antigen species. Other methionine peptides were found only in the large or middle T-antigen forms. These and previous results suggest that the three T-antigen species have the same NH₂-terminal end regions but different COOH termini. A model describing the synthesis of these polypeptides from different regions of the Py virus genome is presented. (22 refs)

- 79-2772 Biological Activity of Polyoma Viral DNA in Mice and Hamsters.** (Eng) Israel, M. A. (Lab. Biology Viruses, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Chan, H. W.; Hourihan, S. L.; Rowe, W. P.; Martin, M. A. *J Virol* 29(3): 990-996; 1979.

The biological activity of polyoma (Py) viral DNA was evaluated by studying its ability to initiate productive infection in weanling mice, as measured by the MAP (mouse antibody production) test, and to induce tumors in newborn hamsters. Viral DNA administered parenterally was 4-5 logs less efficient than Py virions in establishing infection in mice. Supercoiled viral DNA was infectious for mice after parenteral administration, giving mean infective doses of 10^{-3} to 10^{-4} μ g. However, animals fed microgram quantities of Py DNA I did not become infected. The relaxed, circular form of viral DNA (DNA II) was nearly as infectious as the supercoiled form, whereas full-length linear forms (DNA I cleaved with the single-cut restriction endonuclease R.*Bam*HI or R.*Eco*RI) retained 15%-25% of the infectivity determined for DNA I. Approx 10% of newborn hamsters inoculated ip with 0.5 μ g of Py DNA I developed tumors. In contrast, the same amount of viral DNA that had been cleaved in the early region with R.*Eco*RI induced tumors in 43%-50% of inoculated hamsters. The tumors observed after ip administration of Py DNA were all in the abdominal wall near the site of injection. These studies indicate that the efficiency of infection of mice by Py DNA is adequate to allow the use of mice as a model system in experiments requiring the expres-

sion of the Py genome, particularly in studies of recombinant DNA molecules. (37 refs)

- 79-2773 Early Events in the Infection of Permissive Cells with Polyoma Virus: Comparison of Chymotrypsin-treated and Untreated Virus.** (Eng) Chlumecka, V. (Dept. Biochemistry, Univ. Alberta, Edmonton, Alberta T6G 2H7, Canada); D'Obrenan, P.; Colter, J. S. *Virology* 94(1): 219-223; 1979.

Polyoma (Py) virus exposed to chymotrypsin during purification (chymo+ virions) and Py virus purified without exposure to the protease (chymo- virions) were examined with respect to the early events of the infectious cycle. In addition, the abilities of chymo+ and chymo- virions to stimulate the synthesis of cellular and viral DNA were compared, as were the infectivities of DNA's isolated from the two preparations. Exposure to the protease had no effect on the ability of the virions to attach to, penetrate into, and enter the nuclei of mouse embryo fibroblasts. Uncoating, which was concluded to take place exclusively in the nuclei, appeared to be somewhat delayed in the case of the chymo+ virions. The only identifiable product of uncoating was a DNA protein complex having a sedimentation coefficient of the order of 52S-55S. Stimulation of cellular and viral DNA synthesis was greatly reduced in cells infected with chymo+ virions relative to those infected with chymo- virions, but the specific infectivities of DNA's isolated from chymo+ and chymo- virions were precisely the same. (14 refs)

- 79-2774 DNA Sequence Alterations in Hr-t Deletion Mutants of Polyoma Virus.** (Eng) Hattori, J. (Dept. Pediatrics, Sidney Farber Cancer Inst., 44 Binney St., Boston, MA 02115); Carmichael, G. G.; Benjamin, T. L. *Cell* 16(3): 505-513; 1979.

The DNA sequence alterations in several host range- and transformation-defective (hr-t) mutants of polyoma virus were investigated. These mutants are defective in one of the two known viral functions essential for transformation, and they are altered with respect to several minor tumor (T) antigen species. The lesions in some of these mutants have been mapped previously by marker rescue experiments to Hpa II fragment 4 (Hpa II-4, 78.4-91.7 map units) in the proximal part of the early region of the viral DNA. Thirteen of sixteen hr-t mutants examined carry deletions 2 to 5 map units [100-250 base pair (bp)] long in Hpa II-4. Three mutants carry either point mutations or very small deletions/insertions. Eight of the deletion mutants were mapped closely with restriction enzymes. Seven of them have deletions located entirely within the Hae III subfragment A of Hpa II-4 (the Hae A subfragment, 78.4-85.2 map units), and one extends just beyond this subfragment, ending at 85.5 map units. The complete sequence of the wild-type

Hae A subfragment was determined and compared with those of four deletion mutants, NG-18, A-8, 6B-5, and B-2. The deletion in each of these mutants is out of phase: NG-18, 187 bp; A-8, 127 bp; 6B-5, 179 bp; B-2, 241 bp. All are expected to remove protein sequences in the C terminal part of the small t antigen. (42 refs)

- 79-2775 Restriction Endonuclease Cleavage Map of the DNA of JC Virus.** (Eng) Martin, J. D. (Dept. Medical Microbiology, Univ. Wisconsin Medical Sch., Madison, WI 53706); Frisque, R. J.; Padgett, B. L.; Walker, D. L. *J Virol* 29(3): 846-855; 1979.

A restriction endonuclease cleavage map of the DNA of JC virus (JCV), the human polyomavirus associated with progressive multifocal leukoencephalopathy, was derived. The following endonucleases were used: *EcoRI*, *HpaI*, and *PstI* (1 cleavage site each); *HindII* (4 sites); and *HindIII* (3 sites). Map position 0 was arbitrarily assigned to the *EcoRI* site, and the other cleavage sites were ordered with respect to it. The *PstI* site was placed at map position 0.315 and the *HpaI* site at 0.850. The four *HindII* sites were placed at map positions, 0.145, 0.355, 0.855, and 0.975, the three *HindIII* sites at map coordinates 0.550, 0.630, and 0.670. By agarose gel electrophoresis of fragmented DNA, the size of full-length DNA of JCV was estimated to be $5,125 \pm 105$ base pairs (98% \pm 2% of the length of simian virus 40 DNA). (24 refs)

- 79-2776 Persistent BK Papovavirus Infection of Transformed Human Fetal Brain Cells. I. Episomal Viral DNA in Cloned Lines Deficient in T-Antigen Expression.** (Eng) Takemoto, K. K. (Lab. Viral Diseases, Natl. Inst. Allergy and Infectious Diseases, Bethesda, MD 20014); Linke, H.; Miyamura, T.; Fareed, G. C. *J Virol* 29(3): 1177-1185; 1979.

Data relevant to two different virus-cell interactions, persistent infection and cellular transformation, are presented as a result of studies of cultures of human fetal brain (HFB) cells persistently infected with BK human papovavirus (BKV). After infection of permissive HFB cells by BKV, the vast majority of the cells were killed by the virus, but rare survivors were recovered after frequent medium changes. These surviving cells grew and formed visible colonies after 5-6 wk and were thereafter established as permanent cell lines. These cells, designated as BK-HFB cells, were persistently infected and shed BKV. Morphologically, they were small, polygonal cells that had transformed growth properties. Their plating efficiency on solid substrates or in semisolid medium was high, and they were tumorigenic in athymic nude mice. Cloning experiments in medium containing BKV antiserum revealed that BKV did not persist in the cultures in a simple carrier state. All cloned cell lines were initially tumor (T)-antigen

negative and virus-free. However, every clone began to release BKV and again became persistently infected within 3 wk after removal of BKV antiserum. After rigorous antibody treatment, 4/7 clones still released virus spontaneously upon removal of antiserum; three clones have remained virus-free and are apparently cured. Although these cloned cell lines are T- and V-antigen negative when grown in antiserum-containing medium, they retain "free" or episomal BKV genomes; integrated viral DNA was not detected in any of the clones. These free genomes are indistinguishable from prototype BKV DNA and are found in much larger amounts in virus-shedding cell lines. (34 refs)

- 79-2777 Mapping and Ordering of Fragments of BK Virus DNA Produced by Restriction Endonucleases.** (Eng) Freund, J. (Dept. Clinical Microbiology, Univ. Chicago, Chicago, IL 60637); di Mayorca, G.; Subramanian, K. N. *J Virol* 29(3): 915-925; 1979.

The 51 cleavage sites produced in the DNA of BK virus (BKV), a human papovavirus, by a combination of 10 different restriction endonucleases are described. These sites were mapped and ordered relative to one another as well as to the five fragments generated previously by the digestion of BKV with *HindIII* and *EcoRI*. One of the enzymes used in this study, *HaeIII*, recognizes the sequence GGCC (guanine-guanine-cytosine-cytosine), which is present within the genome of BKV 21 times. A large percentage of these sites are within the late region of the BKV genome, a situation similar to that in the simian virus 40 genome. The cleavage map produced by the 10 restriction endonucleases is being used to construct and characterize deletion mutations and to map the integration sites on the viral DNA of BKV-transformed cells. (32 refs)

- 79-2778 BK Virus DNA Sequence: Extent of Homology with Simian Virus 40 DNA.** (Eng) Yang, R. C. (Section Biochemistry, Molecular and Cell Biology, Cornell Univ., Ithaca, NY 14853); Wu, R. *Proc Natl Acad Sci USA* 76(3): 1179-1183; 1979.

The primary nucleotide sequence of three regions of BK virus (BKV) variant (MM) DNA was determined by direct DNA sequence analysis. The region between map positions 0.715 and 0.900 included the initiation points and partial coding sequences of the putative VP2 and VP3 proteins of BKV(MM), the amino acid sequences of which showed >80% homology with those of VP2 and VP3 of simian virus 40 (SV40). The sequence of a potential leader protein (protein X), 66 amino acids long for BKV(MM) and 62 long for SV40, is also deduced. The regions between 0.595 and 0.398 and 0.310 and 0.175 included the coding sequence for the entire small tumor (t) antigen and most of

the large T antigen of BKV(MM). The DNA sequence within these regions comprised >50% of the complete BKV(MM) genome and showed a 70% sequence homology with the corresponding regions of SV40 DNA. This high degree of homology is at variance with the reported homology values of 11%-20% estimated by hybridization measurements of heteroduplex analyses. Possible explanations for the discrepancies are presented. (20 refs)

- 79-2779 Transcription of the Cellular DNA Sequences in a Cloned Host-substituted SV40 DNA Variant.** (Eng) Hartman, J. R. (Dept. Virology, Weizmann Inst. Science, Rehovot, Israel); Laub, O.; Aloni, Y.; Winocour, E. *Virology* 94(1): 82-94; 1979.

The transcription of a cloned host-substituted simian virus (SV40) genome of defined structure was studied in BSC-1 monkey cells coinfecting with wild-type virus and in *in vitro* reactions with Sarkosyl nuclear extracts (transcription complex preparations) of the coinfecting cells. Evidence for the transcription of monkey DNA sequences in substituted SV40 was obtained in both systems. Efforts to detect similar transcripts in uninfected cells or in cells infected with wild-type SV40 alone were not successful. Both the highly reiterated and nonreiterated types of cellular DNA sequences (which are linked in the genome of the cloned substituted SV40 variant) were transcribed in the coinfecting cells, and the RNA transcripts were detected in the nuclear and cytoplasmic fractions. Relative to the amount of wild-type SV40 RNA, 40% of the RNA synthesized after *in vitro* incubation of transcription complex preparations hybridized with substituted SV40 cellular DNA sequences. In contrast, only 15% of the nuclear RNA and 4% of the cytoplasmic RNA from intact cells hybridized with the cellular DNA derived from substituted SV40. The sucrose gradient sedimentation profile of the host-substituted SV40 RNA was uniquely different from and more heterogeneous than that of wild-type SV40 RNA. RNA homologous to the host DNA in the substituted SV40 variant was associated only with lighter (disomal and monosomal) ribosomal fractions. (27 refs)

- 79-2780 Identification of New Polypeptide Species (48-55K) Immunoprecipitable by Antiserum to Purified Large T Antigen and Present in SV40-Infected and -Transformed Cells.** (Eng) Melero, J. A. (Dept. Pathology, New York Univ. Medical Center, 550 First Ave., New York, NY 10016); Stitt, D. T.; Mangel, W. F.; Carroll, R. B. *Virology* 93(2): 466-480; 1979.

Intermediate mol wt antigenic polypeptides were identified by immunoprecipitation and gel electrophoresis in cell lines infected with simian virus 40 (SV40). In each line there was a polypeptide with an apparent mol wt of 94,000 daltons [(94K): large tumor antigen (T Ag)], one with a mol wt of

approx 20K (small T Ag), and several with apparent mol wts between 48K and 55K. With respect to the latter polypeptides, the spectrum differed with cell type. Rabbit anti-T serum was able to immunoprecipitate the large T Ag and 48K bands separately, suggesting that they share antigenic determinants. The new antigens were not breakdown products that originated during the manipulations *in vitro*. Deletion temperature-sensitive mutants of SV40 mapping between 0.54 and 0.59 map units did not affect the appearance or size of the new polypeptide species. The SV40 mutant tsA58 produced a 55K species that was very stable at high and low temperatures, suggesting that the mutation did not affect the new antigen. The results suggest that the 48K-55K species may originate either as host-coded species (perhaps induced by the virus) that share determinants with large T Ag or perhaps as SV40-encoded species sharing only very little of the amino acid sequence of the 94K T Ag. (33 refs)

- 79-2781 T Antigen is Bound to a Host Protein in SV40-transformed Cells.** (Eng) Lane, D. P. (Dept. Zoology, Imperial Coll., London SW7, England); Crawford, L. V. *Nature* 278(5701): 261-263; 1979.

The formation of an oligomeric complex between simian virus (SV40) T antigen and a host protein is reported. When an extract of the SV40-transformed mouse cell line SVA31E7 was immunoprecipitated with a rabbit antiserum against purified T, two polypeptides were specifically immunoprecipitated. The more slowly migrating species had a mol wt of 94,000 and was identical to the T antigen. The faster migrating species had mol wt of 53,000 (53K) and was not detected in SV40 lytically infected monkey cells. These same two polypeptides were also specifically immunoprecipitated by other anti-T sera. The 53K protein separated from T by sodium dodecyl sulfate-gel electrophoresis was no longer precipitated by the rabbit anti-T serum, whereas the T antigen isolated in the same way was still fully reactive. The determinants recognized by the rabbit anti-T serum on T are, therefore, absent from the 53K polypeptide. Its immunoprecipitation from cell extracts by the rabbit serum must occur because it exists in a complex with T. It is postulated that the 53K protein is not virally coded since (1) it is not detectable in lytically infected monkey cells; (2) it shares no detectable antigenic determinants with T, but has its own set of antigenic determinants (immunogenic in syngeneic mice); and (3) DNA sequence and messenger RNA mapping data indicate that it would be difficult for the early region of SV40 to encode a third polypeptide of 53,000 mol wt. (25 refs)

- 79-2782 Experimental Viral Infections of the Inner Ear. II. Simian Virus 40 Induced Tumors of the Temporal Bone.** (Eng) Davis, L. E. (Dept. Neurology, Univ. New Mexico Sch. Medicine, Albuquerque, NM

87131); Nager, G. T.; Johnson, R. T. *Ann Otol Rhinol Laryngol* 88(2,part 1): 198-204; 1979.

The pathological, virological, and serological aspects of tumors of the temporal bone induced by inoculation of newborn Syrian hamsters with simian virus 40 (SV40) are presented. Of the 27 animals that reached weanling age, 23 developed tumors. Four to five months after viral inoculation, 21 hamsters developed undifferentiated sarcomas in the sc space adjacent to the temporal bone. Nine tumors invaded the temporal bone, occasionally extending to the subarachnoid space but not to the inner ear. Choroid plexus papillomas developed in four animals (2 of these animals also had sarcomas), with one tumor demonstrating invasion of the cochlear aqueduct, internal auditory canal, and cochlear modiolus. Cells grown from a sarcoma and a choroid plexus papilloma contained tumor antigen and established that the tumors were SV40-induced. (12 refs)

79-2783 Inability of Antiserum Active in Antibody-dependent Cellular Cytotoxicity and Arming Tests to Protect Against Simian Virus 40 Tumor Cell Challenge. (Eng) Prather, S. O. (Sch. Life Sciences, 319A Manter Hall, Univ. Nebraska, Lincoln, NB 68588); Geller, R. W.; Lausch, R. N. *J Natl Cancer Inst* 62(5): 1273-1277; 1979.

Results of further studies of the interaction of simian virus 40 (SV40) antiserum with hamster lymphoid cells are presented. Preincubation of nonadherent normal spleen, or lymph node cells with (SV40) antiserum from Syrian golden hamsters rendered the cells specifically cytotoxic for SV40-transformed hamster embryo fibroblasts (PARA-7 cells). The capacity of a particular antiserum to "arm" (ie, to be made specifically cytotoxic) was comparable with its ability to mediate antibody-dependent cellular cytotoxicity (ADCC). Experiments were performed to determine if the SV40 antiserum had prophylactic activity. Two hours after passive transfer of serum, cytotoxic effector cells could be demonstrated in the blood, spleen, and mesenteric lymph nodes, and the recipients' sera were active in ADCC tests. Nevertheless, these hosts were not resistant to challenge with small numbers of SV40 tumor cells that were given id or intracardiacally, nor was tumor growth suppressed by the addition of normal lymph node cells to the antiserum-pretreated tumor cell inoculum. Thus, SV40 antiserum, active at high titer in ADCC and arming assays, did not prevent or delay the growth of SV40 tumor isografts. (28 refs)

79-2784 Noncoordinate Expression of SV40-induced Transformation and Tumorigenicity in Mouse Cell Hybrids. (Eng) Howell, N. (Div. Molecular Genetics, Sidney Farber Cancer Inst., 44 Binney St., Boston, MA 02115); Sager, R. *Somatic Cell Genet* 5(1): 129-143; 1979.

The expression of transformation phenotypes and of tumorigenicity was analyzed in a series of somatic mouse cell hybrids formed by the fusion of nontumorigenic BALB/c 3T3 cells and the closely related simian virus 40 (SV40)-transformed SVT2 cells. The study was designed to probe the genetic basis of the multiple phenotypic changes induced by SV40 transformation. These hybrids showed noncoordinate expression of the transformation phenotype. Although they cloned at high efficiency in medium with low serum and expressed the SV40 tumor antigen of the SVT2 parent, the hybrid cells grew poorly without anchorage and exhibited a cell and colony morphology intermediate between that of the parents. Tumorigenicity was assayed quantitatively by sc coinjection into athymic nude mice of serial dilutions of 10^2 to 10^5 hybrid cells with 10^7 lethally irradiated 3T3 cells. The results showed that injection of 10 SVT2 cells was sufficient for tumor formation. In contrast, 100-1,000 times more hybrid cells had to be injected for tumor formation. These and other observations show that most 3T3/SVT2 hybrid cells are not tumorigenic but that each population contains a rare subset of tumorigenic cells. (44 refs)

79-2785 Reversion to Methionine Independence in Simian Virus 40-transformed Human and Malignant Rat Fibroblasts Is Associated with Altered Ploidy and Altered Properties of Transformation. (Eng) Hoffman, R. M. (Genetics Unit, Children's Service, Massachusetts General Hosp., Boston, MA 02114); Jacobsen, S. J.; Erbe, R. W. *Proc Natl Acad Sci USA* 76(3): 1313-1317; 1979.

Many transformed and malignant cells, unlike normal cells, do not grow when methionine (MT) in the growth medium is replaced by its immediate precursor homocysteine. However, rare cells from these populations revert to MT independence. Because MT auxotrophy is found only in transformed and neoplastic cells, experiments were conducted to determine whether MT-independent revertants also become normal with respect to properties associated with transformation. It was found that MT-independent revertants of both human fibroblasts transformed by simian virus (SV40) and malignant rat fibroblasts concomitantly reverted for some of the properties associated with the transformed state. Of the 13 MT-independent revertants described, 5 showed increased anchorage dependence, as reflected by reduced cloning efficiencies in methylcellulose; 8 showed an increased serum requirement for optimal growth; 8 showed decreased cell density in medium containing high serum; and 3 altered their cell morphology significantly. Eight of the 13 had increased chromosome numbers. All lines tested contained immunologically identifiable SV40 tumor antigen. Thus, by selecting for MT independence, it is possible to select for heterogeneous transformation revertants, which indicates that there is a relationship between altered MT metabolism and oncogenic transformation. Therefore, a positive metabolic method to select for transformation revertants

has been developed, and its use has resulted in the selection of human transformation revertants. (20 refs)

- 79-2786 Phospholipid Composition of Substrate Adhesion Sites of Normal, Virus-transformed, and Revertant Murine Cells.** (Eng) Cathcart, M. K. (Dept. Microbiology, Case Western Reserve Univ., Cleveland, OH 44106); Culp, L. A. *Biochemistry* 18(7): 1167-1176; 1979.

Evidence is presented that the phospholipid (PL) composition of the substrate adhesion site (AS) is considerably different from that of the whole cell and surface membrane preparations, that it is enriched in certain PL species, and that it is altered as a result of viral transformation. The PL composition of cell-substratum AS's, obtained after [ethylenebis(oxyethylenetriol)]tetraacetic acid-mediated detachment of cells from the tissue culture substratum, was determined for [32 P]orthophosphate-radiolabeled BALB/c 3T3, simian virus 40-transformed (SVT2), and concanavalin A-selected revertant variant cell lines. All major PL classes were found in the substrate-attached material, but there was an enrichment for specific PL species in this adhesive material compared with whole cell and surface-enriched membranes. The PL composition was remarkably similar for the whole cell and surface-enriched membrane fractions from the three cell lines. However, pronounced differences in the PL composition of the AS's were observed as a result of viral transformation--SVT2 sites were clearly enriched in phosphatidylethanolamine and depleted in phosphatidylcholine compared with the 3T3 sites. This alteration in AS PL's of transformed cells reverted to a 3T3-like value in the adhesive material of revertant cells. The composition of adhesive material of newly attaching cells was also examined to differentiate compositional differences between "footpad" adhesion sites and "footprints", adhesive material pinched off from the posterior of cells as they move across the substratum. Pulse and pulse-chase analyses of the [32 P]PL's revealed some differences in synthesis and turnover rates in the three cell lines; in addition, altered rates of deposition of newly synthesized material into AS's of transformed cells were observed. These data afford further evidence that the cell-substratum AS are highly specialized areas of the cell surface enriched in components that are intricately involved in adhesion. The transformation-dependent changes in AS may help to determine the basis for the altered adhesive properties of transformed cells. (49 refs)

- 79-2787 Ultrastructure of the Cell Coat of Untransformed and Simian Virus 40-transformed Fibroblasts.** (Eng) Dvorak, A. M. (Basic Res. Program, Frederick Cancer Res. Center, Frederick, MD 21701); Roblin, R. O.; Morgan, E. S.; Dvorak, H. F. *J Reticuloendothel Soc* 25(2): 163-177; 1979.

The ultrastructure of cell coats of cultured mouse and hamster fibroblasts and their simian virus 40 (SV40)-transformed counterparts was studied using an osmium potassium ferrocyanide (OPF) technique. Mouse 3T3 fibroblasts had abundant OPF-positive cell coats similar to those previously described on macrophages. By contrast, SV40-transformed 3T3 cells showed little or no OPF-positive material, and none was observed at points of cell contacts. Similar findings were obtained with hamster fibroblasts, except that less OPF-positive cell coat was present on untransformed cells compared with untransformed 3T3 cells. The OPF technique also stained a prominent system of cytoplasmic vesicles and vacuoles that were both open to the cell surface and were found as deep as the nuclear membrane. These dense vesicles were present in all four cell lines studied. They were most prominent in the SV40-transformed hamster cells and could represent a secretory system for placing cell coat materials on the cell surface. A migration inhibition factor-like material was secreted in much greater amounts by virus-transformed cells than by their untransformed counterparts. This material inhibited migration of guinea pig macrophages and reduced their OPF-positive cell coat. The increased production of this material may be responsible for reducing the amount of OPF-positive cell coat of SV40-transformed fibroblasts. (25 refs)

- 79-2788 Regulation of Simian Virus 40 Early and Late Gene Transcription without Viral DNA Replication.** (Eng) Birkenmeier, E. H. (Carnegie Inst., Baltimore, MD 21210); Chiu, N.; Radonovich, M. F.; May, E.; Salzman, N. P. *J Virol* 29(3): 983-989; 1979.

Primary cultures of African green monkey kidney cells were infected with the simian virus 40 (SV40) temperature-sensitive mutant *tsA58* at the nonpermissive temperature of 41 C for 12-20 hr. Under these conditions, a defective T antigen was produced and no viral DNA replication was detected. Viral transcription complexes were extracted from infected nuclei with Sarkosyl and the nascent RNA chains were elongated in vitro. From 60% to 70% of the viral RNA synthesized in vitro hybridized to late gene sequences. In contrast, 80%-90% of the nuclear viral RNA labeled in vivo during a 15-min pulse with 3 H-uridine hybridized to early gene sequences. This suggests that selective degradation of late gene transcripts occurs in vivo. The role of T antigen and viral DNA replication in regulation of SV40 transcription is discussed. (18 refs)

- 79-2789 Methylation and Cleavage Sequences of the *Eco*P1 Restriction-modification Enzyme.** (Eng) Bachi, B. (Biozentrum, Universität Basel, Klingelbergstrasse 70 CH-4056, Basel, Switzerland); Reiser, J.; Pirrotta, V. *J Mol Biol* 128(2):143-163; 1979.

The sites of cleavage and methylation in simian virus 40 (SV40) DNA were mapped and their sequences determined. Incubation of the DNA with *Eco*P1 and S-adenosylmethionine plus ATP resulted in cleavage, whereas methylation was observed in the absence of ATP. The methylation sites were restricted to five relatively small regions that agreed well with the positions estimated for cleavage. A-G-A-C-C appeared to be the entire recognition sequence for *Eco*P1 and, at least in vitro, *Eco*P1 methylated only one strand of the SV40 DNA. The enzyme cleaved the DNA 24-26 nucleotides away in the 3' direction from the site of methylation on one strand and 27-29 nucleotides away on the other strand, giving cuts staggered by 2, 3, or 4 bases, depending on the site. One variant of SV40 has acquired an additional *Eco*P1 methylation and cleavage site by changing an A-G-A-A-C sequence to A-G-A-C-C. (37 refs)

79-2790 Simian Virus 40: Another Way to Assemble a Virus? (Meeting Abstract). (Eng) Baumgartner, I. (Fachbereich Biologie, Universität Konstanz, Postfach 7330, D-7750 Konstanz, W. Germany); Kuhn, C.; Fanning, E. *Hoppe Seylers Z Physiol Chem* 360(3): 231; 1979 (no refs)

79-2791 An Investigation of a Spontaneous Lymphoma Epizootic in Hamsters (Meeting Abstract). (Eng) Vasa Thomas, K. A. (Univ. Tennessee, Knoxville, TN) *Diss Abstr Int B* 39(8): 3611; 1979 (no refs)

79-2792 Evolutionary Variants of Simian Virus 40: Replication and Encapsulation of Variant DNA. (Eng) Lee, T. N. (NCI, Bethesda, MD 20014); Nathans, D. *Virology* 92(2): 291-298; 1979.

The genomes of evolutionary variants of simian virus 40 (SV40) contain reiterations of DNA sequences derived from the origin of replication of viral DNA. To determine the role of such reiterated DNA segments in the evolution of variants, the relative rates of replication and the encapsidation efficiencies were determined for cloned variants with three, five, six, or nine copies of the SV40 origin sequence. The rate of variant DNA replication relative to that of the helper virus in the variant stock was an exponential function of the number of origin segments per genome. In contrast, encapsidation efficiency was not correlated with the number of origin sequences in the variant genome. It is concluded that a major factor in the evolution of SV40 variants is the replicative advantage conferred by the reiteration of the origin of DNA replication. (14 refs)

79-2793 Purification and Characterization of an Early Glycoprotein from Adenovirus Type 2-infected Cells. (Eng) Persson, H. (Dept. Microbiology, Biomedical Center, S-751 23 Uppsala, Sweden); Signas, C.; Philipson, L. *J Virol* 29(3): 938-948; 1979.

The purification and characterization of an adenovirus type 2 (Ad2) early glycoprotein with an apparent mol wt of 19,000 (E19K) in sodium dodecyl sulfate-polyacrylamide gels are reported. In addition, synthesis of this protein in Ad2-infected HeLa and Ad2-transformed hamster and rat cells was investigated with a monospecific antiserum raised against the purified protein. Purification involved detergent solubilization of membrane fractions from infected cells, followed by affinity chromatography on a lectin column and diethylaminoethyl Sephadex chromatography. The purified material contained three polypeptides (E40K, E19K, E17.5K), with approx 90% of the material being in the E19K moiety. All three polypeptides yielded identical tryptic peptide maps. The E19K polypeptide contained glucosamine as revealed by [³H]glucosamine labeling of infected cells and amino acid analysis of the purified protein. Immunoprecipitation with a monospecific antiserum showed that synthesis of the E19K polypeptide began at 2 hr after infection, with a max rate occurring at 4 hr after infection. The polypeptide was also synthesized at a low rate late in the infectious cycle (12-24 hr postinfection). Immunoprecipitation from three Ad2-transformed hamster embryo cell lines and two Ad2-transformed rat cell lines revealed that one of the hamster cell lines (ad2HE₄) and one of the rat cell lines (A₂T₂C₄) expressed this protein. (33 refs)

79-2794 Premature Termination During Adenovirus Transcription. (Eng) Evans, R. (Rockefeller Univ., New York, NY 10021); Weber, J.; Ziff, E.; Darnell, J. E. *Nature* 278(5702): 367-370; 1979.

Evidence for premature chain termination during the transcription of adenovirus type 2 (Ad2) in HeLa cells is reported. Ad2-specific RNA labeled in vitro in nuclei from infected cells showed a bimodal distribution of Ad2-specific chains, with both large (approx 45S preribosomal RNA) and small (approx ≤18S) sequences being observed. Similar results were obtained in vivo. The 6S-18S region from one preparation was hybridized to a series of fragments of the Ad2 genome prepared by restriction endonuclease digestion. The results indicated that premature RNA chain termination occurred after about 1,000-2,000 nucleotides in vitro and in vivo. To determine the frequency of premature termination, the amount of RNA synthesized in 45 sec that was complementary to a variety of DNA fragments was measured. There was three- to sixfold more labeled RNA complementary to 16.45-18.2 than to any other DNA fragment. In calculating the molarity of transcription from the 11.1-18.2 region, an assumption was made that all transcription reads rightward beginning at

16.45. Thus, three to six times as much transcription occurred in this region as in other regions of the large late transcription unit. Although it was not possible to conclude that there were specific termination sites, the extramolar synthesis did not extend past 25.5-27.9. Very few if any of the extramolar premature transcripts appeared as stable messenger RNA in the cytoplasm. It appears that it is RNA polymerase II that prematurely terminates RNA chains within the first 2,000 nucleotides of the large Ad2 transcription unit. (27 refs)

79-2795 Host Response to Adenovirus 2-transformed Hamster Embryo Cells. (Eng) Cook, J. L. (Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Lewis, A. M. *Cancer Res* 39(5): 1455-1461; 1979.

Several adenovirus 2 (Ad2)-transformed LSH hamster embryo cells were isolated, and their tumor-inducing capacity was defined. Ad2 inactivated by UV was used to transform the cells. Evaluation of transformed cell lines produced by Ad2 (prototype strain adenoid 6) or isolates of Ad2 obtained from children in Washington, DC, or West Bengal, India, showed that 13/15 lines induced tumors when injected into newborn inbred LSH or randomly bred NIH hamsters [10^7 cells/hamster, with a median tumor incidence in syngeneic newborns of 65% (range 8%-100%)]. Six of these cell lines did not induce tumors when 10^7 cells were injected sc into weanling animals 21 days old. Similarly derived cell lines transformed by Ad12 or simian virus 40 (SV40) uniformly produced tumors in weanlings following sc inoculation of 10^7 cells. Transplantation of tumors from animals treated as newborns with Ad2-transformed cell lines to other newborns was readily accomplished in 639/640 inoculated newborns. However, only 53/467 weanling hamsters challenged with these tumor lines developed neoplasms. Each of these Ad2-transformed lines contained Ad2 tumor (T) antigen detectable by complement fixation or immunofluorescence. None of six lines yielded Ad2 by Sendai fusion with human embryonic kidney cells or other contaminating agents by a variety of assays. The difference in oncogenicity in weanling hamsters of Ad2-transformed cells and Ad2 newborn tumor lines compared with Ad12- and SV40-transformed cells was not related to tumor dose injected or to differences in in vitro growth properties (eg, doubling times, saturation densities, growth in spinner medium, or colony formation in soft agar). With the two newborn tumor lines tested, weanling rejection of tumor transplants from newborn hamsters could not be overcome by inocula containing 25 times the usual dose of tumor suspension. These two lines produced progressively enlarging neoplasms in 17%-77% of syngeneic weanlings that had been thymectomized as newborns. The results suggest that the production of virus-transformed cells that are not oncogenic in immunocompetent syngeneic hamsters is a general property of Ad2 and that the Ad2-transformed hamster cell system may be a

useful model for studying the interaction of the viral genome, the transformed cell, and developing host immunity in virus-induced carcinogenesis. (32 refs)

79-2796 Thirty-One Human Adenovirus Serotypes (Ad1-Ad31) Form Five Groups (A-E) Based upon DNA Genome Homologies. (Eng) Green, M. (Inst. Molecular Virology, Saint Louis Univ. Sch. Medicine, 3681 Park Ave., St. Louis, MO 63110); Mackey, J. K.; Wold, W. S.; Rigden, P. *Virology* 93(2): 481-492; 1979.

Five distinct DNA homology groups, A to E, were identified among 31 human adenovirus serotypes (Ad1-Ad31) by liquid-phase molecular hybridization with the use of in vitro-labeled viral DNA as a probe. Members within all groups except group A were closely related. In general the DNA homology groupings are consistent with the properties of other "groupings" of human Ad's (oncogenic groups, tumor-antigen groups, G + C content of viral DNA, etc). (60 refs)

79-2797 Transforming Region of Group A, B, and C Adenoviruses: DNA Homology Studies with Twenty-Nine Human Adenovirus Serotypes. (Eng) Mackey, J. K. (Inst. Molecular Virology, Saint Louis Univ. Sch. Medicine, St. Louis, MO 63110); Wold, W. S.; Rigden, P.; Green, M. *J Virol* 29(3): 1056-1064; 1979.

The homology of adenovirus type 5 (Ad5), Ad7, and Ad12 transforming restriction endonuclease DNA fragments with DNA's of 29 Ad types was studied. Ad5 *HindIII*-G (map position 0-7.3), Ad7 *XhoI*-C (map position 0-10.8), and Ad12 (strain Huie) *EcoRI*-C (map position 0-16) and *SaII*-C (map position 0-10.6) fragments were purified, labeled in vitro (nick translation), and annealed with DNA's of Ad1-Ad16, Ad18-Ad24, and Ad26-Ad31. Hybrids were assayed with the use of hydroxylapatite. Ad5 *HindIII*-G hybridized 98%-100% with DNA's of Group C Ad's (Ad1, 2, 5, 6), but only 1%-15% with DNA's of other types. Ad7 *XhoI*-C fragment hybridized 85%-99% with DNA's of Group B Ad's (Ad3, 7, 11, 14, 16, 21), but only 6%-21% with DNA's of other types. Ad12 (Huie) *EcoRI*-C hybridized 53%-68% with DNA's of five other Ad12 strains, 53% with Ad18 DNA, 56% with Ad31 DNA, but only 3%-13% with DNA's of other types. In vitro-labeled Ad12 (Huie) *SaII*-C hybridized 35-71% with DNA's of six other Ad12 strains, 44% with Ad18 DNA, 52% with Ad31 DNA, but only 2%-7% with DNA's of Ad7, Ad2, Ad26, or Ad4. When assayed using S-1 nuclease, *SaII*-C annealed 17%-44% with DNA's of Group A Ad's (Ad12, 18, 31). The melting temperatures of the hybrids of Ad5 *HindIII*-G with all Group C Ad DNA's were 84 C in 0.12 M sodium phosphate (pH 6.8). The melting temperature of the Ad12 (Huie) *EcoRI*-C hybrid with Ad12 (Huie) DNA was 83 C, but it was only 71-77 C with DNA's of other Group A

Ad's. Thus, Groups C and B Ad's both have very homologous transforming regions that are not represented in DNA's of non-Group C Ad's or non-Group B Ad's, respectively. Similarly, Group A Ad's have unique but less homologous transforming regions. These different transforming nucleotide sequences may be reflected in the different oncogenic properties of Groups A, B, and C Ad's in newborn hamsters (highly, weakly, and nononcogenic, respectively). (57 refs)

79-2798 Association of Adenovirus Early Proteins with a Nuclear Fraction That Synthesizes DNA In Vitro. (Eng) Shaw, C. H. (Div. Virology, Natl. Inst. Medical Res., Mill Hill, London NW7 1AA, England); Russell, W. C.; Rekosh, D. M. *Virology* 92(2): 436-448; 1979.

A soluble subnuclear fraction isolated from adenovirus type 5-infected HeLa cells that synthesizes adenovirus DNA in vitro is described. This synthesis is inhibited by immunoglobulins directed against virus-specified proteins synthesized early in infection, but not by immunoglobulins directed against the viral coded tumor antigen (T antigen). Sucrose gradient analysis of the fraction demonstrated that it contained two distinct nucleoprotein complexes. One of these complexes sedimented at about 100S and contained replicating viral DNA molecules. The other, which sedimented at about 40S, contained mature double-stranded DNA of genome length. After a pulse-label with ³⁵S-methionine, the virus-coded 72,000-dalton (72K), single-stranded DNA binding protein (DBP) was found associated with the 100S peak but not with the 40S peak. Following a chase period, however, the DBP also appeared in the 40S peak. Labeling with ³²PO₄ also showed that the phosphorylated form of the 72 K DBP is found initially only in the 100S peak. These results suggest that newly made and phosphorylated DBP associates preferentially with replicating viral DNA. (43 refs)

79-2799 An Adenovirus Protein with the Ends of Replicating DNA Molecules. (Eng) Stillman, B. W. (Dept. Microbiology, John Curtin Sch. Medical Res., Australian Natl. Univ., Canberra, A.C.T. 2601, Australia); Bellett, A. J. D. *Virology* 93(1): 69-79; 1979.

A simple assay for adenovirus DNA-protein complex is described in which DNA, covalently linked to the terminal protein, specifically binds to benzoylethanol-cellulose. The DNA-protein complex can be recovered intact from the column by elution with urea and sodium dodecyl sulfate, or the DNA moiety can be eluted after incubation of the column with protease. With this system, protein was shown to be associated with pulse-labeled single strands of DNA from the terminal restriction enzyme fragments of replicating adenovirus 5 DNA. (42 refs)

79-2800 Three Different Classes of Human Adenovirus Transforming DNA Sequences: Highly Oncogenic Subgroup A-, Weakly Oncogenic Subgroup B-, and Subgroup C-Specific Transforming DNA Sequences. (Eng) Fujinaga, K. (Dept. Molecular Biology, Cancer Res. Inst., Sapporo Medical Coll., Sapporo 060, Japan); Sawada, Y.; Sekikawa, K. *Virology* 93(2): 578-581; 1979.

To compare transforming gene sequences among members of highly oncogenic subgroup A, weakly oncogenic subgroup B, and transforming subgroup C adenoviruses (Ad), DNA-DNA homology measurements were carried out using labeled Ad12 *Hind*III-G (subgroup A), Ad7 *Hind*III-I.J (subgroup B), and Ad5 *Hind*III-G (subgroup C). The measurements revealed three different classes of transforming DNA sequences: one specific for subgroup A (types 12, 18, and 31), the second specific for subgroup B (types 3, 7, and 16), and the third specific for subgroup C (types 2, 5, and 6). The transforming DNA sequences with mol wts of 1.6 to 1.8 x 10⁶ were identical or almost identical members of the same subgroup. One-third or less of the transforming DNA sequences were shared by members of different subgroups. All of the transforming gene sequences were located within a region at the terminal 10% of the viral DNA molecule. The results suggest that different virus-coded transforming proteins are involved in carcinogenesis by these three subgroups of human Ad. (15 refs)

79-2801 Adenovirus-Type-12-induced Rat Brain Tumor Cells: Persistence and Expression of the Viral Genome (Meeting Abstract). (Eng) Ibelgauf, H. (Institut für Genetik der Universität Köln, Cologne, W. Germany); Doerfler, W.; Wechsler, W. *Hoppe Seyler's Z Physiol Chem* 360(3): 289; 1979 (no refs)

79-2802 Identification of Adenovirus Type 12 Candidate Transformation Proteins by Radioimmunoprecipitation with Antisera to *Eco*RI-C-Fragment Transformed Cells. (Eng) Wold, W. S. (Inst. Molecular Virology, St. Louis Univ. Sch. Medicine, 3681 Park Ave., St. Louis, MO 63110); Chinnadurai, G.; Green, M.; Mak, S. *Virology* 94(1): 208-213; 1979.

Polypeptides (PP) coded by early gene block 1 (which includes the transforming region) of Group A human adenoviruses (Ad12, 18, 31) were identified by radioimmunoprecipitation with antisera to *Eco*RI-C fragment-transformed cells. Rat cell lines C-1 and C-2, which were transformed by transfection with the Ad12 *Eco*RI-C fragment (left 16% of genome) were inoculated into syngeneic rats to produce tumors. The tumor sera were used to immunoprecipitate [³⁵S]methionine-labeled PP from Ad12-infected human cells in the early stage of infection. The PP were resolved by electrophoresis in sodium

dodecyl sulfate-polyacrylamide gels and visualized by fluorography. Two additional sera from hamsters bearing tumors induced by inoculation with Ad12 or Ad18 virions were also used. The following PP were immunoprecipitated by the four sera: C-1 serum: 46,000 daltons (46K), 44K, 43K, 40K; C-2 serum: 46K, 44K, 43K, 40K, 16.5K, 10.5K; Ad12 and Ad18 sera: 65K, 60K, 55K, 54K, 47K, 45K, 11K, 10.5K. The sequence relationships between these PP were investigated by the partial proteolysis procedure using *Staphylococcus aureus* V8 protease. The data suggested the following conditions: (1) the C-1- and C-2-specific 46K, 44K, 43K, and 40K PP are highly related; (2) the Ad12- and Ad18-specific 65K, 60K, 55K, 54K, 47K, and 45K PP are highly related; (3) the C-1- and C-2-specific 40-65K PP may be related to, but are not identical with, the Ad12- and Ad18-specific 45-65K PP; (4) all three 10.5K PP are highly related; (5) both 11K PP are highly related and probably are unrelated to the 16.5K or 10.5K PP. The immunoprecipitation results suggest (but do not prove) that the early gene block 1 of Group A Ad's may code a "family" of related PP with apparent mol wts ranging from 40K to 46K or from 40K to 65K, as well as PP of 16.5K and 10.5K. One or more of these PP may play a role in the initiation and/or maintenance of cell transformation. (32 refs)

79-2803 Requirement of Adenovirus Type 12 Gene 401 Function for Initiation of Virus DNA Synthesis. (Eng) Ledinko, N. (Dept. Biology, Univ. Akron, Akron, OH 44325) *J Gen Virol* 42(3): 651-654; 1979.

Virus DNA synthesized in human cells infected with the highly oncogenic adenovirus type 12 (Ad12) temperature-sensitive mutant (H12/*ts*401) following temperature shift-up was characterized. Replicating DNA in human embryo kidney cell cultures infected with either *ts*401 or wild-type human Ad12 was converted into a predominant mature form of virus DNA following a shift from the permissive temperature (31°C) to the nonpermissive temperature (38.5°C). This suggests that the *ts*401 mutant is not defective in virus DNA chain propagation. To determine whether the *ts*401 mutant is defective in the initiation of virus DNA synthesis at the nonpermissive temperature, density labeling experiments in which cells infected with wild-type or *ts*401 virus were incubated with 5-bromodeoxyuridine and ³H-thymidine were carried out. The results showed that the replicating mutant DNA apparently completes a round of replication at the nonpermissive temperature, thereby incorporating bromouracil in the portion of the daughter strand remaining to be replicated. The absence of mutant DNA heavier than hybrid density under the restrictive condition suggests that there is no reinitiation in the replicated molecule. The findings are in agreement with the concept that the gene 401 product is required to initiate, but not to complete, the replication of virus DNA in lytic infection. (8 refs)

79-2804 Cell Origin of Human Adenovirus Type 12-induced Subcutaneous Tumor in Syrian Hamsters. (Eng) Nakajima, T. (Dept. Pathology, Natl. Cancer Center Res. Inst., Tokyo 104, Japan); Mukai, N. *Acta Neuropathol (Berl)* 45(3): 187-194; 1979.

To determine the cell origin of the characteristic adenovirus type 12 (Ad12) tumors derived from the sc tissue of hamsters, Ad12 was inoculated sc into the backs of newborn Syrian hamsters. Twenty-one of 25 animals developed multiple sc tumors close to the inoculation site within 21-90 days; 4 animals also had hepatic neoplasms. Uniform neuroblastic tumor cells forming incomplete rosettes appeared to have encroached into various atrophied muscle fibers. Mitotic figures and bizarre giant cell formations were common. Ad12 virions were demonstrated at each icosahedron symmetry. Many tumor cells possessed a solitary projecting cilium, and cytoplasmic expansions resembling neuroblastic growth cones were also seen. The tumor cells showed numerous tumor (T)-antigen-positive filaments within their cytoplasm. Sequential histologic examination revealed a severe inflammatory reaction, predominantly neutrophilic, at 48 hr. This gradually changed to a lymphocytic reaction that persisted during the 240-hr observation period. Intracytoplasmic T antigens were detectable at 48 hr. The T-antigen-positive cells began to decrease in number and to localize in the muscle layer at 72 hr, and at 240 hr, they retained a close association with the muscle layer. An incipient tumor cell aggregate was found in the interstices of maturing muscle fibers. A sequential study in parallel with electron microscopic examination of normal sc tissue proved that neuroblastic cells closely associated with the muscle spindle anlage could preferentially become the most sensitive target for AD12 tumorigenesis. (21 refs)

79-2805 Cytoplasmic Hepatitis B Surface Antigen and the Ground-Glass Appearance in Hepatocellular Carcinoma. (Eng) Wu, P. C. (Dept. Pathology, Univ. Hong Kong, Queen Mary Hosp. Compound, Hong Kong, China); Lam, K. C. *Am J Clin Pathol* 71(2): 229-234; 1979.

Hepatitis B surface antigen (HBsAg) was identified by an aldehyde fuchsin and immunoperoxidase staining technique and by immunofluorescence in malignant hepatocytes with a ground-glass appearance in only one needle biopsy specimen of a series of specimens taken at a Hong Kong hospital from 130 consecutive patients with hepatocellular carcinoma. The case report of the positive patient, a 14-yr-old girl, is given. HBsAg was identified by aldehyde fuchsin stain in the nonmalignant hepatocytes of 48/83 biopsy specimens that contained nonmalignant liver tissue. The antigen was demonstrable in significantly greater proportions in younger patients. A similar but not identical age relationship was found for hepatitis B antigenemia in Hong Kong. Apparently, the ability to produce HBsAg

declines with age. The usual absence of demonstrable HBsAg in hepatocellular carcinoma cells may be due to a failure of this characteristic to survive into the malignant cell line. Therefore, one cannot rule out the possibility that hepatitis B virus plays a direct role in the pathogenesis of hepatocellular carcinoma. In exceptional circumstances, as when hepatocellular carcinoma appears at an unusually early age, this marker is identifiable in the tumor cells. (24 refs)

79-2806 Crohn's Disease Lymph Node Homogenates Produce Murine Lymphoma in Athymic Mice (Meeting Abstract). (Eng) Das, K. M. (Albert Einstein Coll. Medicine, Bronx, NY 10461); Morecki, R.; Lev, M. *Gastroenterology* 76(5, part 2): 1117; 1979 (no refs)

79-2807 Characterization of the Viral Reverse Transcriptase of a C-Type RNA Virus Produced by an Established Human Lymphoma Cell Line (Meeting Abstract). (Eng) Goodenow, R. S. (Stanford Univ., Stanford, CA 94305) *Diss Abstr Int [B]* 39(9): 4193; 1979 (no refs)

79-2808 Prostate and Transitional Cell Carcinoma: Radioimmunoassay of Viral Tumor-associated Antigens. (Eng) Mickey, D. D. (Duke Univ. Medical Center, Box 3062, Durham, NC 27710); Paulson, D. F. *Nail Cancer Inst Monogr* (49): 51-63; 1978.

Competition radioimmunoassay was used to detect the p30 core protein of C-type RNA tumor viruses in partially purified extracts from human urothelial tumors. Fourteen

percent of benign hyperplastic and adenocarcinoma tissues were positive for p30 antigen from murine leukemia virus (MuLV) and 22% were positive for feline leukemia virus (FeLV) p30 antigen. Of the extracts from 47 surgical specimens grown in short-term tissue culture, 72% were positive for FeLV antigen, but only 29% of 28 extracts were positive for MuLV antigen. It is unlikely that the cultured cells were producing virus particles and releasing them into the culture medium. Sera collected from patients with urogenital carcinomas at various stages showed no antibody activity. It is concluded that urogenital carcinomas do not contain whole RNA tumor viruses and do not contain viruslike proteins in such quantities that they would be useful as screening reagents. However, competing proteins were present in 25% of the carcinoma tissue tested. Identification of the antigenic cross-reactive proteins as viral or nonviral depends on further purification and characterization. (13 refs)

See also:

*(Rev.): 79-2470, 79-2471, 79-2472, 79-2473, 79-2474, 79-2475, 79-2476.

*(Chem.): 79-2560, 79-2564, 79-2594, 79-2643.

*(Phys.): 79-2665, 79-2672.

*(Immun.): 79-2809, 79-2812, 79-2820.

*(Path.): 79-2829, 79-2861, 79-2898.

*(Epid-Biom.): 79-2918.

- 79-2809 **Prevention of Spontaneous Leukemia in AKR Mice by Passive Immunization and Type Specificity of This Protection.** (Eng) Fish, D. C. (Viral Oncology Program, Frederick Cancer Res. Center, Frederick, MD 21704); Gilden, R. V.; Bare, R. M.; Trimmer, R. M.; Huebner, R. J. *J Natl Cancer Inst* 62(4): 943-945; 1979.

The development of spontaneous leukemia in untreated AKR mice and in mice immunized for the first 14 or 31 days of life with goat immune globulin prepared from the sera of goats immunized with radiation leukemia virus was compared. The incidence of leukemia in normal mice at 1 yr of age was 72%, whereas that in mice given 5 immunization injections during the first 14 days of life (Group A) was 19% and that in mice given 10 immunization injections during the first 31 days of life (Group B) was 0%. All differences were significant. By day 500 of life, virtually all untreated mice had died of leukemia, whereas the incidence of leukemia in the Groups A and B mice was 40% and 8% respectively. The incidence at 700 days was still only 8% in the Group B mice. Thus, it appears that the protection in the Group A mice was more in the form of a delay in onset than a complete protection, but essentially lifelong immunization had occurred in the Group B mice. Immune globulin with a high neutralization titer for ecotropic virus provided this lifelong protection, but antibody with a high neutralization titer for murine xenotropic virus did not. (11 refs)

- 79-2810 **The Tumor Dormant State. Comparison of L5178Y Cells Used to Establish Dormancy with Those That Emerge after Its Termination.** (Eng) Weinhold, K. J. (Dept. Microbiology, Thomas Jefferson Univ., Philadelphia, PA 19107); Miller, D. A.; Wheelock, E. F. *J Exp Med* 149(3): 745-757; 1979.

The origin and growth rate of tumor cells that eventually emerge from the tumor dormant state (TDS) were studied by comparing various properties of original and emergent L5178Y lymphoma cells. The TDS established in L5178Y-immunized and -challenged DBA/2 mice was characterized by a prolonged period of clinical normalcy followed by rapid tumor outgrowth. The tumor cells that emerged after termination of the TDS had abnormal marker chromosomes identical to those in the L5178Y cells used in the original challenge inoculum, indicating that the emergent tumor cells were progeny of the challenge inoculum. Original and emergent L5178Y cells had equivalent *in vivo* growth rates when inoculated into normal DBA/2 mice. The emergent L5178Y cells were less susceptible than original cells to *in vitro* lysis by tumor dormant peritoneal cells. Original and emergent L5178Y cells

expressed common tumor-associated target antigens for cytolytic effector cells. Both modulation and masking of these target antigens were ruled out as mechanisms for decreased susceptibility to cell-mediated cytotoxicity. Immunofluorescence revealed heterogeneity in tumor-associated antigen (TAA) expression within both original and emergent cell populations, with decreased intensity of staining in the emergent population. Both populations were equally susceptible to lysis by alloimmune cells, alloantisera, and anti-Thy 1.2 serum, but emergent cells were less susceptible to lysis by serum directed against L5178Y TAA. Quantitative absorption revealed that the emergent L5178Y cells expressed eightfold less serologically detectable TAA than the original cells. These findings indicate that the host immune response developing during establishment of the TDS selects a stable tumor cell subpopulation that expresses decreased amounts of surface tumor-associated target antigens. (25 refs)

- 79-2811 **The Tumor Dormant State. Quantitation of L5178Y Cells and Host Immune Responses During the Establishment and Course of Dormancy in Syngeneic DBA/2 Mice.** (Eng) Weinhold, K. J. (Dept. Microbiology, Thomas Jefferson Univ., Philadelphia, PA 19107); Goldstein, L. T.; Wheelock, E. F. *J Exp Med* 149(3): 732-744; 1979.

Early events involved in establishment of the tumor dormant state are reported and related to the subsequent prolonged clinical remission and eventual termination of tumor dormancy. Sc implantation of DBA/2-derived L5178Y lymphoma cells into DBA/2 mice, followed 10 days later by nodule excision, protected 100% of mice from the rapid outgrowth of an ip challenge of L5178Y cells given 7 days postexcision. Challenged mice remained clinically normal for 48-250 days before the onset of an ultimately fatal tumor outgrowth. The numbers of L5178Y cells in the peritoneal cavity increased logarithmically for 4 days after challenge and then declined to low but detectable levels that persisted throughout the clinically normal period. Cells active in 18-hr *in vitro* cytotoxic assays against ⁵¹Cr-labeled L5178Y target cells were found in the peritoneal cavity. The effector cells were determined to be Thy 1.2-positive. Their activity was tumor-specific and reached peak levels 4 days after tumor challenge and then gradually declined to undetectable levels during the following 70 days. Tumor emergence occurred most frequently during the period when cell-mediated cytotoxicity was no longer demonstrable in the remaining clinically normal mice. A transient peak of low-level cytophilic antitumor antibody was detected about 30 days after tumor cell challenge. The temporal association between the numbers

of tumor cells and the levels of cell-mediated lysis against L5178Y cells indicate the importance of the cell-mediated cytotoxicity response in limiting initial tumor outgrowth and suggest its role as one of the factors responsible for long-term tumor suppression during tumor dormancy. (38 refs)

- 79-2812 Tumor-associated Antigens of Chemically-induced Murine Tumors; The Emergence of MuLV and Fetal Antigens after Serial Passage in Culture.** (Eng) Cleveland, P. H. (Surgical Oncology Service, Dept. Surgery, Univ. California, San Diego, CA); Belnap, L. P.; Knotts, F. B.; Nayak, S. K.; Baird, S. M.; Pilch, Y. H. *Int J Cancer* 23(3): 380-391; 1979.

Using radioiodinated *Staphylococcus aureus* protein A to measure syngeneic, allogeneic, and heterogeneic IgG bound to murine tumor cells, serological analyses of the surface antigens of 3 chemically induced colon carcinomas, 3 chemically induced sarcomas, 1 murine leukemia virus (MuLV)-induced leukemia, 1 radiation-induced leukemia, 1 spontaneous melanoma, and 1 spontaneous sarcoma of BALB/c mice were performed. Unique tumor-associated antigens were found on 3 of the tumors, MuLV-related antigens were found on 8, fetal antigens were found on 7, and two distinct common antigens were found on 7. Common antigen 1 was found on 5/7 latter tumors, and common antigen 2 was found on two. Neither of the common antigens was sarcoma-, carcinoma-, or tissue-type-specific. A number of tumors that did not originally express either MuLV or fetal antigens in primary cultures expressed these antigens after several serial passages in vitro. (40 refs)

- 79-2813 Teratocarcinoma Cells and Cell Surface Differentiation.** (Eng) Edidin, M. (Biology Dept., Johns Hopkins Univ., Baltimore, MD 21218); Ostrand-Rosenberg, S.; Bartlett, P. F. *30th Ann Symp Cancer Res* 67-79; 1978.

The detection of embryo-associated surface antigens on mouse teratocarcinoma cells is reviewed. The properties of the tumor model and the reactivities of some of the antisera prepared against it are covered briefly. Three separate families of antigens were detected by an antiserum prepared against the 402AX teratocarcinoma: antigen I was present on many tumor cells and on teratoma cells but not on normal cells, and it was physically associated with the major histocompatibility (H-2) antigens; antigen II was found in some tumor cells and on teratoma cells; and antigen III was unique to the teratoma cells. Teratocarcinoma cells also expressed antigens determined by some elements of the H-2 complex. Anti-402AX serum reacted well by immunofluorescence with all early stages (up to day 9) of mouse development, antigen I being the predominant teratocarcinoma-defined antigen on the embryos. There was a strong cross-reaction between mouse and human

teratocarcinomas, although the mouse-defined antigens were usually masked on the human tumor. 402AX cells could be destroyed by syngeneic T lymphocytes, and 402AX-sensitized T lymphocytes were able to damage a wide range of tumor cells sharing antigen I. Cells sensitized to the teratocarcinoma in culture were reactive in vitro against cultured embryos. Culture of tumor targets with peritoneal exudate cells resulted in a significant stimulation of cell growth, indicating a potential immune process that acts to promote rather than inhibit growth of the embryo in utero. (39 refs)

- 79-2814 Cell Surface Differentiation of Acute Lymphoblastic cALL-Type Leukemias in Diffusion Chambers.** (Eng) Jager, G. (Institut für Hamatologie der GSF, Landwehrstrasse 61, D-8000 Munich 2, W. Germany); Lau, B.; Pachmann, K.; Rodt, H.; Netzel, B.; Thiel, E.; Huhn, D.; Thierfelder, S.; Dormer, P. *Blut* 38(2): 165-168; 1979.

The peripheral blasts of three untreated patients (2 girls, 1 boy aged 5-11) with childhood acute lymphoblastic leukemia (ALL) were placed in diffusion chambers (5×10^5 cells/chamber) and implanted ip into preirradiated male CBA mice. The chambers were removed on days 6, 9, and 13, and total and differential cell counts were determined. Immunofluorescence studies were performed by labeling the cells with rhodamine-conjugated polyvalent rabbit anti-immunoglobulin (Ig) globulin and anti-T-cell globulin; anti-common ALL globulin (AcALLG) was conjugated with fluorescein. After 6 days of culture, the chamber populations dropped considerably and remained at this level throughout the culture period. Most cells maintained the morphology of leukemic blasts throughout the 13 days. Immunological characterization of the cells revealed marked surface changes. During culture, the percentage of Ig-positive cells simultaneously labeling with AcALLG increased considerably. One explanation of this finding is that most of the ALL blasts in the patients were of the immature B-cell type. Several observations support this view: (1) the morphology of most of the cells remained that of leukemic blasts; (2) growth of normal peripheral mononuclear cells in diffusion chambers does not favor the development of B cells over that of T cells; (3) prior to culturing, many cells of one of the patients carried surface Ig and common ALL antigen simultaneously; 98% of these cells were typically leukemic according to morphological criteria. It seems likely, therefore, that all three ALL's had properties of immature B cells and that the diffusion chamber system allowed these cells to mature. (12 refs)

- 79-2815 Cell-Surface Properties Growth and Malignancy of Haemopoietic Cell Lines (Meeting Abstract).** (Eng) Nilsson, K. (Wallenberg Lab., Univ. Uppsala, Uppsala, Sweden) *Br J Cancer* 39(4): 459-460; 1979 (no refs)

- 79-2816 The Histogenesis of Non-Hodgkin Lymphomas Assessed by Surface Marking (Meeting Abstract).** (Eng) Habeshaw, J. A. (ICRF Medical Oncology Unit, St. Bartholomew's Hosp., London, England); Stansfeld, A. G. *Br J Cancer* 39(4): 483; 1979 (no refs)

- 79-2817 Isolation of a Human B Lymphocyte Membrane Protein with Ia-like Properties.** (Eng) Sullivan, A. K. (Cancer Res. Unit, McGill Univ., Montreal, Quebec, Canada); Jerry, L. M.; Ikeman, R. L.; Mac-cari, R. J.; Li Thi, H.; Sylvester, C. *Can J Biochem* 57(1): 21-31; 1979.

A rapid method for isolating a major surface membrane glycoprotein with immune region-associated (Ia)-like properties from whole, unfractionated cultured human B lymphoblasts is described. The major steps include detergent solubilization, gel filtration, affinity chromatography on Sepharose concanavalin A, and then alkaline acrylamide gel electrophoresis. Specific high-titer rabbit antisera to the isolated protein reacted with cultured and normal peripheral blood B lymphocytes, as well as peritoneal macrophages from a renal dialysis patient. The antisera selectively inhibited the mixed lymphocyte reaction at high dilution. The protein reacted with a heterologous antiserum to HL-B antigens and contained subunits with a mol wt of 33,000 and 27,000, respectively. Resolution of the subunits, however, required a discontinuous sodium dodecyl sulfate gel system. These properties indicate that the protein is similar to murine Ia antigens. The protein was not associated with β_2 -microglobulin, and it showed no structural or antigenic similarity to the major RBC glycoprotein glycophorin. Antisera to the protein failed to precipitate surface-radioiodinated components from similarly treated extracts of cultured human T lymphoblasts. This method now makes available a reference membrane glycoprotein from a differentiated nucleated human cell in sufficient purity and quantity for kinetics and biosynthesis studies. (37 refs)

- 79-2818 Activated Lymphoid Cells Induce Vascularization.** (Eng) Pliskin, M. E. (Dept. Pathology, Univ. Pennsylvania, Philadelphia, PA 19104) *Transplantation* 27(2): 136-138; 1979.

The role of the lymphoid system in the development of neovascularization was investigated. The id injection of phytohemagglutinin (PHA)-treated spleen cells from normal BALB/cByJ mice into syngeneic, whole-body-irradiated recipient mice caused dose-dependent changes in vascularization. Vascularization was enhanced by the PHA-treated spleen cells at doses of 2×10^5 , 5×10^5 , and 1×10^6 ; however, vascularization was inhibited by 4×10^6 cells. Three days following injection of the mitogen-treated cells,

the reaction sites showed increased numbers of polymorphonuclear WBC. The vascular response could have resulted from vasodilation of preexisting blood vessels and/or by angiogenesis. It is postulated, however, that the macrophage in this system may induce angiogenesis as a result of its activation by lymphokines produced by the PHA-treated T cells. (20 refs)

- 79-2819 Spontaneous Human Lymphocyte-mediated Cytotoxicity against Tumor Target Cells. VII. The Effect of Immunodeficiency Disease.** (Eng) Pross, H. F. (Dept. Radiation Oncology, Queen's Univ., Kingston, Ontario K7L 2V7, Canada); Gupta, S.; Good, R. A.; Baines, M. G. *Cell Immunol* 43(1): 160-175; 1979.

Spontaneous lymphocyte-mediated cytotoxicity (SLMC) and antibody-dependent cellular cytotoxicity (ADCC) were assessed in 13 patients with immunodeficiency diseases, 1 with immunodeficiency-thymoma syndrome, 3 with Bruton type agammaglobulinemia, and 9 with common variable hypogammaglobulinemia (CVH). SLMC and ADCC functions were intact (and possibly enhanced) in the thymoma patient. Both ADCC and SLMC were detectable in the patients with X-linked agammaglobulinemia, one of whom had lower than expected SLMC. In all of the immunodeficient patients, the relative inability of B lymphocytes to produce immunoglobulin in vivo or in vitro did not consistently affect the ability of (presumably) other lymphocytes to mediate SLMC and ADCC. However, SLMC in three of the CVH patients was lower than normal. In every case, removal of Fc receptor-bearing cells from the patients' lymphocyte preparations severely depleted SLMC (and ADCC when tested), but cytotoxicity was either unchanged or enhanced by depletion of E rosette forming T cells. The effects of Fc receptor-positive cell depletion, T-cell depletion, culture serum variation, or the addition of antibody-coated erythrocytes to the assay were similar on both SLMC and ADCC effector cells (NK and K cells), regardless of whether patient or normal lymphocytes were tested. The possible significance of the results with respect to surveillance against cancer is discussed. (53 refs)

- 79-2820 The Isolation of Hybrid Cell Lines Producing Monoclonal Antibodies against the p15(E) Protein of Ecotropic Murine Leukemia Viruses.** (Eng) Nowinski, R. C. (Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104); Lostrom, M. E.; Tam, M. R.; Stone, M. R.; Burnette, W. N. *Virology* 93(1): 111-126; 1979.

Hybrid cell lines were prepared by the fusion of BALB/c mouse myeloma cells with spleen cells of C57BL/6 mice that were immunized with the AKR leukemia K36. Approx 10% of the hybrid cells produced immunoglobulins that reacted in antibody binding assays with AKR murine leukemia

virus (MuLV). Seven independent cell lines were isolated by the combined use of low-density transfer and cloning. These cells produced antiviral antibodies at a level of 3-15 $\mu\text{g}/\text{ml}$ of culture fluid. Syngeneic mice inoculated with the hybrid cells (10^6 ip) developed tumors (hybridomas) that secreted high levels of monoclonal antibodies (5-15 mg/ml) into sera or ascites fluid. Five of the hybrid cell lines produced IgM, one produced IgG₂a, and one produced IgG₂b. In high-resolution two-dimensional polyacrylamide gels, these immunoglobulins showed the limited heterogeneity in heavy and light chains that would be expected for monoclonal products. Radioimmune precipitation assays demonstrated that the monoclonal antiviral antibodies reacted with the p15(E) protein of ecotropic MuLV, but not with the p15(E) protein of xenotropic MuLV. In contrast, rabbit antiserum prepared against purified p15(E) reacted equally well with ecotropic and xenotropic MuLV. The sera or ascites fluids from hybridoma-bearing mice had antibody titers 75- to 100-fold higher than the sera from conventionally immunized mice or rabbits. Serological analysis demonstrated that the monoclonal antibodies reacted with the cell surface of virus-producing leukemia cells, but not with normal thymocytes. Furthermore, monoclonal anti-p15(E) antibodies of the IgG₂a subclass mediated lysis of the ecotropic MuLV virion in the presence of complement. The potential usefulness of monoclonal antibodies in the analysis of viral polymorphism is discussed. (32 refs)

- 79-2821 **The Evolution of Alpha-Chain Disease.** (Eng) Navab, F. (Taj Pahlavi Cancer Inst., P.O. Box 14/1154, Tehran, Iran); Mobarhan, S.; Banisadre, M.; Rambaud, J. C.; Mojtabai, A. *Gastroenterol Clin Biol* 2(12): 983-988; 1978.

Five cases of the digestive form of alpha-chain disease (α -CD) occurring in young (≤ 35 yr) Iranian Moslems, two of whom (the 2 female patients) belonged to a high socioeconomic group, are reported. Three patients presented with the usual clinical features of α -CD, chronic diarrhea being the main symptom. The fourth patient was seen at a late phase of the disease, and he had clinical and radiologic evidence of an overtly malignant intestinal tumor. He died within 4 days. The fifth patient presented with obstruction of the small intestine of 1 mo duration as the only clinical feature. A short, narrow, and ulcerated jejunal area with plasmacytic infiltration into the serosa was demonstrated microscopically; this type of lesion is unusual during the early phase of the disease. Stage A lesions were identified in two patients, both of whom responded to chemotherapy. The disease progressed to an immunoblastic sarcoma in the other two patients after a 30- and 21-mo clinical remission, respectively. One of these patients was asymptomatic, and the alpha-chain protein was no longer detectable in the serum when sarcoma was diagnosed. A complete remission was achieved by total abdominal irradiation and combination chemotherapy. (15 refs)

- 79-2822 **Increased Frequency of Gm(2) in Whites with Malignant Melanoma (Meeting Abstract).**

(Eng) Pandey, J. P. (Dept. Basic and Clinical Immunology, Medical Univ. South Carolina, Charleston, SC); Fudenberg, H. H.; Hersh, E.; Gutterman, J. *Clin Res* 27(2): 4734; 1979. (no refs)

- 79-2823 **Skin Cancer Development in Mice Exposed Chronically to Immunosuppressive Agents.**

(Eng) Daynes, R. A. (Dept. Pathology, Univ. Utah Sch. Medicine, Salt Lake City, UT, 84132); Harris, C. C.; Connor, R. J.; Eichwald, E. J. *J Natl Cancer Inst* 62(4): 1075-1081; 1979.

Experiments designed to establish the presence or absence of a relationship between the incidence of neoplasia and the chronic administration of immunosuppressive (IS) drugs are reported. Murine mammary tumor virus-negative inbred female C3Hf/HeN mice were exposed to UV light (dose rate 1.79×10^3 ergs/cm²/sec over the wavelength range 280-320 nanometers; 3 10-min exposures/wk) or benzo(a)pyrene (BP: 50 μ ; of a 0.05% soln applied topically 2x/wk) until tumor development or death. Mice in each carcinogen treatment group were also subjected to four different chronic IS regimens to determine their effect on skin cancer development. The IS agents were cyclophosphamide (30 mg/kg/wk ip), methotrexate (2.0 mg/kg 3x/wk ip), cortisone acetate (2.5 mg/wk), and heterologous antilymphocyte globulin (1.6 mg 3x/wk). IS treatment was continued until drug-related (subjective evaluation) mortality reached 10%. Because of an unexpectedly high morbidity and mortality of mice exposed to chronic IS measures, dosages were kept at a level that permitted them to survive but did not prolong allogeneic skin graft survival and lower antibody titers or diminish the proliferative responses of lymphocytes to mitogens or allogeneic lymphocytes. Nevertheless, the latency periods (time interval between beginning of medication and appearance of skin tumors) of tumors in mice exposed to IS measures were significantly shortened in several groups of mice exposed to UV + cyclophosphamide, cortisone, or antilymphocyte globulin and mice exposed to BP + cortisone. In three groups, spindle cell tumors (fibrosarcomas) shifted to squamous cell carcinomas. A suppressed immune function would not be regarded as the mechanism for the observed responses, because immunosuppression was not detected in the experimental mice. (18 refs)

- 79-2824 **Release of Soluble "Blocking" and "Suppressor" Factors from Normal Lymphocytes Treated**

with RNA from Spleens of Tumour-bearing Mice. (Eng) Pennline, K. J. (Dept. Microbiology, Schs. Medicine and Dentistry, Georgetown Univ., Washington, DC, 20007); Evans, S. B.; Nawrocki, J. F.; Rees, J. C.; Johnson, C. S.; Vallera, D. A.; Dodd, M. C. *Br J Cancer* 39(3): 247-258; 1979.

Investigations were made of the possibility of transferring "negative" or "suppressive" types of immune activity with RNA. RNA extracted from the spleens of tumor-bearing (TLRNA) and tumor-immune (ILRNA) C3H/HeJ mice was shown to transfer to normal lymphocytes (NL) the ability to produce factors that block specific tumor cell cytotoxicity and mediate specific antibody-dependent cell cytotoxicity (ADCC). Aliquots of normal C3H mouse lymphocytes were treated with TLRNA or ILRNA and cultured in vitro in the absence of tumor antigen. Supernatants were collected at 24-hr intervals and tested in a microcytotoxicity assay for blocking and ADCC activities. Factors that inhibited tumor destruction by specifically sensitized lymphocytes at the level of both the tumor cells and effector cells were demonstrable in culture supernatants of NL pretreated with TLRNA (50 or 100 $\mu\text{g}/4 \times 10^6$ cells) but not ILRNA. However, treatment of NL with either RNA resulted in the production of factors that mediated tumor-specific ADCC. Cytotoxicity testing and absorption studies of the tumor cell and a control cell indicated that factors mediating ADCC and blocking at the target cell level were specific for the tumor. Suppressor activity at the effector cell level was not absorbed by tumor cells and represents a separate and distinct mechanism of immunosuppression. These data indicate that RNA faithfully transfers "suppressive" as well as "positive" types of immune responses that have been reported previously for lymphocytes obtained directly from tumor-bearing and tumor-immune animals. (37 refs)

79-2825 Immunobiology and Therapeutic Manipulation of Heterotransplanted Nb Rat Prostate Adenocarcinoma into Congenitally Athymic (Nude) Mice. I. Hormone Dependency and Histopathology. (Eng) Drago, J. R. (Dept. Urology, Sch. Medicine, Univ. California at Davis, UCD Medical Center, 4301 X St., Suite 249, Sacramento, CA, 95817); Gershwin, M. E.; Maurer, R. E.; Ikeda, R. M.; Eckels, D. D. *J Natl Cancer Inst* 62(4): 1057-1066; 1979.

Nb rat prostate adenocarcinomas, induced by the administration of testosterone and estrogen implants, were serially studied as heterotransplants in congenitally athymic (nude) mice. This tumor model is similar to human prostate adenocarcinoma. Eight Nb rat tumors (androgen-dependent tumors 2 Pr-125, 2 Pr-128, and 2 Pr-129-D-11A; estrogen-dependent tumors 52 Pr-16 and 52 Pr-14B; and autonomous tumors 13 Pr-12, 102 Pr-22, and Pr-90) were serially transplanted. The latency period, histology, and hormone characteristics of the various tumors remained stable through 12 transplant generations. The hormone dependency pattern also remained stable. Despite the deficiency of cell-mediated immunity in the nude mouse, the rate-limiting step for successful transplantation appeared to be the hormone status of the host. Such a tumor-hormone interaction may be critical in the ability or failure to transplant specific human tumors into the nude mouse. In additional experiments, the chemotherapeutic responsiveness of heterotransplanted adenocarcinoma 13 Pr-12 was evaluated. This prostate tumor was uninfluenced by hor-

mone manipulation. The effectiveness of 5-fluorouracil, cyclophosphamide, adriamycin, and methotrexate was statistically significant, ($p < 0.003$). This combination of animal model systems proved useful in the evaluation of chemotherapeutic agents heretofore having limited use by urologic oncologists. (42 refs)

79-2826 Accessory Cell Requirements for Lymphoma Growth In Vitro and in Irradiated Mice. (Eng) Umetsu, D. T. (Dept. Pathology, New York Univ. Medical Center, 550 First Ave., New York, NY, 10016); Lerman, S. P.; Thorbecke, G. J. *Cell Immunol* 42(1): 139-154; 1979.

Accessory cell requirements for growth of the transplantable B-cell lymphoma PU-5 and a transplantable reticulum cell sarcoma (RCS) were determined in vivo and in vitro studies. The growth of PU-5 was markedly diminished in γ -irradiated compared with normal BALB/c mice. Transfer of bone marrow, but not of lymph node or peritoneal exudate cells, partially restored the ability of irradiated mice to support lymphoma growth. In vitro growth of PU-5 cells was promoted by silica-sensitive, adherent cells bearing surface immune region-associated (Ia) antigen that were present in peritoneal exudates, spleen, and lymph nodes, but not in bone marrow. Their similar action on PU-5 growth occurred only in rocking cultures. The cells did not have to be histocompatible, and they acted synergistically with 2-mercaptoethanol (2-ME) in the medium. This synergism suggested that two different stimulating effects were involved. The growth-promoting action in vitro was decreased 24 hr after γ -irradiation of the adherent cells in vitro. Growth of a transplantable RCS in SJL/J mice had previously been shown to be inhibited by prior irradiation of the host and to be restored by transfer of lymphoid cells containing a phagocytic component, but in the present studies no consistent growth-promoting effect of accessory cells on RCS's was demonstrated in vitro. Both lymphomas were stimulated by the presence of 2-ME in stationary cultures. The relationship between the in vivo and in vitro lymphoma growth-promoting activities of macrophagelike cells is analyzed. (84 refs)

79-2827 Discrepant Effects of Interferon on Murine Syngeneic Ascites Tumors and Their Solid Metastasizing Counterparts. (Eng) Ryd, W. (Inst. Pathology, Univ. Gothenburg, Gothenburg, Sweden); Hagmar, B.; Lundgren, E.; Strannegard, O. *Int J Cancer* 23(3): 397-401; 1979.

The effects of interferon (IF) on CBA and C57BL/6J mice with transplanted nonviral ascites tumors were studied. IF (12,500 units/day ip for 10 days starting the day after tumor transplant) prolonged survival time in mice inoculated ip with one of two ascites tumors and caused one of the tumors to grow in predominantly solid rather than disseminated form. There was no effect on survival time following inocula-

tion of a more virulent ascites tumor. In a second study, ascites tumors were inoculated sc, and IF was given for 11-15 days beginning with the onset of metastasis. When ascites tumors are transplanted sc, they grow as solid tumors. The mean tumor wts were higher in all IF-treated mice compared with untreated controls, and mean survival times were reduced. In mice receiving one of the less virulent tumors, the tumor-enhancing effect was highly significant and there was a highly significant increase in the development of lung metastases. The incidence and distribution of metastases were not affected by IF in the mice receiving the other two tumors. Animal models for analyzing IF effects should be developed before IF treatment is initiated in human cancer patients. (32 refs)

79-2828 Cell Subpopulations of a Cultured Human Tumor Differing by Rate of Growth in Vivo as Heterotransplants in Nude Mice and by Modal Chromosome Numbers (Meeting Abstract). (Eng) Giovanella, B. C. (Can-

cer Res. Lab., St. Joseph Hosp., Houston, TX, 77002); Yim, S. O.; Stehlin, J. S. *In Vitro* 15(3): 197; 1979. (no refs)

See also:

*(Rev.): 79-2468, 79-2469, 79-2470.

*(Chem.): 79-2478, 79-2511, 79-2551, 79-2554, 79-2555, 79-2557, 79-2597, 79-2633, 79-2641, 79-2643, 79-2653.

*(Viral): 79-2695, 79-2715, 79-2719, 79-2724, 79-2725, 79-2726, 79-2728, 79-2730, 79-2732, 79-2735, 79-2738, 79-2739, 79-2747, 79-2748, 79-2752, 79-2764, 79-2765, 79-2766, 79-2783, 79-2791, 79-2795, 79-2796, 79-2808.

*(Path.): 79-2829, 79-2839, 79-2840, 79-2860, 79-2861, 79-2883.

*(Epid-Biom.): 79-2905, 79-2924, 79-2925, 79-2926.

PATHOGENESIS

79-2829 Genetic Mechanisms in Cancer Predisposition. Report of a Cancer Family. (Eng) Meisner, L. F. (State Lab. Hygiene, 465 Henry Mall, Madison, WI, 53706); Gilbert, E.; Ris, H. W.; Haverty, G. *Cancer* 43(2): 679-689; 1979.

A lower middle class black family is described in which four siblings developed cancer affecting different organs: lymphoma, meningeal sarcoma, osteogenic sarcoma, and adenocarcinoma of the cecum (proband). Antibody titers to Epstein-Barr virus were elevated in all family members except the proband. All family members had normal chromosome constitutions, but a specific fragile site leading to breakage of the long arm of chromosome 16 was found in the father, proband, and two normal sibs. Chromosome preparations from skin biopsies of five unaffected family members demonstrated excessive polyploidy due to endoreduplication that was probably caused by mycoplasma. There was only one other case of cancer in previous generations of this family. There may be at least three nonlinked genetic loci associated with cancer susceptibility that in certain combinations can give rise to the cancer family syndrome. By themselves, these loci would not be significant relative to cancer predisposition. The first locus could be a common gene akin to an oncogene or a provirus. The second could be a germinal or somatic regulatory gene, and the third could be a gene affecting immunocompetence. The dominant oncogene would make for cancer susceptibility, whereas the controlling genes of the other two loci could be associated with cancer resistance, since two mutations would then be required for malignant development. This model would also explain the distribution of cancer susceptibility and resistance in the general population. (57 refs)

79-2830 Chromosome Aberrations of Myeloid and Lymphoid Cells in Cancer Patients and Family Members without Evidence of Cancer. (Eng) Wurster-Hill, D. H. (Dept. Pathology, Dartmouth Medical Sch., Hanover, NH); Cornwell, G. G.; McIntyre, O. R. *Cancer Detect Prev* 2(1): 125-146; 1979.

Studies of chromosome (CS) aberrations of myeloid and lymphoid cells in cancer patients and family members without evidence of cancer are reviewed. CS banding techniques permit the identification of each CS individually and thus facilitate accurate analyses of CS abnormalities. Banding studies to date have clearly demonstrated a nonrandom involvement of certain CS's in the formation of abnormal cell lines in individuals with hematological disorders. Thirteen different

CS's participate in this phenomenon, which is known as clustering. They are CS's 1, 7-9, 14, 17-22, X, and Y, and the disorders with which they are associated include myeloproliferative disorders, myelomas, lymphomas, acute myelogenous leukemia, chronic myelogenous leukemia, anemias, ataxia-telangiectasia, plasma cell leukemia, and non-Hodgkin's lymphoma. Clinically normal relatives of cancer patients with cancer and cancer-prone conditions may also have abnormal clones or CS instability. Two families in which there was increasing CS instability and mitotic activity in the unstimulated blood cultures of four clinically normal members are described. These phenomena indicate the presence of a dynamic process that might be envisioned as one or more chromosomally normal inheritable defects in DNA repair or CS replication that are especially responsive to the detrimental effects of environmental mutagens. Mutagenic stimulus would then promote the occurrence of random or semirandom errors, some of which would be reflected in CS abnormalities. The latter might produce a clone with a constitution compatible with the development of neoplasia. Clustering may reflect the receptor sites for mutagenic agents and/or the genetic loci for the growth control and differentiation of specific cell types that become malignant, or it may result from an altered milieu permissive of clone development and survival and associated with particular etiologic agents. (91 refs)

79-2831 Hodgkin's Disease in Siblings. (Ger) Theiss, W. (Hamatologie und Onkologie, Klinikum rechts der Isar der Techn. Universität, Ismaninger Strasse 22, D-8000 Munich 80, W. Germany); Sauer, E.; Rastetter, J. *Munch Med Wochenschr* 121(9): 309-312; 1979.

The occurrence of histologically diagnosed Hodgkin's disease (HD) in a pair of brothers and a pair of sisters is reported, and similar cases in the literature are reviewed. The first patient was found to have lymphocyte-rich HD in 1946, at age 8; his brother was diagnosed with nodular-sclerotic HD Stage IV in 1973, at age 21. Two other brothers, the parents, and other close relatives had no history of tumors or systemic diseases. Nonclassifiable HD Stage II was found in a 22-yr-old girl in 1970. Her sister developed nodular-sclerotic HD in 1977, at age 33; from 1972-1975, she had tuberculosis of the submandibular nodes. No tumors or systemic disease were found in a brother and sister of this pair, their parents, and other relatives. A literature search showed an incidence of 13 sibling pairs among 3,332 cases of HD; this frequency of siblings is higher than that expected from a random incidence ($p < 0.001$). The findings of sex concordance in 12/13

pairs is also significant ($p < 0.01$). Further pairs found in isolated case reports bring the total to 32 pairs of brothers, 12 pairs of sisters, and 17 sex-discordant pairs. Thus, the increased risk of HD for same-sex siblings is calculated to be 2.5 times that for discordant siblings. There is an approx sevenfold increased risk of the disease for siblings of HD patients (assuming diagnosis is made before either is 45 yr old). It appears that HD has a tendency to be familial; the contribution of genetic and environmental factors cannot yet be evaluated. (51 refs)

79-2832 Genetic Mosaicism and In Vivo Analyses of Neoplasia and Differentiation. (Eng) Mintz, B. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA, 19111). *30th Ann Symp Cancer Res* 27-53; 1978.

Two experimental approaches were employed to study the developmental nature of neoplastic disease: (1) strain-specific malignancies in mice were used to obtain allophenic individuals in which tumor-susceptible and nonsusceptible cell strains coexist throughout life and (2) frankly malignant stem cells were brought into contact with normal, early stem cells in young embryos. Genotypic analyses in allophenic animals revealed that tumor susceptibility was largely localized to the cells of the potentially malignant tissue. As a result of transplant experiments with genetically mosaic nodules, a hypothesis of early mammary tumorigenesis in ordinary (single genotype) animals was advanced. It was hypothesized that normal cells may usually be closely associated with transformed cells at first and may be indispensable for the ultimate capacity of the latter to grow and proliferate independently. A similar supportive role of associated normal cells may characterize the early stages of other malignancies. Multiclonality in hepatomas, and possibly in some other kinds of tumors, may be more common than analyses of tumors from allophenic mice have shown. Experiments in which teratocarcinoma cells were normalized after they were injected into blastocysts are described. The experimental use of genotypic cellular mosaicism in early embryos furnished the first unequivocal case of complete and stable reversal of an animal malignancy to normalcy. The initial conversion to malignancy in teratocarcinomas may come about through induced changes in gene function, rather than gene structure, because of an anomalous environment. Both mutational and nonmutational classes of cancers probably exist: many tumors may be caused by somatic mutation, whereas nonmutational cancers may result from physiological changes in the cell or tissue milieu. Cells may lose their transformed phenotypes if an oncogenic virus is lost or if the cellular chromosome constitution changes in specific ways. Regarding the possible developmental totipotentiality of malignant teratocarcinoma cells, the first instance of mammalian organismic differentiation from preselected mutant cells by a "parasexual" route was demonstrated. (76 refs)

79-2833 Acute Lymphoblastic Leukemia Accompanied by an Eosinophilia of the Peripheral Blood and

the Meninges (Letter to Editor). (Fre) Youinou, P. (Hopital St-Louis, 2, place du Dr. A. Fournier, F 75475 Paris Cedex 10, France); Andrieu, J. M.; Casassus, P. *Nouv Presse Med* 8(7): 527; 1979.

The case is presented of an 8-yr-old child who had a high blood eosinophil count ($10,400/\text{mm}^3$) when leukemia was initially diagnosed. After 16 mo of chemotherapy, eosinophils appeared in the spinal fluid ($140/\text{mm}^3$). The patient died 41 mo after diagnosis with high eosinophil counts in both blood and spinal fluid. (5 refs)

79-2834 A Case of Preleukemia--Reconstitution of Normal Marrow Function after Bone Marrow Transplantation (BMT) from Identical Twin. (Eng) Bhaduri, S. (Dept. Internal Medicine and Pediatrics, Div. Haematology, Univ. Ulm, Steinhovelstrasse 9, D-7900 Ulm/Donau, W. Germany); Kubanek, B.; Heit, W.; Pflieger, H.; Kurrle, E.; Fliedner, T. M.; Heimpel, H. *Blut* 38(2): 145-149; 1979.

The reconstitution of normal bone marrow (BM) function in a 30-yr-old man with preleukemia who received a bone marrow transplantation (BMT) from his identical twin brother is reported. A tentative diagnosis of preleukemia had been made 4 yr previously on the basis of a severe sideroblastic anemia, micromegakaryocytes in the BM, and granulocytopenia. Biopsy 4 yr later showed a hypercellular BM consisting mostly of normoblasts and proerythroblasts, hyperplasia of megakaryocytes, and a complete absence of myelopoiesis. Repeated cytogenetic analysis of the BM revealed that 75% of all metaphases had an extra C-group chromosome. The decision for a BMT was made because of progression of the disease and the danger presented by repeated life threatening infections. The patient received 2.14×10^8 cells/kg body wt. BM biopsies taken at weekly intervals after the BMT showed a steadily increasing cellularity of the BM with a nearly normal picture after 6 wk. Cytogenetic analysis of the BM 3 wk after transplantation revealed a normal karyotype. BM cell count and colony-forming units showed near normal values after 6 wk. (5 refs)

79-2835 Acute Leukemia Complicating Hodgkin's Disease. Five New Cases. (Fre) Jouet, J. P. (Service des Maladies du Sang, Hopital A. Calmette, C.H.U. de Lille, F 59000 Lille, France); Huart, J. J.; Bauters, F.; Goudemand, M. *Nouv Presse Med* 8(8): 613-614; 1979.

Acute nonlymphocytic leukemia was diagnosed in 3 men and 2 women 32-61 mo (av 4 yr) after the diagnosis of Hodgkin's disease. All patients had received polychemotherapy with nitrogen mustard, vincristine, procarbazine, and prednisone for Hodgkin's disease. Two patients were also irradiated. (4 refs)

79-2836 Malignant Lymphomas: Models of Differentiation and Cooperation of Lymphoreticular Cells.

(Eng) Lennert, K. (Dept. General Pathology and Pathologic Anatomy, Univ. Kiel, D-2300 Kiel, W. Germany); Kaiserling, E.; Muller-Hermelink, H. K. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book B), 994 pp.; 897-913; 1978.

Methods of identifying various types of structural cells in normal lymphatic tissue are described, and evidence indicating that specific reticulum cells (RC) are associated with the formation of T and B areas during ontogeny is presented. Four types of RC can be identified in normal human lymphatic tissue by ultrastructural and cytochemical criteria: dendritic (B-cell regions only), interdigitating (T-cell regions only), histiocytic (T- and B-cell regions), and fibroblastic (predominantly at borders of T-cell regions). The various types of RC can also be easily differentiated by enzyme cytochemistry, as characteristic patterns are found for several surface and intracytoplasmic enzymes. The different types of RC are recognizable by light microscopy only with great difficulty, if at all. A characteristic pattern of dendritic or interdigitating RC is found in some low-grade malignant non-Hodgkin's lymphomas of B- or T-cell origin, respectively. Centroblastic/centrocytic lymphoma consists of all cells found in reactive germinal centers, including dendritic RC. Centrocytic lymphoma, another germinal-center neoplasm, also contains dendritic RC, but other B-cell lymphomas are devoid of dendritic RC. T-zone lymphoma contains all elements of T-cell areas, including interdigitating RC. Interdigitating RC are also found in mycosis fungoides, Sezary's syndrome, and T-cell-type chronic lymphocytic leukemia. High-grade malignant lymphomas of B- or T-cell origin usually do not reveal specific RC. Thus, a characteristic pattern of RC reflects the degree of differentiation of a non-Hodgkin's lymphoma. The findings in Hodgkin's disease suggest that it may also be subclassified into at least two biologically different types. The nodular variant with lymphocytic predominance (nodular paraneoplasia) is the neoplastic counterpart of progressively transformed germinal centers, whereas the nodular sclerosing and mixed types of Hodgkin's lymphoma develop in and involve mainly T regions. This difference is reflected by different patterns of specific RC. The findings in malignant lymphomas support the microecologic view of lymphatic tissue. (36 refs)

79-2837 No Maternal Effect in Childhood Leukaemia with Neurofibromatosis (Letter to Editor). (Eng) Bader, J. L. (Clinical Epidemiology Branch, NCI, Bethesda, MD, 20014); Miller, R. W. *Lancet* 1(8114): 503; 1979.

Lineal transmission of neurofibromatosis (NF) in 22 children with the nonlymphoblastic form of leukemia (NLL) was examined. Although maternal transmission occurred in 13 patients (vs paternal in 5 and spontaneous mutation in 4), the excess was not significantly greater than the expected maternal:paternal transmission ratio of 1.82:1. Thus, although maternal (intrauterine) factors apparently exacerbate non-hematological manifestations of NF, different factors probably enhance the induction of NLL. (7 refs)

79-2838 Chromosome Studies of Two Transplantable Leukemias of BN Mice. (Eng) Bregula, U. (Dept. Tumor Biology, Inst. Oncology, 00-973 Warsaw, Poland); Wlodarska, I.; Wezyk, J. *J Natl Cancer Inst* 62(4): 1051-1056; 1979.

Karyotypes of two transplantable murine ascites leukemias, LBN/a2 and LBN/b3, were studied with the use of the trypsin-Giemsa technique. The original tumors arose in adult female mice of strains BN/a and BN/b after prolonged antilymphocyte globulin administration. The karyotypes of both leukemias showed similar patterns. Both were hyperdiploid with modal chromosome numbers of 41 and 42 in LBN/a2 and LBN/b3, respectively. The cells consisted of an average of 37 normal chromosomes and 4-5 abnormal chromosomes. The most consistent karyotype deviation was monosomy of the X-chromosome and of several autosomes: #9, 14, and 7 in the LBN/a2 line and #7, 12, 14, and 9 in the LBN/b3 line. In most LBN/b3 cells and in a lower proportion of LBN/a2 cells, trisomy of chromosome #15 was found. With regard to the occurrence of abnormal marker chromosomes, both tumors exhibited great cell-to-cell variation. Two of the markers were common to both leukemia lines. (23 refs)

79-2839 Human Myeloproliferative Disorders: Clonal Origin in Pluripotent Stem Cells. (Eng) Fialkow, P. J. (Medical Genetics Section, Medical Service, Veterans Admin. Hosp., Seattle, WA, 98108); Denman, A. M.; Singer, J.; Jacobson, R. J.; Lowenthal, M. N. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book A), 528 pp.; pp. 131-144; 1978.

Application of the glucose-6-phosphate dehydrogenase marker system to three myeloproliferative disorders, chronic myelocytic leukemia (CML), polycythemia vera, and idiopathic myeloid metaplasia, showed that each disease involved pluripotent stem cells. Furthermore, the disorders had a clonal origin at the time of diagnosis. The data do not support hypotheses of an origin from the proliferation of normal stem cells; they are much more compatible with a neoplastic origin. The clinical manifestations of each disease are quite different. It is not known whether these manifestations differ because there are different kinds of genetic alterations governing the response of proliferating cells to regulatory factors and/or because different levels of stem cells are involved in each disease. Few normal uncommitted stem cells have been found in CML, but the presence of these cells has been documented in polycythemia vera. Lymphocytes with B-cell characteristics arise from the abnormal CML stem cell. There are two other populations of lymphocytes in patients with CML, and both have T-cell characteristics. One population of T cells may arise from the abnormal CML stem cell, but the other population definitely does not. Marrow fibrosis is the predominant feature in idiopathic myeloid metaplasia and is a not-infrequent occurrence in CML and polycythemia vera. Data suggest that marrow fibrosis is a secondary event

and not an integral part of the abnormal clonal proliferation. (41 refs)

- 79-2840 Adult Lymphoid Neoplasias of T- and Null-Cell Types.** (Eng) Koziner, B. (Memorial Sloan-Kettering Cancer Center, New York, NY, 10021); Mertelsmann, R.; Filipa, D. H.; Good, R. A.; Clarkson, B. D. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book B), 994 pp.; pp. 843-857; 1978.

The clinical, morphological, cytochemical, and cell-marker features of T- and null-cell adult lymphoid neoplasias are presented. Twenty-six patients with acute lymphoblastic leukemia (ALL) and 18 patients with leukemic malignant lymphoma of the diffuse, poorly differentiated, lymphocytic (DPDL) lymphoblastic variety were characterized. There were several similarities between ALL and the DPDL lymphoblastic type in the leukemic phase, which suggested common cell lineages with variable expression in different tissues. Clinically, the DPDL patients had a higher incidence of initial mediastinal and CNS involvement than did the ALL patients. Both entities comprised cases with a predominance of either null or sheep RBC-rosetting cells at 4 and 37 C. There were 8 and 10 patients, respectively, in the T and null-cell DPDL groups, 4 patients had T-cell ALL, and 22 patients had null-cell ALL. High levels of terminal deoxynucleotidyl transferase (TdT) activity were detected in the bone marrow and peripheral blood of all 30 patients studied. A statistically significant relationship was found between the presence of nuclear convolutions and the T-cell type of leukemic disorder. Mediastinal involvement was observed in 9/17 cases with and 9/27 cases without convoluted nuclei. No difference in TdT activity was detected between the two groups. A positive reaction for acid phosphatase in the paranuclear zone was observed in association with nuclear convolutions and rosetting capacity. Analysis of patients studied within the last 2 yr and receiving intensive chemotherapy showed no significant differences in survival between the DPDL and ALL patients, between cases with a predominance of T vs null cells, or between cases with a convoluted vs nonconvoluted nuclear morphology. These studies suggest that the presence of high levels of TdT activity, sheep RBC-rosetting capacity, a convoluted nucleus, and strong acid phosphatase content in the paranuclear area probably denote properties of neoplastic cells of thymic lineage. The variable expression of these markers might represent proliferation at different levels of differentiation and/or phases of the cell cycle. (25 refs)

- 79-2841 The Ultrastructure of Liposarcoma. A Study of 10 Cases.** (Eng) Kindblom, L. G. (Dept. Pathology, Sahlgren's Hosp., Univ. Goteborg, Goteborg, Sweden); Save-Soderbergh, J. *Acta Pathol Microbiol Scand [A]* 87(2): 109-121; 1979.

An ultrastructural study of 10 human liposarcomas is report-

ed. Four of the liposarcomas were well-differentiated, lipomalike, and/or fibrosing; three were myxoid tumors; two were round cell tumors, and one was a pleomorphic tumor. The well-differentiated, lipomalike liposarcomas contained cells with a few large lipid droplets, few organelles and a peripherally located, fairly large nucleus. The well-differentiated, fibrosing liposarcomas contained mostly spindle-shaped, fibroblastlike cells, with abundant rough endoplasmic reticulum and inconspicuous lipid inclusions, surrounded by collagen. One well-differentiated liposarcoma had an area that was similar to brown adipose tissue and hibernoma. The spindle and stellate cells of the myxoid liposarcomas showed abundant rough endoplasmic reticulum and large smooth-membraned vacuoles filled with moderately dense amorphous material, which appeared to be extruded extracellularly by rupture of the vacuoles. Cytoplasmic lipid droplets were seen in most cells, but they were much less prominent than in the well-differentiated lipomalike liposarcomas. Ultrastructurally, there were many similarities between the myxoid and round cell liposarcomas, indicating a close relationship between the two types. The pleomorphic liposarcoma revealed cells with one or more large, irregular nuclei, numerous large vacuoles from dissolved lipids, abundant dilated cisternae of rough endoplasmic reticulum, and round, electron-dense bodies corresponding to PAS-positive hyalin globules seen in the light microscope. It is suggested that the variegated cellular appearance of the different liposarcoma subtypes reflects the wide cellular spectrum seen during the differentiation of adipose tissue and that, histogenetically, all liposarcomas represent a single entity. (15 refs)

- 79-2842 Myxoid Variant of Malignant Fibrous Histiocytoma. Ultrastructural Observations.** (Eng) Lagace, R. (Departement de Pathologie, Universite Laval, Cite Universitaire, Quebec, Canada G1K 7P4); Delage, C.; Seemayer, T. A. *Cancer* 43(2): 526-534; 1979.

Ultrastructural findings in four cases of the myxoid variant of malignant fibrous histiocytoma (MFH) are presented. Light microscopy demonstrated a pseudocapsule composed of moderately vascularized dense connective tissue at the margin of all neoplasms. Two different growth patterns were apparent: in one, a myxoid, poor cellular stroma was dominant; in the other, the tumor was more cellular and demonstrated a storiform pattern. Abundant stromal mucopolysaccharides were present in the myxoid stroma. Electron microscopy (EM) demonstrated four principal cell types in the myxomatous portions of the neoplasms: a primitive mesenchymal cell, spindle cells of a fibroblastic and histiocytic nature, and multinucleated giant cells. It is unlikely that EM could be consistently decisive in differentiating myxoid MFH from other myxomatous lesions of mesenchymal soft tissue, since they share considerable structural similarities. The observations in this study support the concept that fibroblasts and histiocytes arise from the same mesenchymal stem cell. (22 refs)

- 79-2843 Case Report: Association of Giant Cell Arteritis and Pituitary Tumor. Report of Two Cases.** (Eng) Papaioannou, C. C. (Dept. Internal Medicine, Div. Rheumatology, Mayo Clinic, Rochester, MN, 55901); Trautmann, J. C.; Kazmier, F. J.; Hunder, G. G. *Am J Med Sci* 277(1): 85-90; 1979.

The cases of two patients with giant cell arteritis and a concomitant pituitary tumor are reported. The first patient, a 63-yr-old man, presented with a history of headache, fever, and visual blurring. The second patient, a 69-yr-old woman, presented with scalp tenderness, ear pain, headaches, and visual blurring. They were diagnosed as having giant cell arteritis. However, skull roentgenograms revealed an enlarged sella turcica in both patients, a finding that was confirmed by tomograms of the sella. Arteriograms showed the presence of vascular pituitary tumors that were removed transphenoidally. The tumors were chromophobe adenomas, the most common pituitary tumor. Symptoms include orbital headaches, visual disturbance and visual field loss, and hypo- or hyperpituitarism. These two cases illustrate the importance of a careful neuroophthalmologic examination and roentgenograms of the head in patients with giant cell arteritis who have visual field loss. (25 refs)

- 79-2844 Familial Thyroid Adenomata (Meeting Abstract).** (Eng) Schiffrin, A. (McGill Univ., Montreal, Canada); Guyda, H.; Hughes, I.; Winter, J. *Pediatr Res* 13(4, part 2): 385; 1979. (no refs)

- 79-2845 C-Cell Hyperplasia and Medullary Thyroid Carcinoma in the Rat. An Immunohistochemical and Ultrastructural Analysis.** (Eng) DeLellis, R. A. (Dept. Pathology, Tufts Univ. Sch. Medicine, Boston, MA, 02111); Nunnemacher, G.; Bitman, W. R.; Gagel, R. F.; Tashjian, A. H.; Blount, M.; Wolfe, H. J. *Lab Invest* 40(2): 140-154; 1979.

The histogenesis of medullary thyroid carcinoma (MTC) in the rat was defined, the relationships between the development of this tumor and preexisting C-cell abnormalities at various ages were determined, and the suitability of the rat as a model for human familial MTC was evaluated. MTC is a distinctive neoplasm that is derived from the calcitonin (CT)-producing intrathyroidal C-cell system and develops commonly in untreated rats of various strains. Thyroid glands of Long-Evans rats aged 3 mo-3 yr showed a spectrum of C-cell proliferative abnormalities. Compared with 3-mo-old control rats, thyroids from 9- to 12-mo-old animals exhibited mild, diffuse, C-cell hyperplasia (CCH). Thyroids from animals 1-3 yr of age exhibited progressively more severe C-cell abnormalities, including severe diffuse CCH, nodular CCH, and/or MTC. In contrast to the normal basal serum CT levels in controls and in animals with mild diffuse CCH, animals with severe diffuse CCH, nodular CCH, or

MTC had elevated basal serum CT values. Nodular CCH was characterized by the replacement and enlargement of individual follicles by C cells. Larger foci of nodular CCH were characterized by similar changes in multiple adjacent follicles or by an irregular expansion of individual follicles. MTC was characterized by penetration of the follicular basal lamina by C cells with extension into the adjacent thyroid stroma. In addition to the high incidence of thyroïdal C-cell abnormalities, diffuse and/or nodular parathyroid hyperplasia was common. There was no evidence of chronic renal failure in these animals, and the serum Ca levels were within normal limits. Although the stimulus for the initial C-cell proliferation remains unknown, the appearance of MTC is preceded by relatively prolonged phases of CCH. These findings are essentially identical with those noted in human familial MTC, and they indicate that the rat provides a useful model system for studying the regulation of C-cell proliferation during neoplastic development and progression. (34 refs)

- 79-2846 Multiple Keratoacanthoma.** (Ger) Bonniger, F. (Dermatologische Klinik, Ludwig-Maximilians-Universität München, Frauenlobstrasse 9, D-8000 München 2, W. Germany); Burg, G. *Hautarzt* 30(2): 92-94; 1979.

A 59-yr-old man with a 5-yr history of spontaneously healing multiple keratoacanthomas (MKA) of the head and neck is described. There were no cases of MKA in his family. He had been exposed to tear gas occupationally for 2 yr, 27-28 yr before the onset of the disease. The etiology of MKA is unknown: tar, oil, intensive exposure to sunlight, trauma, and viruses have been reported to be etiological factors. It is not known if the tear gas was an etiological factor in this case. Clinically and histologically, MKA are similar to solitary keratoacanthomas. Transformation of these lesions into squamous cell carcinoma is possible. (27 refs)

- 79-2847 Multiple Tumors Arising in Nevus Sebaceus.** (Eng) Lillis, P. J. (Dept. Dermatology, Univ. Iowa Hosps. and Clinics, Iowa City, IA, 52242); Ceille, R. I. *Cutis* 23(3): 310-314; 1979.

The case of a 43-yr-old woman who had three distinct neoplasms and a nevocellular nevus in a single plaque of nevus sebaceus is presented. The tumors had grown out of a birthmark, a large, brown, hairless spot, on her scalp. The neoplasms were an adenoidal basal cell epithelioma, a pigmented basal cell epithelioma, and syringocystadenoma papilliferum. The multitude of lesions in this patient adds support to the concept of nevus sebaceus arising from a pluripotential primary epithelial germ cell. This case illustrates the importance of close follow-up of nevus sebaceus. (11 refs)

- 79-2848 Ultrastructural and Cytochemical Studies on the Pathogenesis of Primary and Transplanted**

Experimental Brain Tumors in Mice (Meeting Abstract). (Eng) Gregory, T. F. (State Univ. New York at Buffalo, Buffalo, NY). *Diss Abstr Int [B]* 39(9): 4143; 1979. (no refs)

79-2849 Epidermal Hemangioendothelioma. (Rus) Sych, L. I. (No affiliation given); Kalamkaryan, A. A. *Vestn Dermatol Venerol* (2): 27-31; 1979.

A rare temporoparietal hemangioendothelioma occurred in a 67-yr-old man shortly after he had suffered a blow to the site. Abnormal signs were noted within 2-3 days of the trauma. Clinically, the lesion was a red-purple, irregularly shaped tumor with defined borders and ulceration in the center. The dermis in the peripheral zone of the tumor contained numerous vascular cavities lined with large cells that had polymorphic hyperchromatic nuclei. (13 refs)

79-2850 Primary Rhabdomyosarcoma of the Cerebrum. An Ultrastructural Study. (Eng) Yagishita, S. (Div. Pathology, Kanagawa Rehabilitation Center, Nanasawa, Atsugi-Shi, Kanagawa, Japan); Itoh, Y.; Chiba, Y.; Fujino, H. *Acta Neuropathol (Berl)* 45(2): 111-115; 1979.

A case of primary cerebral rhabdomyosarcoma in a 51-yr-old woman is reported. The greater part of a subdural, soft gray, tumor located in the right cerebral hemisphere and attached to the midportion of the falx was removed surgically. A recurrent tumor was removed from the same site < 1 yr later. The two surgical specimens were essentially identical histologically, except that the recurrent tumor was more dedifferentiated than the original one. The tumors were polymorphic, but they were composed mainly of poorly differentiated cells interpreted as rhabdomyoblasts without definite cross-striation. Electron microscopy revealed that these cells were rhabdomyosarcomatous cells with strongly acidophilic perikarya and myofibrillary structures in their cytoplasm. The tumors were not teratomatous, as elements other than myoblastic cells were absent. The rhabdomyosarcoma in this case probably arose from intracranial mesenchymal tissue present in the meninges. (27 refs)

79-2851 Progression of Precancerous Lesions. Development of an Experimental Study System in the Hamster Cheek Pouch (Meeting Abstract). (Eng) Ferguson, J. W. (Dept. Dental Sciences, Univ. Otago, Dunedin, New Zealand). *J Dent Res* 58(Special C): 1214; 1979. (no refs)

79-2852 Symptomatic Epilepsy in Cerebral Metastases. (Ger) Neundorfer, B. (Neurologische Klinik, Universitat Heidelberg, Theodor-Kutzer-Ufer, D-6800 Mannheim 1, W. Germany); Meyer-Wahl, L.; Meyer, J. G. *Munch Med Wochenschr* 121(12): 431-432; 1979.

Causes of epilepsy were analyzed in a series of 411 patients in whom the first attack appeared at > 25 yr of age. Cerebral metastases were identified as the cause of epilepsy in 8/441 patients; they included 3 men and 5 women, av age 57.4 yr (the av age of the entire series was 45 yr). The primary tumor was identified as a bronchial carcinoma in 3 patients, breast carcinoma in 2, melanoma in 1, carcinoma of the sigmoid colon in 1, and carcinoma of the urinary bladder in 1. The metastases were localized to the cerebroparietal region in 7 patients and to the occipital region in 1. (14 refs)

79-2853 Familial Brain Tumour (Letter to Editor). (Eng) Thuwe, I. (Psychiatric Res. Centre, Univ. Goteborg, St. Jorgen's Hosp., S-42203 Hisings Backa, Sweden); Lundstrom, B.; Walinder, J. *Lancet* 1(8114): 504; 1979.

Six confirmed cases and one suspected case of primary cerebral tumor were found in a single family on a Swedish coastal island. There was consanguinity between the parents in only one of the patients, but for every patient the families of both parents could be traced back at least to the 18th century to the same isolated country district. A high proportion of other family members had epilepsy, and several had pathological electroencephalograms, in some cases with focal signs. (no refs)

79-2854 Metastatic Breast Carcinoma in an Intracranial Meningioma. (Fre) Di Bonito, L. (Istituto d'Anatomie et Histologie Pathologique, Ospedale Maggiore, v. Stuparich 1, 34125 Trieste, Italy); Bianchi, C. *Sem Hop Paris* 55(3/4): 171-172; 1979.

A meningioma containing metastatic breast carcinoma tissue was discovered in the left frontal lobe of a 68-yr-old woman who died of progressive breast cancer. Literature studies suggest that the coexistence of meningioma and breast carcinoma is not mere chance. (6 refs)

79-2855 Giant Central Ossifying Fibroma of the Mandible: Report of Case. (Eng) Carlisle, J. E. (Oral and Maxillofacial Surgery Program, Medical Coll. Georgia Sch. Dentistry, Augusta, GA); Hammer, W. B. *J Oral Surg* 37(3): 206-211; 1979.

The occurrence of central ossifying fibroma of the mandible in a 59-yr-old black woman is reported. Central ossifying fibroma is a slowly progressing lesion that is generally asymptomatic until swelling or deformity occurs. Large lesions may outgrow their blood supply and become infected. No hereditary predisposition has been described for this tumor, and its pathogenesis is unclear. The present patient presented with a 1.5-yr history of a progressively enlarging mass that involved the right side of the mandible. The lesion was diag-

nosed as fibrous dysplasia and was treated by partial incision and recontouring. The lesion recurred with signs of persistent infection that could not be controlled by conservative treatment. The diagnosis was changed to giant ossifying fibroma. Surgical resection of the tumor and involved bone was performed, and follow-up examinations have shown no signs of recurrence. There was some evidence of bone regeneration. In general, the prognosis for this tumor is excellent and recurrence after proper treatment is rare. (9 refs)

- 79-2856 Palatal Metastases in Renal Cell Carcinoma.** (Eng) Susan, L. P. (Al-Mashtal, New Baghdad, 20A/23/5, Baghdad, Iraq); Daughtry, J. D.; Stewart, B. H.; Straffon, R. A. *Urology* 13(3): 304-305; 1979.

Two cases of renal cell carcinoma in which the presenting condition was a palatal tumor are reported. The spread of the tumors to the palate was likely due to communication between the vertebral veins and the greater venous plexus of the head. (9 refs)

- 79-2857 Cementoblastoma: Report of Case.** (Eng) Farman, A. G. (Sect. Oral Biology, Dental Coll., Univ. Riyadh, PO Box 5967, Riyadh, Saudi Arabia); Kohler, W. W.; Nortje, C. J.; Van Wyk, C. W. *J Oral Surg* 37(3): 198-203; 1979.

Fifty-five cases of cementoblastoma appearing in the literature and a new case involving a 19-yr-old man are reviewed. Most of the large and expanding lesions were found in persons < 20 yr old, those found in older individuals being generally smaller. Mandibular lesions were greater than threefold more common than maxillary lesions. The most common sign was swelling of the affected area, and the most frequent symptom was low-grade pain, possibly a sequela to concurrent caries. All of the lesions were round, dense, radiopaque masses fused to one or more teeth; the roots of the affected teeth were resorbed to varying degrees. In the present case, the tooth and attached tumor were totally enucleated under general anesthesia. Radiographic and visual inspection of the specimen showed a distinct cartwheel pattern of calcific spokes radiating from the center of the lesion. Postoperative healing was uncomplicated, and there was no evidence of recurrence during a 2-yr follow-up period. There has been no report of a malignant cementoblastoma or of malignant transformation in a benign cementoblastoma. (36 refs)

- 79-2858 Basal Cell Carcinoma in a Hair Transplant Recipient Site.** (Eng) White, J. W. (Dept. Dermatology, Wilford Hall USAF Medical Center, Lackland AFB, TX, 78236). *Cutis* 23(3): 322-325; 1979.

A basal cell carcinoma (BCC) developed in the central scalp

of a 41-yr-old man at the site of a hair transplant plug placed 5 yr previously. The patient presented with a gradually enlarging, painful ulcer of the scalp that had begun 6 mo previously as a pustule with an erythematous border. Histological examination revealed an infiltrating ulcerated BCC in actinic damaged skin contiguous to fibrosis consistent with scar tissue. A foreign body granuloma containing doubly refractile linear particles was noted in the upper dermis lateral to the BCC. The patient had brown hair and eyes, no freckles, and described himself as one who sunburns easily. The transplanted skin (protected by the hair of the occiput for 36 yr) and the intervening scar were exposed to sunlight for only 5 yr. The adjacent scalp was exposed for at least 14 yr. The exposure occurred in a geographic area known for intense exposure occurred in a geographic area known for intense sunlight. Because there was normal skin between the granuloma and the BCC, the former was probably incidental. This case does not answer the question of trauma as the cause of BCC. It is, however, the only skin carcinoma in a person without evidence of past or present premalignant or other malignant skin lesions. It occurred in skin or scar exposed to the sun for only 5 yr, which was probably not enough time to have been the sole cause of cancer. Thus, it is possible that there was a causal relationship between the hair transplantation and BCC in this case. If so, the trauma was more likely a contributory factor than the sole cause of the carcinoma. (15 refs)

- 79-2859 In Vivo studies of Melanoma Cells Cultured With 5-Bromodeoxyuridine (Meeting Abstract).** (Eng) Gyi, K. K. (Georgetown Univ., Washington, DC, 20007); Wrathall, J. R. *In Vitro* 15(3): 197; 1979. (no refs)

- 79-2860 Growth and Metastasis of Human Melanoma Xenografts in the Hamster Host.** (Eng) Richmond, R. E. (Dept. Biological Sciences, Northern Kentucky Univ., Highland Heights, KY, 41076); Morton, D. L. *J Natl Cancer Inst* 62(4): 761-763; 1979.

The growth and metastasis of three human melanoma cell culture lines (UCLASO-M-12, UCLASO-M-7, and WEG-1) following transplantation into the cheek pouches of antilymphocyte serum (ALS)- and irradiation-immunosuppressed hamsters were studied. Tumor nodules were found in the cheek pouches of hamsters receiving any one of these lines, and by 90-100 days, multiple lung metastases were produced in nearly all hamsters. All cheek pouch tumors were identified as malignant melanomas, and the metastatic nodules were histologically identical. One or two animals within each group also developed gross metastases in other organs. Although grossly evident metastases were apparent in the lungs only after 90-100 days, microscopic foci of tumor cells were present within 20-30 days posttransplantation. The metastases may have been due to the biologic aggressiveness of the transplanted tumor cells and to the profound immunosup-

pression in the hosts caused by the combined whole-body irradiation-ALS technique. (10 refs)

- 79-2861 Epidermodysplasia Verruciformis Versus Disseminated Verrucae Planae: Is Epidermodysplasia Verruciformis a Generalized Infection with Wart Virus?** (Eng) Jablonska, S. (Dept. Dermatology, Warsaw Sch. Medicine, 02-008 Warsaw, Koszykowa 82, Poland); Orth, G.; Jarzabek-Chorzelska, M.; Rzeska, G.; Obalek, S.; Glinski, W.; Favre, M.; Croissant, O. *J Invest Dermatol* 72(3): 114-119; 1979.

Ten patients with epidermodysplasia verruciformis (EV) induced by human papilloma virus type 3 (HPV-3) and 10 patients with HPV-3-induced verrucae planae (VP) of long duration were studied to determine whether the two lesions can be distinguished on the basis of clinical, histologic, or immunologic criteria. HPV DNA's isolated from VP-type EV lesions or from the VP flat warts were alike and distinct from those of HPV-1, -2, and -4. The clinical picture was largely the same in the HPV-3-induced EV and VP, the main difference being that the EV cases were more refractory to treatment. The wartlike lesions of EV also resembled VP histologically; both had characteristic vacuolization of the cytoplasm, pyknosis of the nuclei in the upper malpighian and granular layers, and a basketlike appearance of the horny layer. In contrast, the red plaques on the trunks in EV induced by HPV-4 showed pronounced vacuolization of the nuclei in the entire malpighian and granular layers, and nucleoli and granules were prominent. Most EV and VP patients showed depressed, nonspecific, cell-mediated immunity. No malignant conversion was seen in patients infected with HPV-3, whereas it occurred in two patients infected with both HPV-3 and HPV-4. Pigmented plaques were the most important adverse prognostic sign in EV induced by HPV-3. (21 refs)

- 79-2862 Adenocarcinoma of the Larynx.** (Eng) Mal-lonee, M. S. (U.S. Navy, Okinawa, Japan); Maniglia, A. J.; Goodwin, W. J. *Ear Nose Throat J* 58(3): 115-118; 1979.

A 60-yr-old man presented with hoarseness of 2 mo duration and an enlarging, tender left neck mass. He had been treated with 6,000 rads for a T₁N₀M₀ moderately well-differentiated squamous cell carcinoma 2 yr earlier. Indirect laryngoscopy showed edematous left arytenoid cartilage and a sluggish left vocal cord. A granular erythematous lesion was seen in the left ventricle and on the left true vocal cord. Microscopic examination of the specimen obtained after total laryngectomy and left radical neck dissection revealed poorly differentiated adenocarcinoma (AC) of the left true cord. Nine of 41 nodes were also positive for AC. Review of the literature shows that AC of the larynx is rare. There are generally two types: adenoid cystic (cylindroma) and indeterminant. Hoarseness and dyspnea are the most common symptoms of

both. Men are affected more often than women, and both are affected mainly in the fifth to seventh decades. The poor prognosis of laryngeal AC may be due to its tendency toward noncontiguous spread. The adenoid cystic carcinomas tend to spread along nerve sheaths or to metastasize via hematogenous routes. The case reported may be radiation-induced or a second primary tumor. It is also possible that the original tumor was an AC that was misdiagnosed as squamous cell carcinoma. (5 refs)

- 79-2863 Dermoid Cyst of the Lung. A Case Report.** (Fre) Baril, A. (Service d'Anatomie Pathologique, CHU Bellevue, boulevard Pasteur, 42023 Saint-Etienne Cedex, France); Boucheron, S.; Moulin, J.; Abelanet, R. *Arch Anat Cytol Pathol (Paris)* 27(1): 53-57; 1979.

A case of dermoid cyst of the lung in a 38-yr-old woman is reported, and the literature on dermoid cysts is reviewed. The patient was admitted with a persistent pain in the left shoulder that began after a bronchopulmonary episode with cough and mucopurulent expectoration. X-ray studies showed a homogeneous opacity of av density in the left upper lobe of the lung. Bronchial mucosa samples obtained by bronchoscopy showed inflammation, but no abnormal growth or mucosal dysplasia. Aspirated sputum did not show tumor cells. Six months later, the shoulder pain had disappeared, but the cough and mucopurulent sputum persisted. An upper lobe lobectomy was decided upon despite no change in the x-ray picture or any other biological or histological examinations. A mass the size of a small orange was found in the posterior apical segment of the upper lobe, and enlarged lymph nodes were removed from the area of the aorta. The cyst contained hair and abundant sebaceous material. Among the theories on the histogenesis of dermal cysts in nongenital organs, the most likely appears to be that they arise from blastomeres that have not been repressed in embryonal development. Pulmonary sites are rare, with only 28 reported in the literature since 1861. (46 refs)

- 79-2864 In Vitro ³H-Thymidine Uptake by Esophageal Mucosa in Cat Experimental Esophagitis (Meeting Abstract).** (Eng) Livstone, E. (Dept. Medicine, Yale Univ., New Haven, CT); Sheahan, D.; Reinprecht, C.; Contino, C.; Selling, J.; Biancani, P.; McCallum, R. *Gastroenterology* 76(5, part 2): 1187; 1979. (no refs)

- 79-2865 On the Malignant Potential of Acquired Short Esophagus.** (Eng) Menguy, R. (Genesee Hosp., 224 Alexander St., Rochester, NY, 14607). *Arch Surg* 114(3): 260-263; 1979.

Adenocarcinoma developed in an esophagus lined with columnar epithelium (CE) in a 62-yr-old man with eso-

phageal reflux of 10 yr duration. The latter had led to esophageal shortening. In areas with CE, the metaplastic glandular tissue had a variegated appearance ranging from a quiescent CE to severe dysplasia to focal invasive adenocarcinoma. The more severe dysplastic changes were found close to two foci of carcinoma. These two discrete foci suggested that the potential for malignant degeneration inherent in CE complicating long-standing gastroesophageal reflux is high. (12 refs)

- 79-2866 Stomal Polypoid Hypertrophic Gastritis. A Polypoid Gastric Lesion at Gastroenterostomy Site.** (Eng) Koga, S. (Second Dept. Pathology, Faculty of Medicine, Kyushu Univ. 60, 3-1-1, Fukuoka 812, Japan); Watanabe, H.; Enjoji, M. *Cancer* 43(2): 647-657; 1979.

Stomal polyps (gastritis cystica polyposa, GCP) occurring at the gastroenterostomy site in four men were studied, as were the gastrectomy sites in 38 patients with previous gastroenterostomy who had recently undergone resection of the stomach. The GCP lesions were all located on the gastric side of the anastomosis, and they were most conspicuous near the greater curvature. Sessile polypoid protrusions were observed over the gastric mucosa beside the anastomotic stoma. The chief histologic findings were elongation of the gastric pits, hyperplasia and cystic dilation of the pseudopyloric glands, and epithelial invasion of the submucosa. Similar but less severe changes were observed in the mucosal folds adjacent to the polyps. Scar and granulation tissues were seen at the anastomotic junction of the stomach and jejunum. Among the resection patients, 25 showed localized mucosal hypertrophy or thickening at the anastomotic site. In 9/25 patients, the thickened folds were grossly and macroscopically of a polypoid appearance similar to that of GCP but less severe. The GCP lesions might be considered stomal polypoid hypertrophic gastritis due to chronic irritation by the duodenal juice, resulting in a localized hypertrophy of the fundic mucosa at the anastomotic stoma. (21 refs)

- 79-2867 Familial Polyposis Coli: Heterogeneous Polyp Expression in 2 Kindreds.** (Eng) Lynch, H. T. (Dept. Preventive Medicine and Public Health, Creighton Univ. Sch. Medicine, 2500 California St., Omaha, NB, 68178); Lynch, P. M.; Follett, K. L.; Harris, R. E. *J Med Genet* 16(1): 1-7; 1979.

Two extended kindreds are described that show marked variability in the clinical expression of colorectal adenomatous polyps, ranging from the classical presentation of familial polyposis coli (FPC) to the occurrence of only occasional or solitary adenomatous polyps in the colon. In one family, two individuals had diffuse polyposis at very early ages (7 and 10 yr old) and six others (23-72 yr old) had solitary polyps only. Of the patients with solitary polyps, two had associated colonic malignancies (26 and 35 yr old) and another had a

prophylactic colectomy at age 46. In the second family, 5/11 patients with evidence of polyps showed the classical presentation of FPC and the remainder showed marked phenotypic variation. In the absence of reliable markers and/or distinguishing physical signs signifying genotypic status (as in Gardner's syndrome), it may not be possible to discriminate between those patients who may have reduced penetrance of the FPC gene (yet with high colon cancer risk) and the patient who is free of the FPC gene, but has sporadic polyps of an unknown etiology. It is concluded that these data, although limited to only two families, indicate the need for a more careful description of clinical signs, particularly the number, site, and distribution of colon polyps in high-risk patients from families with a tendency to colon cancer and/or classical FPC. (28 refs)

- 79-2868 Probable Parasellar Meningioma in a Pregnant Woman.** (Eng) Manganiello, P. D. (Endocrinology Unit, Dept. Obstetrics and Gynecology, Medical Coll. Georgia, Augusta, GA, 30909); Meltz, R. C.; Andros, G. J. *Obstet Gynecol* 53(3, Suppl): 435-465; 1979.

The case of a 28-yr-old woman who developed a probable intracranial meningioma during the second trimester of her fifth pregnancy is reported. Presenting symptoms were unilateral periorbital edema of the right eye with paralysis of its intrinsic muscles, headaches, and slight thyroid enlargement. A cerebral arteriogram and axial computerized tomography revealed a probable meningioma arising from the cavernous sinus. Her symptoms improved following spontaneous delivery at 34 wk gestation, and by 4 mo after delivery they had disappeared. Arteriograms showed a definite decrease in the size of the tumor mass. A literature search for meningiomas diagnosed during pregnancy revealed only 39 cases reported since 1927. The spatial relationship of these tumors with surrounding structures is important. In tumors located close to the optic or oculomotor nerves, in the orbit, or in the cavernous sinus, only a slight increase in size produces clinical signs. Since meningiomas are highly vascular tumors, the increased vascularity and engorgement of the pregnant patient's vascular supply to the tumor contribute to the symptoms produced by the tumor. Although the tumor mass expands, there is no increased hyperplastic activity. (18 refs)

- 79-2869 Polyps of the Colon after Ureterosigmoidostomy.** (Fre) Fievez, M. (Institut de Morphologie Pathologique, 6270 Lovervall, France); Rubay, J.; Lefevre, P.; de Pierpont, B. *Arch Anat Cytol Pathol (Paris)* 27(1): 40-44; 1979.

Two cases of intestinal polyps that developed at the site of implantation of ureters in the sigmoid colon are reported. The first patient was an adolescent boy who had been born with an exstrophic bladder. At 2 yr of age, a bilateral ureterosigmoidostomy was performed that had to be abandoned at age

9 yr because of recurrent infection; a cutaneous ureterostomy was then performed. Seven years later, a pediculated polyp that was 3 cm in diameter was removed from the sigmoid colon at the site of the ureterostomy. The second patient, an 18-yr-old girl, also was born with an exstrophic bladder. She originally had a sigmoid implantation of the ureters; subsequently, they were moved to the skin and then to a cecal neobladder. Kidney infections were recurrent and the patient finally died of renal insufficiency. At autopsy, two 3-cm-diameter polyps were found in the colon at the sigmoid implantation site. Histologically, the polyps were composed of intestinal glands with large strands of epithelial tissue and nests of von Brunn cells. Numerous areas of metaplastic transformation of transitional epithelium into colonic mucous epithelium were observed. It is suggested that the polyps originated from urothelial cells from the implanted ureters. (23 refs)

- 79-2870 β -Glucuronidase in Schistosomal Intestinal Polypi of the Colon.** (Eng) El-Zoghby, S. M. (Dept. Applied Medical Chemistry, Medical Res. Inst., Alexandria Univ., Alexandria, Egypt); El-Kholy, Z. A.; El-Shrkawy, A.; Rashad, M.; El-Kilany, S.; Abaza, H.; Gawish, Y. S. *Acta Vitaminol Enzymol (Milano)* 32(1/4): 7-11; 1978.

β -Glucuronidase activity was determined in 20 patients with schistosomal polyposis of the large bowel and 10 patients with a normal colon. β -Glucuronidase activity was significantly increased in schistosomal polypi (339-11,615 units) compared with normal mucosa (313-1406 units; 1 unit liberates 1 μ g phenolphthalein in 1 hr at 37 C). No β -glucuronidase activity was detected in schistosoma ova suspensions. Histopathological examinations showed the classical schistosomal granulomatous reaction. There was no evidence of malignant or premalignant changes in any of the 20 bilharzi patients; however, all the patients had excessive mucus in the colon and rectum. It is hypothesized that the increase in the enzymatic activity of these patients may be due to (1) the presence of a great number of different types of WBC in the granulomas of the schistosomal reaction, (2) the presence of excess mucus, which, with its protein content, could activate the enzyme to a great extent, and (3) the liver dysfunction (usually mild) that was found in the patients. It is concluded that there is no proof of a carcinogenic effect of β -glucuronidase activity in the large intestine of patients with schistosomiasis, nor is its increase an indication of malignancy, as is the case in the urinary bladder affected by schistosomiasis. (15 refs)

- 79-2871 Primary Sarcoma of the Diaphragm.** (Ita) Tufano, G. (I Facolta di Medicina e Chirurgia, Universita di Napoli, Naples, Italy). *Rass Int Clin Ter* 58(17): 1191-1200; 1978.

An abdominal tumor was diagnosed preoperatively by x-rays

in a 38-yr-old man who presented with hypochondrial and epigastric pains. A large tumor of the diaphragm was found at laparotomy. The tumor was diagnosed histologically as a spindle cell sarcoma that probably arose from the stroma. (12 refs)

- 79-2872 Peutz-Jeghers Syndrome Associated with Gastrointestinal Carcinoma.** (Eng) Cochet, B. (Clinique Medicale Therapeutique, Hopital Cantonal, CH 1211 Geneva 4, Switzerland); Carrel, J.; Desbaillets, L.; Widgren, S. *Gut* 20(2): 169-175; 1979.

The malignant potential of Peutz-Jeghers hamartomatous polyps, generally considered benign, is illustrated by two familial cases. Metastasizing gastrointestinal carcinomas associated with the syndrome developed in a 56-yr-old woman and her 29-yr-old son. Both mother and son died from duodenal and gastric carcinomas, respectively, which developed in hamartomatous polyps with extensive metastases. In both cases, there were dysplastic areas within the polyps. These features indicate that hamartomatous polyps may, in some cases, be precursors of digestive tract carcinomas. (19 refs)

- 79-2873 Defining the Precursor Tissue of Ordinary Large Bowel Carcinoma: Implications for Cancer Prevention.** (Eng) Lane, N. (Div. Surgical Pathology, Dept. Surgery, Coll. Physicians and Surgeons, Columbia Univ., New York, NY, 10032); Fenoglio, C. M.; Kaye, G. I.; Pascal, R. R. In: *Gastrointestinal Tract Cancer*. Lipkin, M.; Good, R. A., eds. (New York, London: Plenum Medical Book Co.) Sloan Kettering Inst. Cancer Series 602 pp.; 295-324; 1978.

In this attempt to define the precursor (precancerous, or preneoplastic) tissue of ordinary large-bowel carcinoma (Ca), only those benign proliferations known as adenomas (AN's) and hyperplastic polyps (HP's) and only ordinary moderately and well-differentiated adenocarcinomas were considered. HP's have no statistically significant relationship as a precursor tissue to Ca or AN; nonetheless, they are frequently found in colons bearing AN and/or Ca as anatomically separate lesions. In contrast to HP's, several statistical correlations can be noted among the size, shape, and histological patterns of AN's. The av small AN (1-1.5 cm) tends to be pedunculated and to have a tubular pattern. Among larger AN's, a greater proportion tend to be sessile and to have a papillary (villous) pattern. HP's are 10 times as common as AN's and, in turn, small AN's are 10 times as common as large AN's; therefore, the latter constitute only about 1% of all benign proliferations being considered. Ca, intramucosal and invasive, can occur in small AN's, but only the large AN's will harbor invasive Ca with significant frequency ($\geq 10\%$). The likelihood of Ca increases with AN size plus the tendency of larger AN's to be sessile and have papillary features. The cellular origin of Ca involves minute or microscopic dimensions. A

small lesion, to be acceptable as representing the morphology of a neoplasm at its inception, would not be more than a few millimeters or even a fraction of a millimeter in size. Minute or microcancer has been observed in adenomatous tissue but has not been reported as a de novo process in normal mucosa, in spite of unlimited opportunity to find it. De novo Ca also does not seem to occur in familial polyposis, even though there is a tremendous predilection for Ca in this condition. Evidence from a 25-yr clinical study also supports the contention that most ordinary colon adenocarcinoma arises from adenomatous tissue. (19 refs)

- 79-2874 Familial Polyposis Coli.** (Eng) Bussey, H. J. (St. Mark's Hosp., London, England); Morson, B. C. In: *Gastrointestinal Tract Cancer*. Lipkin, M.; Good, R. A., eds. (New York, London: Plenum Medical Book Co.): Sloan Kettering Inst. Cancer Series 602 pp.; pp. 275-294; 1978.

The sex and age distribution, symptoms, inheritance, incidence in the general population, diagnosis, histology, treatment, relation with malignancy, and similarity to Gardner's syndrome of familial polyposis coli (FPC) are reviewed. There is an equal sex distribution, and the average age at diagnosis is 35. Rectal bleeding is the most common symptom, and the disease is characterized by the presence of hundreds or thousands of adenomatous tumors in the colon and rectum. It is now firmly established that the lesions are genetic in origin, most victims having inherited the disease from an affected parent. There is a high incidence of intestinal carcinoma in FPC patients, and the incidence is particularly high in patients with untreated disease. The evidence suggests that colorectal cancer arises in preexisting adenomas. Several recent reports suggest that all FPC may be Gardner's syndrome with varying degrees of expressivity. FPC is a useful model for study of the development of adenoma and carcinoma in the large intestine. The adenomas in FPC are indistinguishable from those found more generally in the nonpolyposis population; adenomas from both sources can show progressively increasing epithelial atypia that finally equates with that seen in invasive carcinoma. The disease is also closely associated with other growth patterns, mostly of a neoplastic nature, some benign and some malignant. (27 refs)

- 79-2875 Five Cases of Leiomyosarcoma of the Small Intestine.** (Jpn) Terada, K. (Dept. Surgery, Kochi Prefectural Central Hosp., Kochi, Japan); Kondo, K.; Hashimoto, T.; Iwata, K.; Nishiyama, A.; Hisano, K. *Gan No Rinsho* 25(2): 143-147; 1979.

The case reports of five patients (3 men, 2 women aged 45-59 yr) with small intestinal leiomyosarcoma are presented. One of the tumors was in the duodenum and four were in the jejunum; all five tumors showed exophytic growth. One pa-

tient had metastatic foci in the liver, one had massive metastases to the liver, and the third had massive metastases to the liver, peritoneum, and lymph nodes. (10 refs)

- 79-2876 Vagotomy and Gastric Cancer.** (Hun) Gombkoto, B. (Sebeszeti es Urologiai Osztaly, Megyei Korhaz, Eger, Hungary); Tasi, I. *Orv Hetil* 120(5): 259-264; 1979.

Gastric carcinoma was found in two patients who had undergone vagotomy. Selective vagotomy was performed for duodenal ulcer in a 57-yr-old woman. A partly mucocellular scirrhous carcinoma of the stomach was diagnosed 5 yr later. Truncal vagotomy was performed in a 52-yr-old man 8 yr after Billroth-II gastric resection for duodenal ulcer. A partly medullary adenocarcinoma of the gastric stump was found 16 yr after the vagotomy. The findings suggest that there is a possible relationship between vagotomy and gastric cancer. (44 refs)

- 79-2877 Patterns of Tumor Cell Aggregation and Metastasis in Scirrhous Carcinoma of the Stomach, Borrmann Type IV.** (Jpn) Kodama, Y. (Dept. Surgery II, Kyushu Univ. Sch. Medicine, Fukuoka 812, Japan); Inokuchi, K. *Gan No Rinsho* 25(2): 89-93; 1979.

The patterns of tumor cell aggregation and metastasis in 43 patients with Borrmann Type IV scirrhous carcinoma of the stomach were investigated. Of the 24 patients with lymphatic involvement, 21 showed small nest-type aggregation (SNA) and 3 showed free cell aggregation (FCA). Tumor cell aggregation in the 29 patients with lymph node involvement was of the nest type in 22 and the free cell type in 7. Lymphatic involvement in the submucosal layer (19/24) and serosa (9/24) was most common. Transitional cancer cells occurred in all 29 patients with lymph node involvement, with 24% showing FCA and 76% SNA. FCA was mainly associated with low levels of lymph node transition and lymphatic infiltration, SNA with higher levels. SNA was associated with low to moderate degrees of fibrosis in the submucosal layers of the stomach wall, FCA with moderate to high degrees of fibrosis. Free cells in the mucosal layer infiltrated diffusely into the interstitium with little lymphogenous spread, but small nests of cells were accompanied by frequent lymphogenous spread. (5 refs)

- 79-2878 The Viability of Free Cancer Cells in the Peritoneum of Patients with Gastric Cancer.** (Jpn) Tanida, O. (First Dept. Surgery, Tottori Univ. Sch. Medicine, Yonago-shi 683, Japan); Takeuchi, T.; Kaneshima, S.; Iit-suka, Y.; Sasaki, Y.; Koga, S. *Gan No Rinsho* 25(2): 101-106; 1979.

Changes in the morphology and viability of free cancer cells

in the peritoneum of 10 stomach cancer patients were evaluated by enzymological staining, Giemsa staining, and autoradiography with ^3H -thymidine. The patients were six men and four women aged 41-69 yr. After abdominal incision but before surgery, 150 ml warm culture fluid were injected into the area of Douglas' space and then collected immediately and stained. A second sample was taken from the same area by the smear method and stained. The free cancer cells showed moderate degeneration but high viability. Cancer cells on the gastric serosa also showed high viability. Thus, the implantation and proliferation of free cancer cells on the peritoneum is possible. Ip injection of 10 mg mitomycin C (MMC) markedly decreased the viability of the free cells in Douglas' space. Ip injection of MMC after gastric surgery was judged to be an effective method of preventing the peritoneal dissemination of cancer cells. (13 refs)

- 79-2879 Malignant Change of Juvenile Polyp of Colon: A Case Report.** (Eng) Tung-hua, L. (Capital Hosp., Chinese Acad. Medical Sciences, Peking, China); Min-chang, C.; Hsien-chiu, T.; Lan, C.; Chieh, L. *Chinese Medical J* 4(6): 434-439; 1978.

Malignant change of a juvenile polyp of the descending colon (near the splenic flexure) occurred in a 16-yr-old boy. The pathologic diagnosis was malignant change of a juvenile polyp of the descending colon into a signet ring cell carcinoma with tumor thrombi in the lymphatics and blood vessels. A review of the world literature on juvenile polyps over the past 20 yr failed to reveal any report indicating that this condition is precancerous or potentially malignant. However, the present case is believed to be a typical one of juvenile polyp undergoing malignant change because the carcinoma developed entirely within the polyp. Its pedicle and the surrounding colonic wall were devoid of cancer. (12 refs)

- 79-2880 Anabolic Steroid Therapy and Intrahepatic Cholangiocarcinoma.** (Eng) Stromeyer, F. W. (Hepatic Pathology Dept., Armed Forces Inst. Pathology, Washington, DC, 20306); Smith, D. H.; Ishak, K. G. *Cancer* 43(2): 440-443; 1979.

The case of a 47-yr-old man who developed a cholangiocarcinoma 3 yr following the commencement of anabolic steroid (oxymetholone) therapy for refractory hypoplastic anemia is reported. The tumor showed mucin production and was devoid of hepatocellular elements; there were metastases to the lungs and abdominal lymph nodes. Previous reports have suggested a possible relationship between anabolic steroid therapy and hepatocellular carcinoma. However, in many of these cases there are doubts concerning the histologic diagnosis, malignant potential, or nature of the association between steroid therapy and tumor. Although a causative relationship between the steroid therapy and tumor in the present case is not established, the case does suggest that intrahepatic cho-

langiocarcinoma should be included among the group of hepatic lesions possibly related to anabolic steroid therapy. (35 refs)

- 79-2881 Spontaneous Liver Tumors in Aged Germfree Wistar Rats.** (Eng) Pollard, M. (Lobund Lab., Univ. Notre Dame, Notre Dame, IN, 46556); Luckert, P. H. *Lab Anim Sci* 29(1): 74-77; 1979.

The incidence of spontaneous liver tumors in 132 germfree Lobund Wistar rats aged > 30 mo was studied. A total of 115 of these rats (76/90 males and 39/42 females) had 5-20 tumors per liver. In addition, three rats had cystic lesions and two showed extensive necrosis in their livers. A wide range of histologic changes was observed; 11 rats had changes compatible with a diagnosis of hepatocellular carcinoma. No clearly defined liver cirrhosis or metastatic lesions were observed. Large numbers of rats also developed benign adenomas of the pituitary, thymus, breast, adrenal cortex and medulla, parathyroid, and thyroid. Similar benign tumors were observed in germfree rats aged 20-29 mo, and benign liver neoplasms were observed in 16/70 males and 2/22 females. Liver lesions were observed among relatively few of the 200 conventional Wistar rats examined up to 25 mo of age; few of these animals lived as long as 30 mo. The etiology of the liver lesions in the aged germfree rats is not known. (17 refs)

- 79-2882 Atypical Carcinoma of Kidney. Possibly Originating from Collecting Duct Epithelium.** (Eng) Cromie, W. J. (Dept. Urology, National Naval Medical Center, Bethesda, MD, 20014); Davis, C. J.; DeTure, F. A. *Urology* 13(3): 315-317; 1979.

A case of atypical renal carcinoma showed cellular elements of both renal cell and transitional cell carcinoma. The unusual histologic features suggest that the tumor originated in the collecting duct epithelium. (10 refs)

- 79-2883 Childhood Cancer and the SBLA Syndrome.** (Eng) Lynch, H. T. (Dept. Preventive Medicine/Public Health, Creighton Univ., Omaha, NB, 68178); Guirgis, H. A. *Med Hypotheses* 5(1): 15-22; 1979.

Two kindreds manifest a syndrome characterized by a hereditary predisposition to sarcoma (S), brain tumors and breast cancer (B), leukemia, lymphoma, and laryngeal and lung cancer (L), and adrenal cortical carcinoma (A), known as the SBLA syndrome, are described. In these two nuclear families the progeny of breast cancer-affected mothers manifested early childhood malignant neoplasms. These observations have led to the development of a genetic-environmental interactive model that incorporates Knudsen's two-hit hypothesis as a

partial explanation for the exceedingly early onset of cancer in the progeny. Given the assumption that the first hit was germinal with transfer of the deleterious SBLA gene at conception, it is postulated that the second or somatic hit occurred early in utero. For example, it is likely that the rhabdomyosarcomas occurring in progeny of one family and the adrenal cortical carcinoma occurring in progeny of the second family most probably had their origins in utero. Given the chronology of the tumor occurrences, the mothers from the families must have manifested occult carcinoma of the breast while their cancer-affected fetuses were developing. This hypothesis provides a rational explanation for the interaction of the SBLA genotype (first hit) and other etiologic events (putative oncogenic virus, a derepressed oncogene, and tumor-specific antigens) that may have crossed the placenta and contributed to cancer in the fetus (second hit). This theory is based on the supposition that the mothers had already sustained both hits (germinal and somatic) and that their fetuses received the germinal hit at conception but sustained the second hit transplacentally. (16 refs)

79-2884 The Problem of Carcinoma Developing in a Fibroadenoma. Recent Experience at Memorial Hospital. (Eng) Fondo, E. Y. (135 E. 71st St., New York, NY, 10021); Rosen, P. P.; Fracchia, A. A.; Urban, J. A. *Cancer* 43(2): 563-567; 1979.

Fourteen new cases of unsuspected carcinoma developing in fibroadenomas are reported. The patients averaged 42 yr of age at the time of diagnosis, and four had a previous history of exogenous hormone usage. Mammography revealed an abnormality in two-thirds of the cases. The microscopic appearance was similar to that of carcinoma unassociated with fibroadenoma. Twelve patients had noninvasive carcinoma, one had intraductal and infiltrating duct carcinoma, and one had intraductal carcinoma in the fibroadenoma and infiltrating carcinoma in the adjacent breast parenchyma. The fibroadenomas involved by carcinoma generally showed a fairly abundant benign epithelial component, but there was no evidence of striking or unusual epithelial hyperplasia in the breast tissue adjacent to the carcinoma-containing fibroadenomas. Four of the 14 patients also had carcinoma in the contralateral breast, the bilateral disease being synchronous in 2 patients. All patients are alive and clinically free of disease following treatment (local excision in 3 patients, mastectomy in 11). Contralateral breast biopsy at the time of diagnosis with a careful lifetime follow-up are appropriate because of the high risk of contralateral invasive carcinoma. (11 refs)

79-2885 Detection of Asymptomatic Breast Cancer in the Second Breast by Mammographic Follow-Up: A 5-Year Survey. (Eng) Rosner, D. (Dept. Breast Surgery, Roswell Park Memorial Inst., Buffalo, NY, 14263); Nemoto, T.; Dao, T. L. *Prev Det Cancer Part II Vol I* 1281-1285; 1978.

The detection of asymptomatic cancer in the other breast by routine periodic mammography was analyzed among 408 women who had previously undergone one mastectomy for carcinoma. A total of 221 patients were followed for 2-5 yr (av 3 mammography studies/patient); the remaining 187 were followed for a relatively short time (1-2 studies/patient). Mammography demonstrated abnormalities in 50 patients. Biopsy of the suspicious area in 31 of these patients revealed carcinoma in 12 and various benign lesions in 19. Mammographic findings in the 12 carcinoma patients were a mass in 5, a cluster of calcifications in 3, and a mass with calcifications in 4. Nine patients had invasive carcinomas, (2 lobular and 7 adenocarcinomas) and 3 had in situ lobular carcinomas. Positive axillary nodes were found in only 1/7 patients who underwent mastectomy for invasive carcinoma. Two patients who did not undergo biopsy in spite of abnormal mammographic findings developed clinically apparent carcinomas 12 and 24 mo later. During the study period, eight breast cancers were discovered by clinical examinations prior to mammography. The tumor size was larger in this group, and all had axillary nodal metastasis. Four are free of disease, one is living with metastasis, and three died of metastatic disease. Among the nine patients whose invasive carcinomas were detected by mammography, seven are free of disease and two died. (12 refs)

79-2886 Adenocarcinoma of the Vagina in a Patient with Gonadal Dysgenesis. (Eng) Shingleton, H. M. (Dept. Obstetrics and Gynecology, Univ. Alabama, University Station, Birmingham, AL, 35294); Younger, J. B.; Beasley, W. E.; Levy, D.; Gore, H.; Lawrence, W. D. *Obstet Gynecol* 53(3, Suppl): 925-975; 1979.

A case of adenocarcinoma of the vagina in a 21-yr-old black woman with gonadal dysgenesis is presented. The tumor contained papillary areas and areas of adenocarcinoma with small gland spaces. In the papillary areas, the cytoplasm was scanty and pale-staining, forming a 'hobnail' pattern. The histologic pattern suggested derivation from the paramesonephros. Evidence of mucin production by the tumor also favored a paramesonephric origin, as did the absence of fat in the electron micrograph. Electron microscopy identified two cell populations: pale-staining (light) cells that were large and polygonal with short, stubby microvilli on the luminal surfaces; and dark cells, which were seen less frequently and were smaller than the light cells. The light cells resembled those seen in a previously reported vaginal clear cell diethylstilbestrol-related adenocarcinoma with respect to basal-located lysosomes of unusual shape, high metabolic activity, and abundant glycogen and granular endoplasmic reticulum. The development of vaginal adenocarcinoma in this patient is unique in that she had no exogenous estrogen influence antenatally and an estrogen deficiency postnatally. (19 refs)

79-2887 Granulosa Cell Deficient Follicles. Occurrence, Structure, and Relationship to Ovarian Terato-

carcinogenesis in Strain LT/Sv Mice. (Eng) Eppig, J. J. (Jackson Lab., Bar Harbor, ME, 04609). *Differentiation* 12(2): 111-120; 1978.

A comparison study was made of the diameters of oocytes in follicles having a single layer of granulosa cells (GC's) in 4-wk-old LT/Sv mice and in mice of various common strains. There is a unique population of these follicles in strains LT/Sv and C58/J in which the oocytes are significantly larger than the oocytes in single GC-layered follicles of other common strains (C57BL/6J, BALB/cJ, and DBA/2J). These unique follicles are referred to as GC-deficient (GCD) follicles, since oocytes of these sizes are usually found in follicles with more than a single layer of GC's. The parthenogenetic embryos that give rise to ovarian teratomas in strain LT/Sv are usually found in GCD follicles. Some of the ova of strains LT/Sv and LTXBP, but not the ova of the other strains, are capable of spontaneous parthenogenetic activation after meiotic maturation. Although the ovulated ova of strain LTXBP are capable of spontaneous parthenogenetic development, the frequency of GCD follicles and teratocarcinogenesis is low. Therefore, the frequency of ovarian teratocarcinogenesis is correlated with the simultaneous occurrence of two atypical conditions: (1) the capability of the matured ova to undergo spontaneous parthenogenetic activation and, (2) the high frequency of GCD follicles. GCD follicles containing oocytes with a diameter $> 65 \mu\text{m}$ were studied by electron microscopy. The follicles are usually enclosed within a layer of flattened thecalike cells. A basal lamina separates these cells from a single layer of cuboidal GC's. GC processes traverse the zone pellucida to contact the oocyte, which shows ultrastructural characteristics typical of oocytes in the final growth stages. It is proposed that the GCD follicles are competent to participate in the normal functions of follicular cells relating to oocyte growth and meiotic maturation. (13 refs)

79-2888 Primary Pure Insular Ovarian Carcinoids. (Eng) Holtz, G. (9 Duffy St., Charlestown, SC, 29407); Tucker, E.; Holtz, F. *Obstet Gynecol* 53(3, Suppl): 855-875; 1979.

The 23rd and 24th literature cases of primary pure insular ovarian carcinoids are presented. One of the patients, a 66-yr-old woman, presented initially with carcinoid heart disease. Severe tricuspid insufficiency, pulmonary stenosis, and mild aortic insufficiency were documented. A unilateral salpingo-oophorectomy (S-OP) was performed. Argentaffin reaction was positive. The second case was seen in a 46-yr-old woman who presented with complaints of urinary incontinence and frequency. This patient underwent a total abdominal hysterectomy and bilateral S-OP. Argentaffin reaction was weakly positive. No teratomatous elements or evidence of luteinization was seen in either patient. If the diagnosis of carcinoid syndrome in the absence of hepatic involvement is made, an extensive search should be made for lesions other than an ovarian primary. This must include a search for non-

carcinoid malignant lesions that may present with the carcinoid syndrome. Most of these tumors behave in a benign manner, with only three cases of documented metastases being reported. In view of this, unilateral S-OP is adequate therapy for the premenopausal patient if the opposite ovary is normal. Postoperatively, patients should be followed with serial 5-hydroxyindolacetic acid determinations, with any rise being diagnostic of recurrence. (10 refs)

79-2889 A Histologic Study of Ovarian Endometriosis with Emphasis on Hyperplastic and Atypical Changes. (Eng) Czernobilsky, B. (Dept. Pathology, Kaplan Hosp., Rehovot, Israel); Morris, W. J. *Obstet Gynecol* 53(3): 318-323; 1979.

The histologic features of ovarian endometriosis were studied in 194 affected patients seen over a 3-yr period (1972-1974). The mean age was 38.3 yr, and 181 patients were white and 13 were black. The lesions were bilateral in 42 patients, localized in the left ovary in 88, and localized in the right ovary in 64. All patients had endometrial-type epithelium and stroma. Ovarian-type epithelium was demonstrated in 26 patients, papillary epithelial-lined projections in 7, and hobnail cells in 36. Reactive epithelial changes were observed in 43 patients, and this feature was usually associated with severe stromal inflammation and areas of epithelial denudation and regeneration. Severe epithelial atypism and degenerative changes were present within the cyst linings of seven patients. In two of these patients, severely atypical changes were also observed within glandular structures; in three, stromal inflammation was present underneath the epithelium. Adenomatous hyperplasia was seen in one of these patients as well as in four other patients. Although the areas of severe epithelial atypism in ovarian endometriosis may be of reactive origin, the possibility that in some instances these changes may have a neoplastic potential must be considered. (15 refs)

79-2890 Adenocarcinoma of the Endometrium in Pregnancy. (Eng) Sandstrom, R. E. (James Homer Wright Pathology Labs., Massachusetts General Hosp., Boston, MA); Welch, W. R.; Green, T. H. *Obstet Gynecol* 53(3, Suppl): 735-765; 1979.

The seventh case of endometrial adenocarcinoma coexistent with an intrauterine pregnancy is reported. This case is believed to be the first noted as an incidental finding in therapeutic abortion. Because microscopical examination of the uterine contents of the patient, a 37-yr-old woman, revealed foci of well-differentiated adenoacanthoma in addition to products of conception, a hysterectomy and bilateral salpingo-oophorectomy were performed 3 wk later. All seven patients were white and were 21-43 yr old. Most complained of dysmenorrhea, irregular menses, or hypermenorrhea of recent onset and short duration. Five patients were multiparous, two nulliparous. Only one had a history of previous

estrogen therapy, and that was only for a short period. Except for obesity, constitutional factors associated with endometrial carcinoma (EC) were not reported. The tumor was limited to a small focus in four cases. Four of the neoplasms were adenoacanthomas, tumors that account for only 20%-30% of the endometrial adenocarcinomas in the general population. The one patient who died had an adenoacanthoma and was the only patient with extensive myometrial invasion at hysterectomy. The apparent rarity of EC in association with pregnancy may be related to several factors: EC generally occurs in the postmenopausal period; patients prone to EC are, as a group, less fertile; the hormonal milieu of pregnancy with high progesterone levels may be unfavorable for tumor survival. (15 refs)

- 79-2891 Massive Necrosis of Uterine Leiomyoma Following Administration of a Single Dose of Medroxyprogesterone.** (Spa) Molina, R. (Servicio de Ginecología, Hospital General del Sur, Maracaibo, Venezuela); Torres, R. *Invest Clin (Maracaibo)* 19(2): 62-67; 1978.

A 37-yr-old woman with an unremarkable gynecological history developed acute abdomen approx 2 wk after she was inoculated with a single 150-mg dose of medroxyprogesterone acetate for contraception. Peritonitis and uterine myoma were diagnosed. Total hysterectomy was performed after symptomatic treatment. Nearly complete necrotic uterine leiomyomas were found. The acute abdomen was due to the tumor necrosis. The patient has been symptom-free 3 yr after surgery. (7 refs)

- 79-2892 Carcinoid of the Uterine Cervix. A Case Report with Light and Electron Microscopic Studies.** (Eng) Habib, A. (Dept. Pathology, Mt. Sinai Sch. Medicine, City Univ. New York, 100th St. and Fifth Ave., New York, NY, 10029); Kaneko, M.; Cohen, C. J.; Walker, G. *Cancer* 43(2): 535-538; 1979.

The case of a 33-yr-old woman with primary carcinoid of the uterine cervix is reported. The woman had had three pregnancies, one of which ended in abortion. A radical Wertheim hysterectomy with bilateral pelvic lymphadenectomy, aortic lymphadenectomy, and right salpingo-oophorectomy was performed. There has been no evidence of recurrent disease in the 2.5 mo since surgery. Microscopic examination of the cervix revealed a tumor forming solid nests, trabeculae, and glands. The tumor infiltrated the entire thickness of the cervical wall, but it did not extend to the parametria or exocervical margin. Several lymphatics contained tumor cells. The tumor cells showed argyrophil granules, but they were negative for the argentaffin reaction. Electron microscopy revealed the presence of numerous neurosecretory granules and microfilaments. The tumor was classified as a well-differentiated carcinoid of the uterine cervix. Carcinoid of the uterine cervix is a distinct tumor entity and should be distinguished from

the more common epidermoid and adenocarcinoma variants. (26 refs)

- 79-2893 Morphogenesis of Cervical Carcinoma.** (Rus) Murav'ev, G. N. (Rest. Inst. Oncology and Medical Radiology, Minsk, USSR). *Vopr Onkol* 25(2): 13-19; 1979.

The diagnostic efficacy of alterations in the stroma of the cervix uteri for distinguishing between proliferative or dysplastic changes and preinvasive or early carcinoma was evaluated. Surgery and biopsy specimens were obtained from 30 patients with inflammatory processes, 60 with endocervicitis, 20 with erosions, 30 with ectropion, 125 with erosions plus epidermization and dysplasia, 57 with leukoplakias plus dysplasia, 180 with preinvasive intraepithelial carcinoma, 40 with Stage I carcinoma, 30 with Stage II carcinoma, and 20 with Stage III carcinoma. Patients with inflammations but without proliferation of the epithelium featured various changes in stroma structure; these changes were confined to the site of the pathologic focus. Progression of tumor development was associated with the appearance of acid mucopolysaccharides, increased vascularization, edema, and expansion of the boundaries of the tumor field. The rate of stromal and vascular changes correlated with the boundaries of the tumor field. (11 refs)

- 79-2894 The Basement Membrane in Experimentally Induced Atypias and Carcinoma of the Uterine Cervix in Mice. An Immunofluorescence Study.** (Eng) Rubio, C. A. (Dept. Pathology, Karolinska Sjukhuset, S-10401 Stockholm 60, Sweden); Biberfeld, P. *Virchows Arch [Pathol Anat]* 381(2): 205-209; 1979.

Basement membrane-specific antigens of the squamous epithelium of the uterine cervix were investigated in 19 normal C57Bl mice, in 7 mice with cervical atypia, and in 3 mice with invasive carcinoma. Cervical atypia and carcinoma were induced by local application of 1% benzo(a)pyrene in acetone. Basement membrane-specific antigens were demonstrated by immunofluorescence with sera from patients with bullous pemphigoid. Both normal squamous cervical epithelium and atypical cervical epithelium showed the presence of a continuous, clearly delineated basement membrane. Clusters of invasive squamous carcinoma were also surrounded by a fluorescent basement membrane, but it appeared fragmented or discontinuous. The results suggest that the ability of cervical squamous cells to secrete basement membrane antigens is not completely lost during carcinogenesis, thus substantiating previous observations in the human cervix. (16 refs)

- 79-2895 Germ Cell Neoplasms Arising in Gonadoblastomas.** (Eng) Hart, W. R. (Dept. Pathology, Univ. Michigan Medical Sch., 1335 E. Catherine St., Ann

Arbor, MI, 48109); Burkons, D. M. *Cancer* 43(2): 669-678; 1979.

The clinicopathologic findings in six phenotypic women with features of 46, XY pure gonadal dysgenesis who developed germinomas and other germ cell tumors in gonadoblastomas are reported. All stages in the evolution of germinoma from the germ cells of gonadoblastoma were observed. The earliest form corresponded to in situ germinoma, in which the nests of gonadoblastoma became distended with a monotonous proliferation of neoplastic germ cells. In the next phase, clusters of infiltrating cells coalesced to form a small nodule of microinvasive germinoma without producing a sizeable mass. Metastases have not yet occurred postoperatively in the two patients with these incipient microinvasive forms. Larger invasive germinomas occurred in four patients, two having pure germinomas and two having germinomas mixed with either mature teratoma or endodermal sinus tumor. Two of these tumors were complicated by metastatic spread to the lymph nodes, but moderate dosage radiation therapy resulted in long-term survival and probably cure in at least one of these patients. Although it is debatable whether gonadoblastomas are true neoplasms or blastomatoid dysgenetic malformations, they have the same malignant potential as tumors that arise de novo in the gonad or extragonadal sites. They also appear to have the same overall favorable prognosis and radiosensitivity as de novo ovarian germinomas. (15 refs)

79-2896 Testicular Tumor: Report of Three Familial Cases. (Eng) Lowe, W. C. (Dept. Medicine, Veterans Admin. Hosp., East Orange, NJ); Akgun, S. *Cancer Detect Prev* 2(1): 75-82; 1979.

The occurrence of malignant testicular neoplasms (TN) in two brothers and their nephew is reported. One brother was admitted at age 44 with a 5- x 5-cm mass in the right scrotum and a 6- x 6-cm mass in the right lower abdomen. All laboratory data were normal except for a lactic dehydrogenase level of 1,800 units. After radical orchiectomy (Or-x), histological section showed a mixed tumor consisting of seminoma and embryonal cell elements. His nontwin brother noted gradual enlargement of the right scrotum at age 23. He underwent right radical Or-x 1 yr later for testicular teratocarcinoma. A 16-yr-old nephew of the brothers noted a small mass in his right testis that eventually became painful. Histologic section after right Or-x showed embryonal cell carcinoma with choriocarcinoma elements. According to a review of the literature, malignant TN are rare. Most familial cases are in the age group 20-40. Cryptorchism and trauma to the testis are often associated with TN, although they had not occurred in the three reported patients. It is relatively easy to demonstrate that TN are probably genetically determined in experimental animals, but not in men. However, studies of the number of chromosomes in human TN showed that hyperdiploid, -triploid, and -tetraploid tumors were common. In a study of TN by the quinacrine fluorescence technique, all were found to contain a Y chromosome. This report of multiple TN in one family supports the hypothesis that these neoplasms are determined partly by genetic susceptibility. (32 refs)

79-2897 Mixed Brenner and Adenomatoid Tumor of the Testis. An Ultrastructural Study and Histogenetic Considerations. (Eng) Nogales, F. F. (Hospital Universitario, Avda, Spain); Matilla, A.; Ortega, I.; Alvarez, T. *Cancer* 43(2): 539-543; 1979.

The association of Brenner and adenomatoid tumor in the tunica vaginalis testis of a 37-yr-old man is reported. The adenomatoid tumor was the predominant pattern. Intimately admixed and randomly situated within it were numerous Brenner nests separated from the surrounding fibroblasts by a well-developed basal lamina. Ultrastructurally, both neoplastic patterns were evident, and they shared some, but not all, features. The shared features included intracellular spaces lined by cytoplasmic projections, deeply indented nuclei, tight desmosomal contacts, and cytoplasmic microfilaments. The fact that both tumors arose from the tunica vaginalis, coupled with their many ultrastructural similarities and intimate association, suggests that both were of mesothelial origin. (15 refs)

79-2898 Characteristics of Cell Cultures Derived From Hyperplastic Hemal Nodes (Meeting Abstract). (Eng) Amborski, G. F. (Louisiana State Univ., Sch. Veterinary Medicine, Baton Rouge, LA, 70803); Amborski, R. L.; Seger, C. L. *In Vitro* 15(3): 196; 1979. (no refs)

79-2899 Studies on Metastatic Spread of Primary Tumours (Meeting Abstract). (Eng) Tarin, D. (Dept. Histopathology, Royal Postgraduate Medical Sch., Hammersmith Hosp., London, England); Price, J. E. *Br J Cancer* 39(4): 476; 1979. (no refs)

See also:

- *(Rev.): 79-2417, 79-2418, 79-2445, 79-2446, 79-2447, 79-2451, 79-2452, 79-2454, 79-2458, 79-2459, 79-2460, 79-2461, 79-2464, 79-2465, 79-2467.
- *(Chem.): 79-2492, 79-2497, 79-2498, 79-2523, 79-2526, 79-2527, 79-2541, 79-2542, 79-2543, 79-2548, 79-2549, 79-2563, 79-2574, 79-2581, 79-2589, 79-2592, 79-2593, 79-2597, 79-2600, 79-2603, 79-2606, 79-2610, 79-2611, 79-2612, 79-2614, 79-2615, 79-2616, 79-2625, 79-2634, 79-2638, 79-2639, 79-2644, 79-2646, 79-2649, 79-2655, 79-2656, 79-2659, 79-2662.
- *(Phys.): 79-2670, 79-2676, 79-2677, 79-2679, 79-2680, 79-2684, 79-2689, 79-2690, 79-2691, 79-2693.
- *(Viral): 79-2720, 79-2744, 79-2754, 79-2766, 79-2768, 79-2782, 79-2784, 79-2786, 79-2804.
- *(Immun.): 79-2815, 79-2816, 79-2821, 79-2822, 79-2825, 79-2827, 79-2828.
- *(Epid-Biom.): 79-2901, 79-2907, 79-2908, 79-2909, 79-2915, 79-2927, 79-2932, 79-2938, 79-2939, 79-2958, 79-2959, 79-2965, 79-2966, 79-2970.

EPIDEMIOLOGY AND BIOMETRY

- 79-2900 Pregnancy, Breast-Cancer Risk and Maternal-Fetal Genetics (Letter to Editor).** (Eng) Doll, R. (Dept. Regius Professor Medicine, Radcliffe Infirmary, Oxford, England). *Lancet* 1(8115): 559; 1979.

An alternative explanation for the observation that the proportion of women who are single is less among those with breast cancer (BC) than among those with other types of cancer (OC) between 20 and 24 yr of age is given. At young ages the marriage rate is appreciable. The incidence of BC under 40 yr of age increases more rapidly with age than the incidence of OC. The proportion of BC patients who are single will be less than that of patients with OC when women of marriageable age are grouped together in 5-yr age groups. As the marriage rate declines, this effect gradually disappears, until the greater incidence among single women becomes evident. (1 ref)

- 79-2901 Histologic Comparison of Mammary Carcinomas among a Population of Southwestern American Indian, Spanish American, and Anglo Women.** (Eng) Black, W. C. (Cancer Res. and Treatment Center, Univ. New Mexico, 900 Camino De Salud, N. E., Albuquerque, NM, 87131); Bordin, G. M.; Varsa, E. W.; Herman, D. *Am J Clin Pathol* 71(2): 142-145; 1979.

Primary carcinomas of the breast were studied in age-matched New Mexican women of Southwestern American Indian, Spanish American, and Anglo ancestry. The women were from an area served by the New Mexico Tumor Registry. The principal tumor pattern in each group was infiltrating ductal carcinoma. The remaining histologic tumor types were similar except for an increased frequency of lobular carcinoma among the Anglo women compared with the other groups. The carcinomas had an irregular, stellate pattern (as opposed to a circumscribed pattern) in 36/45 Indian patients, 39/45 Spanish American patients, and 36/45 Anglo patients. The nuclear grade was most often I among the Indian women and most often II among the other groups. The cellular inflammatory response to tumor was generally absent-to-negligible or slight-to-moderate in all groups. Intraductal carcinoma was present within the substance or margin of the tumors in 23 Indian women, 23 Spanish American women, and 31 Anglo women. Indian women presented with a less favorable tumor stage at diagnosis compared with the other groups. The data do not demonstrate clearly defined differences in the incidence of specific histologic types of mammary carcinoma among the populations studied. (10 refs)

- 79-2902 Dietary Hypotheses Concerning the Etiology of Human Breast Cancer.** (Eng) MacMahon, B. (Dept. Epidemiology, Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA, 02115). *Nutr and Cancer* 1(2): 38-41; 1979.

Epidemiologic and experimental evidence for an association between diet and human breast cancer (BC) is presented, and some hypotheses on the role of diet in the etiology of BC are presented. The three principal features of the distribution of BC in humans are the striking geographic variation in incidence and mortality, its inverse association with age at first pregnancy, and a cluster of associations pointing to a critical role for ovarian activity in the genesis of the disease. Although there is no direct evidence of an influence of specific dietary factors on BC, some such factor is probably involved, as indicated by the overall geographic correlation between BC risk and food consumption and by the association of risk with body wt. Several studies have found a positive correlation between fat consumption and BC incidence. Mammary carcinogenesis in rats and mice is inhibited by calorie restriction and enhanced by high-fat diets. The mechanism of action of the high-fat diet is not known, but the possibility of alteration of hormonal states through dietary fat is repeatedly stressed. Considerable evidence points to the importance of the childhood years in determining BC risk. It is suggested that specific dietary factors associated with age at menarche may be the same factors that are the early determinants of BC risk. Studies relating BC risk to a particular pattern of estrogen metabolism have found substantial differences between high- and low-risk populations. These hormone patterns are determined in part by diet, and the specific factors should be determined. (25 refs)

- 79-2903 Opening Address.** (Eng) Mogren, H. (AB Marabou, Sundbyberg, Sweden). *Nutr and Cancer* 1(2): 26; 1979.

The opening address to the sixth Marabou (Sweden) international nutrition symposium dealing with the roles of food, diet, and nutrition in the causation and prevention of cancer is presented. These factors appear to be linked to approx 40% of all cancers in men and 60% of all cancers in women. The role of nutrition in cancer is not well understood. Eating patterns may play a major role in determining the risks of developing certain types of cancer, such as cancer of the breast, prostate, colon, and stomach. Many epidemiologists and nutritionists believe that a large proportion of these cancers may be the result of a diet too rich in total calories, saturated fats,

cholesterol, and alcohol. Instead of presenting basic facts and developments in nutrition, newspapers and magazines too often prefer to sound the alarm and to reproduce quasi-scientific curiosities. It is important to make the point that diet, not any single food, is important in good nutrition. (no refs)

- 79-2904 Health Problems of Anaesthetists and Their Families in the West Midlands.** (Eng) Tomlin, P. J. (Univ. Dept. Anaesthetics, Queen Elizabeth Hosp., Birmingham B15 2TH, England). *Br Med J* 1(6166): 779-784; 1979.

The occurrence of congenital anomalies among the children of 314 anesthetists in the West Midlands region (10% of the anesthetists in England and Wales) was studied. The children born to 60 anesthetists during periods when they were not engaged in anesthetic practice served as controls. Thirty percent of the anesthetists had problems related to unexpected infertility or abortion. The abortion rate was 9.8% in the control group, compared with 18.2% in families in which either parent was an anesthetist ($p < 0.05$). Girls with anesthetist parents were born underweight; boys were not similarly affected. Of the children born to anesthetists, 9.3% had congenital abnormalities or developmental problems, compared with 4.3% of the control group. A significantly greater proportion of girls had major clinical disorders. There was an appreciable excess of locomotor disorders compared with the rate in the general population. Three children had apparent neurological problems, two developed tumors (lymphoma, ependymoma), and a third developed leukemia. Breast cancer occurred in 2/76 female anesthetists and in the wife of an anesthetist; the wife, a dentist, regularly gave dental anesthetics. Six of the 238 male anesthetists also developed cancer (1 lung, 1 rectal, 4 skin cancers). (34 refs)

- 79-2905 Breast Cancer among Atomic Bomb Survivors. The Relationship of Prognosis to Pathologic Findings.** (Eng) Tokunaga, M. (Second Dept. Pathology, Sch. Medicine, Kagoshima Univ., Kagoshima, Japan). *Acta Pathol Jpn* 29(2): 197-209; 1979.

The relationship between intensity of exposure to ionizing radiation, pathologic findings, and prognosis was studied among 360 breast cancer patients living in Hiroshima or Nagasaki at the time of the bomb (ATB) or afterward (not in the city: NIC). The patients were classified by exposure dose into four groups: (1) NIC + 0 rads (nonexposed), (2) 1-49 rads, (3) 50+ rads, and (4) exposure dose unknown. The incidence of breast cancer was significantly higher among Group 3 women than among those in Groups 1 and 2. The 5- and 10-yr survival rates were highest in Group 3 and lowest in Group 2. The 5-yr survival rates tended to decrease gradually with age ATB in Groups 1 and 2, but not in Group 3. There were no statistically significant relationships between radiation dose and histologic type or size distribution

of the tumors. In Groups 1 and 2, 5-yr survival rates were considerably higher among patients with tumors < 5 cm in size than among those with tumors ≥ 5 cm; this relationship between tumor size and prognosis was reversed in Group 3. The prognosis was better in Group 3 than in the other groups, regardless of histologic grade of tumor. The cellular reaction in the carcinoma stroma was significantly greater in Group 3 than in the other groups, and prognosis tended to be better in patients showing a marked cellular reaction. The data suggest that radiation may have both a cancer-inducing effect and a role in the cellular immune reaction against cancer tissue. (23 refs)

- 79-2906 Comparative Clinicopathological Study of Breast Cancer among Japanese and American Women.** (Jpn) Sakamoto, G. (Dept. Pathology, Cancer Inst., Japanese Foundation Cancer Res., 1-37-1 Kami-Ikebukuro, Toshima-ku, Tokyo 170, Japan); Sugano, H.; Hartmann, W. H. *Jpn J Cancer Clin* 25(3): 161-170; 1979.

Breast cancer patients who underwent radical mastectomies during 1956-1974 at the Cancer Institute of Health (CIH) in Tokyo, Japan (2,604 women), and during 1956-1976 at the Vanderbilt University Hospital (VUH) in Nashville, Tennessee (755 women), were compared with respect to histologic type of tumor and 10-yr survival rates (patients seen between 1956-1967 only). The incidence of infiltrative and noninfiltrative lobular cancer was higher in the VUH group (21 and 68 patients, respectively) than in the CIH group (7 and 54, respectively). At the time of surgery, the American women were older than the Japanese women: 54.3% were 30-59 yr old vs 83.4% of the Japanese women, and 45.5% were > 60 yr old vs only 15.2% of the Japanese women. The overall 10-yr survival rate was 63.8% for the CIH and 46.9% for the VUH patients. With respect to lymph node metastasis, the critical point in 10-yr survival rates for Japanese women was the involvement of more than three to four axillary lymph nodes, and the critical point for American women was the presence of any lymph node involvement. For American women the 10-yr survival rate was significantly lower for postmenopausal (34.1%) than for premenopausal women (61.3%), but the survival rates for pre- and postmenopausal Japanese women were the same. (12 refs)

- 79-2907 Estimated Risk and Occurrence of Breast Cancer in Asymptomatic and Minimally Symptomatic Patients.** (Eng) Egan, R. L. (Mammography Section, Emory Univ. Clinic, 1365 Clifton Road, N.E., Atlanta, GA, 30322). *Cancer* 43(3): 871-877; 1979.

Mathematical procedures, some unique to this study, were applied to 114 suggested breast cancer risk factors, with as many as 10 subsets, using data obtained from historical physical, and x-ray examinations of 7,252 patients performed at

a clinic from 1963 to 1977. There were 30,904 individual breast examinations. Biopsies of 3,733 breasts revealed 713 primary breast carcinomas (exclusive of lobular carcinoma in situ) and 3,020 benign lesions. One-fifth of the cancers were unrelated to symptoms; 82% were free of axillary lymph node metastasis. There was no sign or symptom that predicted pre-clinical cancer. Interaction of numerous indicators subjected to strong statistical procedures could contribute to establishing the risk of even early breast cancer. The results of hierarchic discriminant analyses demonstrated the feasibility of using simultaneously large numbers of risk factors in a systematic way to pinpoint patients with mammary cancer. Based on usual clinical and x-ray assessment of the women, 12.5% of the noncancer patients required biopsy to demonstrate 70% of the cancers with a cancer to benign rate of 1 to 4. Using the same data with discriminant analyses, 5.6% of the patients would require biopsy, at the rate of 1 cancer to 1.8 benign lesions; 92% of the cancer patients could be placed in 11.9% of the population. A computerized system has been developed for widespread application to provide the clinician with a highly objective and totally consistent assessment of risk for breast cancer in each patient. (5 refs)

- 79-2908 Adolescent Breast Masses in Nigerian Igbo.** (Eng) Onuigbo, W. I. (FRCPath, PMB 1098, Enugu, Nigeria). *Am J Surg* 137(3): 367-368; 1979.

A total of 137 adolescent Nigerian Igbo (128 females and 9 males aged 11-20 yr) with breast masses were identified through surgical biopsy records dating back to 1970. In seven of the nine males, the histologic diagnosis was gynecomastia. Of the other two, one had an onchocerca worm granuloma on a muscle and the other had a neurofibroma. Of the 128 female patients, 101 were diagnosed as having fibroadenoma, 19 dysplasia, 5 sarcoma, and 1 each adenocarcinoma, abscess, and lymph node mass. Alveolar soft part sarcoma is a rare tumor but was encountered in two girls (aged 15 and 18 yr). This series differed from an American series in that the peak age for gynecomastia was later in adolescent Igbo and the peak age for fibroadenoma was later in adolescent Americans. These patterns may have a hormonal basis. (12 refs)

- 79-2909 ABO Blood Groups and Rhesus Factor in Patients with Tumors and Tumorlike Lesions of the Ovaries.** (Rus) Rybalka, A. N. (Dept. Obstetrics and Gynecology, Crimean Medical Inst., Simferopol', USSR); Andreev, P. V.; Tikhonenko, L. F.; Koval'chuk, N. A. *Vopr Onkol* 25(3): 28-30; 1979.

The distribution of blood groups ABO and the Rhesus (Rh) factor was determined in a group of 1,750 women with various tumors and tumorlike lesions of the ovaries. There was a decreased frequency of group O (16.1% vs 32.1% in healthy donors) and an increased frequency of group AB (25.8% vs 8.6%) among patients with mucinous tumors. Among pa-

tients with the cysts of the corpus luteum, there was a decreased frequency of group O (24.1%) and an increased frequency of groups A (45.6% vs 39.6%) and B (22.8% vs 19.7%). Among patients with endometrioid tumors, there was a decreased frequency of group A (32.7%) and an increased frequency of groups O (36.5%) and AB (15.4%). There was a decreased frequency of group B (12.5%) among patients with malignant tumors. The occurrence of the Rh-negative phenotype was significantly decreased among almost all patients with tumors and cysts of the ovaries (3.0%-7.48%), but not among patients with mucinous and malignant ovarian tumors (16.1% and 14.1%, respectively). (9 refs)

- 79-2910 Long-Term Estrogen Use and Endometrial Carcinoma (Letter to Editor).** (Eng) Obrink, A. (Dept. Gynecologic Oncology, Radiumhemmet, S-104 01 Stockholm, Sweden); Collen, J.; Bunne, G.; Tjernberg, B. *Acta Obstet Gynecol Scand* 58(1): 123; 1979.

Analysis of 622 patients with endometrial adenocarcinoma and 1,428 age-matched controls demonstrated a progressive increase in the number of women taking noncontraceptive estrogens for > 6 mo in both groups. Among the patients, the number increased from 8.1% in 1974 to 35.6% in 1977; these values for the controls were 6.6% and 16.7%. Further analysis revealed that between 1976 and 1977, short-term use (< 3 yr) was similar in the controls and patients, but long-term use (3-6 yr) was more than five times as common among the patients. (no refs)

- 79-2911 Assessing the Risks from Menopausal Estrogen Use: What Can We Learn from Trends in Mortality from Uterine Cancer?** (Eng) Weiss, N. S. (Dept. Epidemiology, Sch. Public Health and Community Medicine, Univ. Washington, Seattle, WA, 98195). *J Chronic Dis* 31(12): 705-708; 1978.

Errors in estimating endometrial cancer mortality rates and the application of mortality data to assessing the risks from menopausal estrogen use are surveyed. The best measure of endometrial cancer mortality in the US that is routinely available is the sum of the following two cause-of-death categories, cancer of the corpus uteri and uterine cancer not otherwise specified (NOS). However, the ability to document a trend in endometrial cancer mortality by tabulating deaths in these categories is limited by the fact that cervical cancer deaths, which once constituted a substantial proportion of deaths assigned to uterine cancer NOS, are with time probably contributing less and less to this category. Nevertheless, endometrial cancer mortality probably has had a true decline during the 1950's and 1960's, most likely as a result of increasingly early recognition and treatment of the disease. It is uncertain whether the spurious or the real reasons for declining mortality from corpus cancer + uterine cancer NOS

are continuing into the late 1970's. Thus, it is difficult to correlate time trends in uterine cancer mortality with the increased prevalence of estrogen use. Failure to observe an increase in the apparent mortality rate might only mean that the rise in estrogen-associated deaths was not enough to compensate for the fall that was otherwise taking place. These uncertainties could be clarified by a contemporary review of deaths assigned to uterine cancer NOS to determine their "true" nature and by obtaining data regarding the volume of sales of commonly used estrogen preparations. (13 refs)

- 79-2912 Occupation and Prostatic Cancer. A Review and Retrospective Analysis Based on Death Certificates in Two California Counties.** (Eng) Ernster, V. L. (Dept. Epidemiology and International Health, Univ. California, San Francisco, CA, 94143); Selvin, S.; Brown, S. M.; Sacks, S. T.; Winkelstein, W.; Austin, D. F. *J Occup Med* 21(3): 175-183; 1979.

Possible relationships between occupation and prostatic cancer (PC) were examined by comparing the occupations of men who died of PC in two northern California counties, Alameda (368 PC deaths) and San Francisco (334 deaths), with controls matched for sex, age, race, date of death, and county of residence. The data are presented as the mean odds ratio, which is an expression of the risk of being employed in a specific occupation/industry associated with cases compared with controls, and as a comparison of the observed number of PC deaths for each occupation/industry with the expected number. The data were analyzed separately for the two counties. Of 28 occupations that were sufficiently represented for consideration in both counties, elevated odds ratios were found for bookkeepers, shipping and receiving clerks, compositors and typesetters, and shipfitters. Of the 21 industries sufficiently represented in both counties for cross-county comparisons, elevated odds ratios were confirmed for horticultural services; newspaper publishing and printing; motor vehicle dealers; drug stores, drugs, chemicals, and allied products; and miscellaneous personal services. This study tentatively supports the possibility that selected occupations and industries have an excess risk for the development of PC. (23 refs)

- 79-2913 Prostate Cancer Epidemiology: Widowerhood and Cancer in Spouses.** (Eng) Greenwald, P. (Div. Epidemiology, New York State Dept. Health, Tower Building, Empire State Plaza, Albany, NY, 12237); Kirmss, V.; Burnett, W. S. *J Natl Cancer Inst* 62(5): 1131-1136; 1979.

A study was made to determine whether the duration of widowerhood (WHD) among men with prostate cancer differs from that of matched controls. Duration of WHD and the incidence of cancer deaths in spouses were studied in 169 widowers dying with prostate cancer, 451 matched control widowers dying of any causes except prostate cancer, and 150

matched control widowers dying of other types of cancer. The av duration of WHD and frequency of multiple wives were the same for patients with prostate cancer and controls. The incidence of breast cancer in predeceased wives (ie, wives dying before their husbands) of the prostate cancer patients was 9.9%, compared to with 5.8% in predeceased wives of the controls. The respective endometrial cancer rates for predeceased wives of patients and controls were 2.0% and 0.2%. Neither these results nor the results from a confirmatory study of cancer among wives of married prostate cancer patients were statistically significant. Overstatement of WHD status on 6.1% of death certificates, lack of case-control differences in durations of WHD, and decreasing widower-married mortality ratios as age-specific groupings were made smaller indicated that the high rates of prostate cancer among widowers may be due to artifacts of classifying and tabulating vital records. (12 refs)

- 79-2914 Descriptive Epidemiology of Testicular and Prostatic Cancer in Los Angeles.** (Eng) Ross, R. K. (Dept. Community and Family Medicine, Univ. Southern California Sch. Medicine, 2025 Zonal Ave., Los Angeles, CA, 90033); McCurtis, R. W.; Henderson, B. E.; Menck, H. R.; Mack, T. M.; Martin, S. P. *Br J Cancer* 39(3): 284-292; 1979.

Data from the Los Angeles County Cancer Surveillance Program (CSP) for the years 1972-1975 were used to study the descriptive epidemiology of testicular and prostatic cancer. The very high black/white ratio and late age peak of prostatic cancer contrasted sharply with the very low ratio and early age peak of testicular cancer. However, both sites had higher rates among upper occupational and social class groupings. Available descriptive and analytical research suggests that the etiology of prostatic cancer is most probably related to hormonal influences rather than to a horizontally transmitted agent, but the etiology of testicular cancer is most probably related to endogenous or exogenous hormonal influences in utero or in infancy or to in utero exposure to other exogenous agents. (61 refs)

- 79-2915 Epidemiologic Association Between Endemic Nephropathy and Urinary System Tumors in an Endemic Region.** (Eng) Stoyanov, I. S. (Inst. Oncology, Medical Acad., Sofia 1156, Bulgaria); Chernozemsky, I. N.; Nicolov, I. G.; Stoichev, I. I.; Petkova-Bocharova, T. K. *J Chronic Dis* 31(12): 721-724; 1978.

All cases of endemic nephropathy (EN) and/or malignant urinary tract tumors occurring in 27 villages of the Vratza district of Bulgaria between 1965 and 1974 were analyzed. Fifteen villages had a high EN incidence, and 12 (controls) were nonendemic. In the former group, 645 cases of EN and 177 cases of urinary tract tumors were recorded, whereas the corresponding figures in the control villages were 14 and 30, respectively. Of the cancer patients, 44.6% had concurrent

EN. EN was registered first in 62.8%. The highest risk for urinary tract cancer occurred in patients with EN, followed by persons living in endemic villages but unaffected by EN. Patients with EN had an 88-fold higher risk of urinary tract cancer than persons living in control villages. The data suggest that there is a common etiologic factor(s) in EN and urinary tract cancer. (8 refs)

- 79-2916 Cancer Risks Associated with Employment in the Leather and Leather Products Industry.** (Eng) DeCoufle, P. (Environmental Epidemiology Branch, NCI, Bethesda, MD, 20014). *Arch Environ Health* 34(1): 33-37; 1979.

A recent study of relationships between occupation and cancer at Roswell Park Memorial Institute, Buffalo, New York, identified significantly high risks of bladder cancer among men and women with a history of employment in plants manufacturing leather and leather products. Eleven men and four women with bladder cancer had been employed between 1956 and 1965 in these plants, and their relative risks were estimated to be 6 and 4 times those of clerical workers, respectively. Among the men, the relative risk increased with duration of employment and remained elevated after adjustment for smoking habits. Male leatherworkers also had significantly increased risks for cancers of the buccal cavity and pharynx and larynx that could not be explained by differences in smoking habits. Malignant lymphomas were also associated with elevated risks among men and women who had worked in the leather industry. A review of processes and agents found in leather manufacture revealed several steps that involved exposure to potentially carcinogenic materials, including tannin extracts and tannic acid plus azo and other synthetic dyes, that have induced cancer in laboratory animals. There should be further studies of persons employed in the leathermaking and fabrication industries to characterize the nature of exposure-response relationships. (30 refs)

- 79-2917 Primary Liver Cancer and Occupation: Brazoria County, Texas (Meeting Abstract).** (Eng) Divine, B. J. (Univ. Texas Health Science Center, Houston Sch. Public Health, Houston, TX). *Diss Abstr Int [B]* 39(10): 4827; 1979. (no refs)

- 79-2918 Hepatocellular Carcinoma and Hepatitis B Virus Markers in Europe (Letter to Editor).** (Eng) Vargas, V. (Dept. Internal Medicine, Ciudad Sanitaria de la Seguridad Social, Universidad Autonoma, Barcelona, Spain); Pedreira, J. D.; Vilaseca, J.; Ruiz, J.; Esteban, R.; Hernandez, J. M.; Guardia, J.; Bacardi, R. *Lancet* 1(8118): 721-722; 1979.

Hepatitis B virus markers were found to correlate with the

appearance of hepatocellular carcinoma in both cirrhotic and normal livers in an area of Spain where the carrier rate for hepatitis B surface antigen is lower than that in Greece, Asia, or Africa, but higher than that in Britain or the US. (1 ref)

- 79-2919 Important Epidemiological Considerations and Problems in Evaluating Health Effects of Chemicals (Meeting Abstract).** (Eng) Discher, D. P. (Dept. Industrial and Environmental Medicine, San Jose Medical Clinic, 45 South 17th St., San Jose, CA, 94115). *Tex Rep Biol Med* 37: 94; 1978. (no refs)

- 79-2920 Dose-Response Relationship of Neutrons and γ Rays to Leukemia Incidence among Atomic Bomb Survivors in Hiroshima and Nagasaki by Type of Leukemia, 1950-1971.** (Eng) Ishimaru, T. (Radiation Effects Res. Foundation, Hiroshima, Japan); Otake, M.; Ichimaru, M. *Radiat Res* 77(2): 377-394; 1979.

The incidence of leukemia during 1950-1971 in a fixed mortality sample of atomic bomb survivors in Hiroshima and Nagasaki was analyzed as a function of neutron and γ kerma and marrow doses. Two dose-response models were tested for acute leukemia (AL), chronic granulocytic leukemia (CGL), and all types of leukemia, respectively. Each model postulates that leukemia incidence depends upon the sum of separate risks imposed by γ and neutron doses. In Model I, the risk from both types of radiation is assumed to be directly proportional to the respective doses, but Model II assumes that whereas the risk from neutrons is directly proportional to the dose, the risk from γ rays is proportional to dose-squared. The dose response of the two types of leukemia was found to differ by type of radiation. The data suggested that the response of AL was best explained by Model II, but the response of CGL depended almost linearly upon neutron dose alone, because the regression coefficients associated with γ radiation for both Models I and II were not significant. With respect to AL, at least Model II, it appears that both kinds of radiation can induce this type of leukemia. Although both models fit the data for CGL, the induction of CGL appears principally to depend linearly upon neutron dose alone. If AL and CGL are considered together as all types of leukemia, Model II appears to fit the data slightly better than Model I, but neither model is statistically rejected by the data (25 refs)

- 79-2921 Childhood Cancer and Drugs in Pregnancy.** (Eng) Sanders, B. M. (Childhood Cancer Res. Group, Dept. Regius Professor Medicine, Univ. Oxford, Oxford OX1 9QG, England); Draper, G. J. *Br Med J* 1(6165): 717-718; 1979.

A study was carried out on 11,169 matched case-control pairs

of children (aged up to 15 yr) included in the Oxford Survey of Childhood Cancers to determine whether an association exists between cancer in children and drugs given to their mothers during pregnancy. The mothers of children who developed cancer reported about 25% more illness during pregnancy than mothers of healthy control children. The case:control ratio was particularly high for pulmonary tuberculosis and epilepsy, raising the possibility that the drugs (isoniazid and phenytoin) used in the treatment of these illnesses might be carcinogens. However, despite a slight excess of lymphoma in the children of these mothers, the investigation provided no real evidence of a transplacental drug effect in the etiology of childhood cancer. (6 refs)

79-2922 Electrical Wiring Configurations and Childhood Cancer. (Eng) Wertheimer, N. (Dept. Preventive Medicine, Univ. Colorado Medical Center, Box C-245, Denver, CO, 80262); Leeper, E. *Am J Epidemiol* 109(3): 273-284; 1979.

An excess of electrical wiring configurations suggestive of high-current flow was noted in Colorado in 1976-1977 near the homes of children who developed cancer, as compared with the homes of control children. The finding was strongest for children who had spent their entire lives at the same address, and it appeared to be dose-related. It did not seem to be an artifact of neighborhood, street congestion, social class, or family structure. The high-current configuration was associated with an excess of leukemia, lymphoma, and nervous system tumors. The reason for the correlation is uncertain; possible effects of current in the water pipes or of alternating current magnetic fields are suggested. (19 refs)

79-2923 Tumor Incidence in Human Allograft Recipients. (Eng) Penn, I. (Dept. Surgery, Veterans Admin. Hosp., 1055 Clermont St., Denver, CO, 80220). *Transplant Proc* 11(1): 1047-1051; 1979.

The most common types of tumors that arose de novo after transplantation in 693 patients and the management of transplant patients with preexisting renal neoplasms are discussed. Excluding patients with skin and lip cancers (277) and with carcinoma in situ of the uterine cervix (38), lymphomas occurred in 150/461 transplant patients (33% incidence vs a 3%-4% incidence in the general public). The types of cancer seen most frequently in the general public occur mostly in older age groups; their scarcity in transplant patients may be related to the youth of the recipients, whose average age was 40 and who developed their cancers an average of 38 months after transplantation (range, 1-158 months). The predominant type of lymphoma, reticulum cell sarcoma (93/150 lymphomas), was 350 times more common in renal transplant recipients than in the general population. The CNS was involved in an unusually large number of patients (54/130 patients with non-Kaposi's lymphomas). The risk of skin cancer in renal

transplant recipients was seven times greater than in the general public, with the excess consisting mostly of squamous cell carcinomas. A series of 96 patients with preexisting renal and ureteral tumors was studied. Of 37 patients with symptomatic tumors who underwent transplantation within 12 months after completion of treatment for their malignancies, 51% experienced recurrences or metastases. The corresponding figure for the 25 patients who received transplants > 12 months after anticancer treatment was 4%. Therefore, it is recommended that, whenever possible, transplantation be delayed for at least 1 year after completion of therapy. (7 refs)

79-2924 Epidemiology of Hodgkin's Disease. (Hun) Istvan, L. (Haematologiai Osztaly, Markusovszky Korhaz-Rendelőintézet, Szombathely, Hungary); Gicz, S. *Orv Hetil* 120(6): 319-324; 1979.

An epidemiologic analysis of 166 cases of Hodgkin's disease (HD) diagnosed in the county of Vas, Hungary, during 1953-1973, is presented. The patients included 107 men and 59 women. Compared with the occupational pattern of the general population, industrial workers and white-collar workers were slightly overrepresented among the patients. Although the yearly incidence of carcinoma as a whole nearly doubled during the 20-year period, there was no change in that of HD other than cyclic increases at 3- to 4-year intervals. The onset of the first symptoms peaked during the winter months. The incidence was highest in the age groups 15-40 years and 50-65 years. There was no correlation between the incidence of hepatitis and that of HD. A comparison with 20,000 blood donors, 569 patients with gastric carcinoma, 1,070 patients with gastric ulcer, and 114 patients with chronic lymphocytic leukemia (CLL) showed that the frequency of blood group O was significantly lower among the HD patients and that of A and AB was somewhat higher. The blood group distribution was nearly the same as in the CLL patients. The incidence of Rh D factor was lower in the blood donors and the gastric carcinoma patients, and it was similar in the HD and CLL patients. (84 refs)

79-2925 Abnormal Lymphocyte Responses in Residents of a Town with a Cluster of Hodgkin's Disease. (Eng) Plouffe, J. F. (Div. Infectious Diseases, Dept. Medicine, Ohio State Univ. Coll. Medicine, Columbus, OH, 43210); Silva, J.; Schwartz, R. S.; Callen, J. P.; Kane, P.; Murphy, L. A.; Goldstein, I. J.; Fekety, R. *Clin Exp Immunol* 35(2): 163-170; 1979.

The immunological status of residents of a small rural town in which there was a time-space cluster of Hodgkin's disease (7 cases over a 20-year period) was investigated. A large elevator for the storage of navy beans was located in the residential area which was frequently covered with dust from the elevator. The lymphocyte response of 40 healthy town residents,

12 of whom were members of a family with a Hodgkin's disease patient, to commercial phytohemagglutinin (PHA) and to several extracts of navy beans was assayed in vitro. The lymphocytes of these residents showed increased levels of transformation when incubated with navy bean extracts, compared with lymphocytes of nonresident controls. A PHA with the ability to stimulate lymphocytes was isolated from navy beans and characterized. It had a mol wt (upon gel filtration) of 120,000, a subunit mol wt of 30,000, and it contained 7.8% neutral carbohydrate. The purified lectin agglutinated human RBC types A, B, and O. These preliminary results suggest that environmental mitogens may have significant effects on lymphocyte transformation of a chronically exposed population. Other investigations of time-space aggregates of Hodgkin's disease should include a search for environmental substances that may alter the immune system. (30 refs)

- 79-2926 Host Origin of Lymphomas in Organ Transplant Recipients.** (Eng) Penn, I. (Dept. Surgery, Univ. Colorado Medical Center, Denver, CO, 80262). *Transplantation* 27(3): 214; 1979.

Studies of six organ transplant recipients who developed lymphomas demonstrated that all the lymphomas were of host origin. This was determined by HLA typing of tumor cells and donor and host peripheral blood lymphocytes or by examining tumor cells for X chromatin or for the Y chromosome in cases in which the donor and recipient were of different sexes. Necropsy examination revealed no evidence of neoplasia in the donors, and it was unlikely that the lymphomas were present in the recipients before transplantation. (7 refs)

- 79-2927 Spontaneous Abortions in Sibship of Children with Congenital Malformation or Malignant Disease.** (Eng) Spira, A (Unite de Recherches Statistiques, Institut National de la Recherche Medicale, 94800 Villejuif, France); Lazar, P. *Eur J Obstet Gynaecol Reprod Biol* 9(2): 89-95; 1979.

Information about the incidence of spontaneous abortion (SA) in the sibship of 751 children with cancer/leukemia (CL), congenital heart disease (CHD), strawberry nevus (SN), or mental retardation (MR) was obtained. The number of children in the family and the proportion of mothers having had at least one other pregnancy differed significantly among the four groups, being lowest in the CHD and SN groups. The mean rate of SA's was significantly higher in the CHD and SN groups, and the rate of infant mortality due to congenital malformations or chromosomal aberrations was significantly higher in the CHD groups than in the other groups. The groups did not differ with respect to the gynecologic history of the mother, and this variable was not related to the incidence of SA. The SA rate was significantly in-

creased among mothers who had been exposed to ionizing radiation, compared with those who had not, and among mothers who smoked cigarettes. Irradiation or smoking habits in the fathers was not related to SA incidence. Even when maternal exposure to ionizing radiation and smoking was eliminated, the SA rate was significantly higher in the CHD and SN groups compared with the CL and SRC groups. It is hypothesized that the increased incidence of SA among mothers with CHD children might be a manifestation of a correlation between susceptibilities to certain anomalies of the karyotype and some types of congenital malformations. (18 refs)

- 79-2928 Benzene Exposure in the Rubber Coating Industry--A Follow-Up.** (Eng) Pagnotto, L. D. (Massachusetts Dept. Labor and Industries, Div. Occupational Hygiene, 39 Boylston St., Boston, MA, 02116); Elkins, H. B.; Brugsch, H. G. *Am Ind Hyg Assoc J* 40(2): 137-146; 1979.

A follow-up study was made of 38 workers in the rubber coating industry who were exposed to benzene concentrations of 5-90 ppm for 1-24 yr. One case of mild benzene poisoning was reported, but the worker recovered rapidly when he was transferred to a job that did not involve benzene exposure. Some of the other workers had blood pictures that were considered slightly abnormal. The Hb of one worker remained low (12.5 g) and exchanged from 1961 to 1977. However, no blood dyscrasias or leukemias were reported in the group since the termination of benzene exposure in 1964. Four workers retired early because of disabilities and three others died; one from lung cancer, another from heart disease, and the third from coronary sclerosis. The remaining workers were alive and free of serious health problems in 1977. In light of the apparently small margin of safety associated with the former American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) of 25 ppm benzene, the recent reduction to the current ACGIH TLV of 10 ppm seems reasonable. (9 refs)

- 79-2929 Oesophageal Cancer Studies in the Caspian Littoral of Iran: Results of a Case-Control Study.** (Eng) Cook-Mozaffari, P. J. (Medical Res. Council External Staff, Dept. Regius Professor Medicine, Univ. Oxford, Oxford, England); Azordegan, F.; Day, N. E.; Ressicaud, A.; Sabai, C.; Aramesh, B. *Br J Cancer* 39(3): 293-309; 1979.

A case-control study was conducted in northern Iran, where very high rates of esophageal cancer (EP) occur among both men and women, to study factors identified in a previous study as potentially causally related to EP. Information was obtained by questionnaire and by interview. Other tumors (lung, stomach, breast, large bowel, larynx, and pharynx) were included to distinguish findings specific for EP from the general characteristics of cancer patients and to verify that expected associations, such as between lung cancer and ciga-

rette smoking, would emerge under the prevailing field conditions. Each patient had two controls matched for village of residence, age, sex, and language group. Reinterviewing was performed to a limited extent to assess the accuracy of replies to questionnaires. The following were found not to be associated with EC: consumption of sheep's milk, yoghurt, wild spinach, and sesame oil; chewing of nass (tobacco and lime mixture), a habit confined to men; carpet-making; consumption of a special foodstuff (crushed pomegranate seeds, pepper, and raisins) during pregnancy; and salting and sun-drying of meat. The use of opium, bread, and tea could not be assessed in the retrospective framework. Strongly associated with EC risk were low socioeconomic status and a low intake of fresh fruits and vegetables. The two factors had an independent effect, and they were more marked for EC than for the other tumors. (13 refs)

- 79-2930 Esophageal Cancer and Alcohol Consumption: Importance of Type of Beverage.** (Eng) Tuyns, A. J. (International Agency Res. Cancer, 150 Cours Albert Thomas, 69372 Lyon Cedex 2, France); Pequignot, G.; Abbaticci, J. S. *Int J Cancer* 23(4): 443-447; 1979.

The role of alcohol consumption in esophageal cancer in Normandy, France, was assessed by a retrospective study of 312 male patients and 869 age-matched controls. The linear relationship between the logarithm of risk and overall daily alcohol consumption was confirmed after adjustment for tobacco. The role of each specific alcoholic beverage was further investigated by computing relative risks for individuals consuming a given beverage and for those drinking other beverages only, within each overall alcohol consumption category. It was concluded: (1) that there is a linear relationship between the logarithm of risk of esophageal cancer and overall daily ethanol consumption, whatever the beverage; (2) that the effect is more marked for strong beverages (ie, liqueurs, brandies) than for lighter beverages; and (3) that there is an additional risk related to apple brandy and cider. One possible reason for the extra risk related to cider and its distillates might be the presence of carcinogens in these beverages. (13 refs)

- 79-2931 Environmental Health Relationships Between Some Selected Physico-Chemical Parameters in Drinking Water and Cancer Morbidity in the Metropolitan Area of Minneapolis--St. Paul (Meeting Abstract).** (Eng) Rodriguez, G. (Univ. Minnesota, Minnesota, MN, 55455). *Diss Abstr Int [B]* 39(9): 4290-4291; 1979. (2 refs)

- 79-2932 Cancer in Universal and Left-sided Ulcerative Colitis: Clinical and Pathologic Features.** (Eng) Greenstein, A. J. (Dept. Surgery, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, NY, 10029);

Sachar, D. B.; Pucillo, A.; Vassiliades, G.; Smith, H.; Kreel, I.; Geller, S. A.; Janowitz, H. D.; Aufses, A. H. *Mt Sinai J Med (NY)* 46(1): 25-32; 1979.

Cancer developed in 30/267 ulcerative colitis patients admitted to one hospital between 1960 and 1976. The clinical records of these 30 patients were examined to elucidate the clinical and pathologic features of the malignancies. There were 26 patients with 29 colorectal cancers (CRC's), and 4 patients with extraintestinal cancers (adenocarcinoma of the bladder, basal cell carcinoma of the skin, Hodgkin's disease of the lymphocyte-depletion type, and diffuse histiocytic lymphoma involving the lymph nodes of the right groin). The CRC's in this series were more often multiple and more proximal in distribution than colon cancers in the noncolitis population. Signs specifically associated with CRC included a palpable abdominal mass, intestinal stricture and obstruction, and late recrudescence of symptoms. Twenty-one of the 26 patients with CRC had universal colitis and 5 had left-sided disease. Compared with a standard population, the observed-to-expected ratio of CRC incidence among all the ulcerative colitis patients was 8, with the ratio being 11 in universal colitis and 3 in left-sided disease. It is important to recognize that after a sufficiently long interval, cancer may develop in left-sided as well as in universal ulcerative colitis. The mortality from CRC was 50% at 18 mo, with 10/13 deaths being due to metastases. There were 10 5-yr survivors. Long-term survival was not limited to patients with subclinical cancers, since 8/9 5-yr survivors studied by barium enema had positive radiologic findings preoperatively. The long-term mortality from colitis-associated cancer did not appear to be worse than that from CRC in the general population. (32 refs)

- 79-2933 Association Between Chloroform Levels in Finished Drinking Water Supplies and Various Site-specific Cancer Mortality Rates.** (Eng) Hogan, M. D. (Biometry Branch, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC, 27709); Chi, P. Y.; Hoel, D. G.; Mitchell, T. J. *J Environ Pathol Toxicol* 2(3): 873-887; 1979.

Statistical and biological problems of the indirect approach to assessment of the impact of environmental agents on public health are illustrated using the potential association between various site-specific cancer mortality rates and chloroform (CF) levels in finished drinking water supplies. Both unweighted and weighted regression procedures were used to determine to what extent (if any) the results might depend on the weighting scheme adopted. CF exposure data were collected in 1975, but the cancer mortality rates with which they were being associated were for 1950-1969. Given the long cancer latent period, the most relevant exposure data would be those from 1925-1959. Even if the available exposure measurements were consistent over long periods of time, the CF readings might still not be representative of the exposure history of the total population at risk. One of the most

significant factors affecting the results of any general linear regression analysis is selection of the explanatory variables. In this study, the issue of cancer latency period was ignored, and data were lacking on certain risk factors, such as cigarette smoking. Within these restrictions, there was some correlation between CF levels in finished drinking water supplies and cancer mortality for specific sites such as the bladder and rectum-intestine. Associations such as these, however, may not be very meaningful and need to be interpreted with great care. (13 refs)

79-2934 Is the Colon Cancer Incidence in Some Eastern Nebraska Counties the Highest in the World? (Meeting Abstract). (Eng) Mahboubi, E. (Eppley Inst Res. in Cancer, Univ. Nebraska Medical Center, Omaha, NE, 68105); Sayed, G.; Shubik, P. *Proc Am Assoc Cancer Res* 20: 226; 1979. (no refs)

79-2935 Epidemiological Analysis of the Incidence of Cancers of the Gastrointestinal Tract in a Well-defined Population (Meeting Abstract). (Fre) Faivre, J. (Registre Bourguignon des Cancers Digestifs, 7, boulevard Jeanne-d'Arc, F 21033 Dijon Cedex, France); Legoux, J. L.; Martin, F.; Klepping, C. *Gastroenterol Clin Biol* 3(1): 88; 1979. (no refs)

79-2936 Stomach Cancer Mortality in the North Central States: High Risk Is Not Limited to the Foreign-Born. (Eng) Kriebel, D. (Center Biology Natural Systems, Washington Univ., Box 1126, St. Louis, MO, 63130); Jowett, D. *Nutr and Cancer* 1(2): 8-12; 1979.

Ethnicity, water supply, socioeconomic status, and urbanization were examined as possible stomach cancer factors among all 174 counties of Minnesota, Wisconsin, and upper Michigan. Multiple regression analysis was used. The proportion of residents born in northern Europe best explains the pattern of stomach cancer mortality in these counties. The settling patterns of Finns, and to a lesser extent of Poles, Norwegians, Danes, and Swedes, are strongly associated with county stomach cancer rates. Socioeconomic status and water supply are less significant. Disregarding socioeconomic status and water supply factors, estimated stomach cancer mortality for these populations is still disproportionately high. Other researchers have identified diet as a major risk factor in stomach cancer and as the prime cause of the ethnicity-stomach cancer association. It is suggested that the natives shared the stomach cancer risk, perhaps by adopting the "high-risk" diet of the foreign-born. (22 refs)

79-2937 Incidence of Stomach Cancer in the Cote d'Or Region of France. Results of Two Years of Sys-

tematic Registration of a Well-defined Population. (Fre) Faivre, J. (Registre Bourguignon des Cancers Digestifs, Faculte de Medecine, 7, boulevard Jeanne-d'Arc, 21033 Dijon Cedex, France); Legoux, J. L.; Martin, F.; Cabanne, F.; Klepping, C. *Gastroenterol Clin Biol* 2(12): 967-972; 1978.

Statistics on stomach cancer obtained from a cancer registry in the Cote d'Or region of France (455,727 inhabitants) are presented. During 1976 and 1977, stomach cancer represented 19% of the reported 913 cancers of the gastrointestinal (GI) tract. The crude annual incidence rate was 24.8/100,000 for men and 13.7/100,000 for women. Stomach cancer was the second most prevalent GI tract cancer in men, ranking after cancer of the rectum, and the third most prevalent GI tract cancer in women, ranking after cancer of the colon and rectum. Histological diagnosis, which was possible in 137 of the cases, showed that 69 were adenocarcinomas and 41 were anaplastic carcinomas. Av age at diagnosis was 70.9 yr in men and 72.7 yr in women. Among the 111 men with stomach cancer, 10 had undergone surgery > 20 yr previously for ulcers (9 partial gastrectomies, 1 gastroenteroanastomosis). In the 2-yr period analyzed, there were 148 deaths due to stomach cancer, representing 25% of the GI tract cancer mortality. (19 refs)

79-2938 Gastric Polyps: Clinical Picture and Management (Meeting Abstract). (Eng) Fabry, T. L. (Dept. Medicine, Mount Sinai Hosp., New York, NY); Frankel, A.; Waye, J. D. *Gastroenterology* 76(5, part 2): 1129; 1979. (no refs)

79-2939 Gastric Polyps: Review of the Literature and Analysis of 37 Cases. (Spa) Esteva, E. A. (Servicio de Gastroenterologia, Hospital Nacional "Profesor Alejandro Posadas", Spain); Cosen, J. N. *Acta Gastroenterol Latinoam* 8(4): 249-258; 1978.

The clinical symptoms in 37 patients (18 men and 19 women, median age 65 yr) with gastric polyps were epigastralgia (17 patients, melena (14), anemia (17), emesis (10), asthenia (9), loss of wt (9), jaundice (4), diarrhea (3), ulcer (3), anorexia (3), dysphagia (2), and palpable tumor (2). Solitary polyps were found in 27 cases: 16 were sessile, 2 pedunculated, the others were not characterized. Twenty-one of these 27 tumors were < 2 cm, and 19 were localized in the antrum, 6 in the corpus, and 2 in the subcardiac region. Multiple polyps were found in 10 patients. Biopsy findings were available for 27 patients: chronic gastritis was found in 14, hyperplasia in 5, adenoma in 3, adenocarcinoma in 3, carcinoid tumor in 1, and lipoma in 1. In the 13 patients studied, gastric secretion was increased in 2, normal in 1, and reduced in 2, and achlorhydria was found in 8. (46 refs)

79-2940 Epidemiology of Gastric Cancer. (Eng) Tulinius, H. (Faculty Medicine, Univ. Iceland, P.O. Box 523, Reykjavik, Iceland). *Nutr Cancer* 1(2): 61-69; 1979.

The epidemiology of gastric cancer (GC) in Iceland and in the world is reviewed, and the etiologic hypotheses that have been formulated, particularly those concerned with the effect of diet, are considered. The incidence of GC has fallen over the last several decades both in Iceland and in the world, with few exceptions. The exceptions are mainly areas with very low GC frequency. Mortality from GC has also fallen during this same period. Genetics and immunological makeup appear to play a relatively small role in the etiology of GC. Nitrosamines, which can be produced both within the human body and by food processing, are potent chemical carcinogens and also are site-specific. Some nitrosamines cause adenocarcinoma in the glandular stomach of experimental animals. It is hypothesized that a large proportion of the GC risk is environmental and that this environmental effect is produced through diet. Case-control studies have shown negative associations between GC and vegetables, fruits, and milk; vitamin C is postulated as a protective factor. (33 refs)

- 79-2941 Stomach Cancer in Mali. Epidemiological, Clinical, and Therapeutic Data on 70 Cases.** (Fre) Guindo, A. (Ecole Nationale de Medecine et de Pharmacie du Mali, B.P. 1805, Bamako, Mali); Duflo-Moreau, B.; Dembele, M.; Daou, F.; Duflo, B. *Ann Gastroenterol Hepatol (Paris)* 15(1): 23-26; 1979.

Seventy cases of gastric cancer were detected in the capital city of Bamako, Mali, during an 18-mo period. The patients included 51 men and 19 women all black, aged 25-68 yr (av 49 yr). Most patients were in the lower socioeconomic level. A high-carbohydrate diet based on rice and millet and the consumption of dried smoked and unsmoked fish, homemade peanut butter, and foods with a high potash content were characteristic. Meat, fruit, and vegetable consumption was low. This diet, and especially components such as dried fish, potash, and, possibly, aflatoxin-contaminated peanut butter, are possible etiologic factors. The monotonous diet is likely to cause atrophic gastritis, which may reduce the defense capacity of the gastric mucosa against carcinogens. The 70 cases correspond to an incidence of 22.2/100,000 in men and 8.3/100,000 in women, but the real incidence is probably much higher (44.4-66.6/100,000 in men and 16.6-24.9/100,000 in women) because many cases are treated by quack doctors. (22 refs)

- 79-2942 Introductory Comments.** (Eng) Stare, F. J. (Dept. Nutrition, Harvard Sch. Public Health, Boston, MA). *Nutr and Cancer* 1(2): 37; 1979.

The great differences in the frequency of various types of cancer in different parts of the world indicate that some environmental factor, most likely diet, is responsible. High rates of cancer of the breast, prostate, and colon generally go together, suggesting a common cause (S). Countries with high rates of these cancers generally have low rates of stomach

cancer and vice versa, most likely indicating separate causes. Diet could affect cancer risk in at least three ways: (1) a dietary deficiency could either promote cancer or fail to offer some naturally occurring form of dietary cancer protection; (2) some direct cancer-causing agent, a food additive or a natural or artificial contaminant, could be eaten; and (3) specific excesses, eg, total calories, alcohol, saturated fats, and cholesterol, could be related to cancer. (no refs)

- 79-2943 Diet and Human Carcinogenesis with Special Reference to Colon Cancer.** (Eng) MacLennan, R. (Unit Cancer Epidemiology and Biostatistics, International Agency Res. Cancer, 150 Cours Albert-Thomas, F-69372 Lyon Cedex 2, France). *Nutr and Cancer* 1(2): 42-45; 1979.

Studies of the role of diet in human cancer, especially colon cancer (CC) are reviewed. Brewery workers in Dublin have a two-fold excess for rectal cancer but only a slight excess for CC, but such workers in Copenhagen show no excess in cancer at either site. Certain beverages, such as alcohol or hot tea, may increase the delivery of carcinogens. Specific dietary components may induce activating or inactivating enzymes and thus modify the response to carcinogen exposure. Nitrosamines and other nitroso compounds may be formed in vivo from ingested dietary precursors. Dietary fat and meat consumption are highly correlated with CC. Obesity has been associated with higher cancer risk, but the mechanism is not known. Dietary fiber may exert a protective effect against CC, and retinoids may inhibit the progression of premalignant epithelial lesions. The geographical variation in the incidence of CC suggests that environmental factors, rather than genetic factors, play the primary etiological role. The distribution of cancers within the large intestine also varies with incidence rate. It has been postulated that bile acids are metabolized to carcinogens that act in the colon and rectum. The amount of dietary fat determines the bile acid concentration and bacterial flora composition in the large intestine. These hypotheses are supported by results from studies of populations at high, medium, and low risk for CC. (23 refs)

- 79-2944 Detection, Localization, and Treatment of Occult Bronchogenic Carcinoma in Nickel Workers.** (Eng) Nelems, J. M. (Dept. Surgery, St. Paul's Hosp., Vancouver, B.C., Canada); McEwan, J. D.; Thompson, D. W.; Walker, G. R.; Pearson, F. G. *J Thorac Cardiovasc Surg* 77(4): 522-525; 1979.

A prospective analysis of sputum cytologic abnormalities occurring in 268 asymptomatic men who had been exposed to nickel sulfide in a sinter plant prior to 1963 was carried out during 1973 and 1974. Twelve men, 11 of them smokers and 1 a former smoker, had malignant cells in their sputum. Their chest radiographs were all normal. Two of these men refused further investigations; one presented 3.5 yr later with extensive squamous cell carcinoma of the right lower lobe and the

other presented 5 yr later with an extensive squamous cell tumor involving the right maxillary sinus. All the remaining asymptomatic men were subsequently found to have squamous cell carcinomas of the lung or larynx. One patient was treated with radiation therapy, and nine were treated via pulmonary resection. Two of the 10 patients died, 1 from recurrent carcinoma and 1 from postoperative hemorrhage. The remainder are alive and well an av of 36 mo postoperatively, and their sputum remains negative for malignant cells. All but one tumor showed invasive properties. The incidence of squamous cell tumors in this study was threefold higher than the incidence that would have been expected from subjects exposed only to cigarette smoke. Cancer related to industrial pollutants may behave differently from cancer associated with smoking alone. (14 refs)

79-2945 Lung Cancer Mortality and Residential Proximity to Industry (Meeting Abstract). (Eng)

Gottlieb, M. S. (Tulane Univ., Sch. Medicine, Public Health and Tropical Medicine, New Orleans, LA, 70112); Shear, C. L.; Seale, D. B. *Proc Am Assoc Cancer Res* 20: 197; 1979. (no refs)

79-2946 Trends in Respiratory System Cancer Mortality in Louisiana: Geographic Distributions in 1950-1969 and 1967-1976 Compared. (Eng)

Rothschild, H. (Dept. Medicine, Louisiana State Univ. Medical Center, New Orleans, LA, 70112); Voors, A. W.; Weed, S.; Vial, L. J.; Welsh, R. A.; Johnson, W. D. *Am J Public Health* 69(4): 380-381; 1979.

Parish (county)-specific mortality rates due to respiratory system cancers (RSC) in Louisiana were investigated for the period 1967-1976. The av annual mortality from RSC increased during the study period, compared with the period 1950-1969, and the increase was observed among whites and nonwhites of both sexes. The age-adjusted rate was much higher among males than females, but the rates for females increased more than those for males. Also, the rates for whites increased more than those for nonwhites. Of Louisiana's 64 parishes, the av annual mortality due to RSC was greater than expected in 19 and less than expected in 7. High mortality rates (in the upper quartile) among males were clustered in the southern part of the state, whereas those for females showed an apparently random geographic distribution. The data indicate that the high mortality rates from RSC in Louisiana are probably sufficiently stable over time to provide substantive data for study. (7 refs)

79-2947 Mortality Experience of Workers in a Vinyl Chloride Monomer Production Plant. (Eng)

Buffler, P. A. (Univ. Texas Sch. Public Health, P. O. Box

20186, Houston, TX, 77025); Wood, S.; Eifler, C.; Suarez, L.; Kilian, D. J. *J Occup Med* 21(3): 195-203; 1979.

A mortality follow-up study was conducted of 464 white men employed in a vinyl chloride monomer (VCM) production plant for at least two consecutive mo between 1948 and 1975. Eight of the 28 deaths in this group were due to malignant neoplasms, 4 from lung cancer. No angiosarcomas or other liver tumors were observed. The eight persons who died of cancer were initially exposed to VCM prior to 1963, and the four with lung cancer, prior to 1958. Six of the 28 deaths, including 2/8 cancer deaths, occurred among a subgroup of 165 workers exposed to 1,4-dioxane. The total number of observed cancer deaths was not significantly different than that expected, but a significant excess was noted for malignant neoplasms of the respiratory system. The effects of smoking, duration of exposure to VCM, and level of exposure and the combined effect of duration and level of exposure were analyzed separately. A 5 yr latency requirement was maintained for all analyses except for the smoking analysis. Using a minimum latency period of 5 yr from the date of initial exposure to VCM, the excess of respiratory cancer was moderate but not significant for the 314 employees satisfying this criterion. Both a longer duration and a higher level of exposure during the first 5 yr were associated with a significant excess of respiratory cancer. However when duration and level of exposure were combined, the results were not significant. In spite of the discrepancy in the results of dose-response analyses, the results suggest that a relationship exists between exposure to VCM and respiratory cancer. (27 refs)

79-2948 The Role of Vitamin A and Ascorbic Acid in Relation to Respiratory System Cancers (Meeting Abstract). (Eng)

Lopez-S, A. (Dept. Medicine, Louisiana State Univ. Sch. Medicine, New Orleans, LA); Yates, B.; Johnson, W. D.; Nze, R. *Am J Clin Nutr* 32(4): 954; 1979. (no refs)

79-2949 Cancer Morbidity and Causes of Death among Danish Brewery Workers. (Eng)

Jensen, O. M. (International Agency Res. Cancer, Unit Epidemiology and Biostatistics, 130 cours Albert Thomas, 69008 Lyon, France). *Int J Cancer* 23(4): 454-463; 1979.

Cancer morbidity and mortality were examined in a retrospective cohort study of 14,313 male members of the Danish Brewery Workers' Union (BWU) to determine the health effects of heavy beer drinking, in particular with respect to the etiology of colorectal cancer. Brewery workers are allowed to consume six bottles (2,100 ml) of light pilsener beer (alcohol content 3.7 g/100 ml) on the premises of the brewery per working day. The cohort included 1,063 mineral-water factory workers with no free ration of beer. Cancer morbidity and mortality were compared with those of the general popu-

lation after adjustment for age, sex, area, and time trends. Cancer morbidity during 1943-1972 was increased for cancer of the pharynx [relative risk (RR) = 2.09] esophagus (RR = 2.09), liver (RR = 1.51), and larynx (RR = 1.98). The risk of these tumors was highest among workers who had had a ration of free beer during ≥ 30 yr of employment. An increased risk of lung cancer (RR = 1.16) corresponded with the risk among persons of low socioeconomic class. These tumor sites accounted for a 9% excess of all malignant neoplasms among members of the BWU. No increased risk for cancer of the colon (RR = 1.07) or rectum (RR = 1.02) was observed. In contrast to studies of alcoholics, only deaths from the above-mentioned cancers, liver cirrhosis (RR = 1.77), and motor vehicle accidents (RR = 1.33) were in excess; total mortality was only slightly above expectation (RR = 1.06). It is suggested that the statistical association between beer and colorectal cancer is of a noncausal nature, that the heavy consumption of weak alcoholic beverages may increase the risk of upper respiratory-digestive tract cancers, and that part of the disease pattern associated with alcoholism may be unrelated to alcohol consumption. (44 refs)

79-2950 Induction of Aryl Hydrocarbon Hydroxylase Activity and Pulmonary Carcinoma. (Eng)

Gahmberg, C. G. (Dept. Bacteriology and Immunology, Univ. Helsinki, Helsinki, Finland); Sekki, A.; Kosunen, T. U.; Holsti, L. R.; Makela, O. *Int J Cancer* 23(3): 302-305; 1979.

Aryl hydrocarbon hydroxylase (AHH) activity in lymphoblasts from normal adult Finns, patients with pulmonary carcinomas, and patients with other types of malignancy was studied. High absolute AHH activity was observed in lymphoblasts from 15% of the normal subjects, 39% of the patients with pulmonary carcinoma, and 17% of the patients with other malignancies. Among the patients with pulmonary carcinoma, those with high AHH activity were an av of 5 yr younger than those with low AHH activity; this difference was statistically significant. Among the parents and children of the high-AHH individuals, there was a 50% frequency of high AHH activity. The data suggest that, after induction, high absolute AHH activity is controlled by an autosomal dominant gene (*Ahh*). Its frequency in the Finnish population is estimated to be 8%. These and other data indicate that after induction, increased absolute AHH activity is important in the development of lung cancer. (17 refs)

79-2951 Rising Lung Cancer Mortality among Nonsmokers. (Eng) Enstrom, J. E. (Sch. Public Health, Univ. California, Los Angeles, CA, 90024). *J Natl Cancer Inst* 62(4): 755-760; 1979.

Data on lung cancer mortality trends among nonsmokers and among the total US population were analyzed. Data from two representative surveys of lung cancer deaths in the US as well

as national mortality statistics and other epidemiologic studies show that the lung cancer mortality rate has risen substantially between 1914 and 1968 among persons who never smoked cigarettes. For white men and women, the relative increase for ages 35-84 yr has been about 30- and 7-fold, respectively. Most of the relative increase occurred before 1935 and was probably due to changes in diagnostic criteria. However, increases have continued up to the present for male nonsmokers, who now apparently have an annual age-adjusted lung cancer death rate of about 25/100,000 persons aged 35-84 yr. The rising lung cancer rate among nonsmokers indicates that factors in addition to personal cigarette smoking have had a significant effect on the mortality rate from this disease. In spite of the limited quality of these data, they suggest that a more complete understanding of lung cancer etiology is needed. (29 refs)

79-2952 Heart Disease, Cancer and Vehicle Travel. (Eng) Robinson, A. A. (16 Quayle St., Sandy Bay, Hobart, 7005, Tasmania). *Med Hypotheses* 5(3): 323-328; 1979.

An hypothesis explaining the observed increase in heart disease, lung and breast cancer, and diabetes associated with the increased use of motor vehicles from 1920 to 1975 is proposed. Because of the influence of visual stimulation on the CNS, an imbalance resulting in upsets in metabolic and neural control is caused. This imbalance can in turn cause a variety of ills that may appear completely unrelated to motor vehicle use. (3 refs)

79-2953 Cancer Mortality in Oil Refinery Workers. (Eng) Hanis, N. M. (Dept. Epidemiology and Preventive Medicine, Faculty Medicine, Univ. Western Ontario, London, Canada); Stavaky, K. M.; Fowler, J. L. *J Occup Med* 21(3): 167-174; 1979.

Cancer mortality rates among Imperial Oil Limited employees during 1964-1974 were examined in a cohort study. Compared with nonexposed employees, workers whose jobs exposed them daily to crude petroleum or its products had more than three times the risk of esophageal and stomach cancer and about twice the risk of lung cancer. In the exposed group, the risks of both cancers increased with increasing duration of employment. In the absence of more complete information on the similarity of the exposed and nonexposed employees, these results cannot be ascribed with certainty to a carcinogenic effect of petroleum. However, there was no other clear explanation for the results, and further study is required. When refinery workers were compared with non-refinery workers without consideration of exposure to petroleum in either group, the refinery workers were found to have twice the risk of cancer of the intestines (including rectum) and other digestive organs. No relationship with duration of employment was evident. Although the increased in-

testinal cancer mortality in the refinery workers was not consistent, bias was not an obvious explanation for the observed relationship. Therefore, the presence on a refinery site of a carcinogen other than petroleum cannot be ruled out. (17 refs)

- 79-2954 The Problem of Asbestosis in Spain.** (Eng) Segarra, F. (Instituto Territorial de Barcelona, Servicio Social de Higiene y Seguridad del Trabajo, Barcelona, Spain). *J Occup Med* 21(4): 279-280; 1979.

The occurrence of asbestosis in Spain is discussed. All of the asbestos used in Spain is imported, about 90% of it being chrysotile. About 75% of the industries associated with a risk of asbestosis are located in the Barcelona area; they include fibrocement, textile, insulation, and automobile parts industries. Of 1,003 workers examined (most from 2 fibrocement industries), 247 had asbestosis. There was a definite correlation between the incidence of the disease and the duration of exposure. Pleural lesions were seen in 220 patients, lung involvement in 108. About 50 asbestosis cases were reported in Spain from 1948 through 1974, and nearly 300 have been detected since 1974. The incidence of lung, colon, and rectal cancer and pleural mesothelioma among the exposed workers has also increased in comparison to that of the general population. It is expected that asbestosis and cancer among exposed workers will increase in the future, partly due to the deplorable hygienic conditions in industries associated with a risk of asbestosis. (9 refs)

- 79-2955 Smoking and Health: Statistical Data.** (Afr) Coetzee, A. M. (Afdeling Bedryfsgeondheid, Universiteit van Pretoria, Pretoria, S. Africa). *S Afr Med J* 54(11): 425-426; 1978.

Statistical data on cigarette smoking in South Africa in 1977 are presented. Of the population aged > 16 yr, 29.3% were smokers and 11.8% heavy smokers (> 20 cigarettes/day). The lung cancer mortality in the age bracket 20-65 yr was 1,082, which represented an excess of 691 cases. (1 ref)

- 79-2956 The Mortality of Men in the Rhondda Fach, 1950-1970.** (Eng) Cochrane, A. L. (MRC Epidemiology Unit, Cardiff, Wales); Haley, T. J.; Moore, F.; Hole, D. *Br J Ind Med* 36(1): 15-22; 1979.

A more detailed analysis of material from a 20-yr follow-up survey of men in the mining community of Rhondda Fach, Wales, confirms the similarity between the standardized mortality ratios (SMR's) of miners and exminers in radiological categories 0, 1, 2, 3 and A (120.3, 116.5, 119.0, 115.7, and 120.1 respectively) as well as the difference between these SMR's and that of the nonminers (98.7). The specific death

rates showed a raised SMR for bronchitis and other respiratory diseases, excluding pneumoconiosis (PMC), for all categories, including category 0, but little difference between those for category 0 and those for simple PMC. For carcinoma of the lung, the SMR's were equally low for nonminers and for miners and exminers. The SMR for stomach carcinoma was 113.0 for nonminers, 159.5 for men in category 0, 108.2 for men in categories 1-3, and 184.0 for men in categories A-C. The SMR's for stomach carcinoma are unusual, but they give no support to the suggestion that exposure to dust is an etiological factor. There is little or no suggestion of death rates being related to category of PMC except, of course, when PMC is being dealt with as a specific cause of death. The SMR for leukemia was low. A comparison between the survival rates of men aged 55-64 in Leigh, Lancashire, and those in the Rhondda Fach suggests that nonminers in the two areas have similar survival rates, but that survival rates for category 0 and simple PMC are lower in the Rhondda Fach. (26 refs)

- 79-2957 Variations in Nasopharyngeal Cancer Incidence among Specific Chinese Communities (Dialect Groups) in Singapore.** (Eng) Shanmugaratnam, K. (Dept. Pathology, Univ. Singapore, Outram Road, Singapore 3, Singapore). *IARC Sci Publ* 20: 191-198; 1978.

A total of 729 cases of nasopharyngeal carcinoma (NPC: 93.6% confirmed histologically) were diagnosed among the Singapore Chinese population during 1968-1972. Variations in NPC incidence among certain Chinese communities or dialect groups in this population were analyzed. Age-standardized incidence rates for the total Chinese population were 18.4/100,000/yr for men and 7.0 for women; the respective rates for the specific Chinese communities were 14.1 and 4.7 for Hokkien, 18.3 and 6.2 for Teochew, 29.1 and 11.0 for Cantonese, 14.2 and 3.3 for Hainanese, 12.6 and 4.8 for Hakka, and 12.2 and 6.0 for the other dialect groups. All of the Chinese communities in Singapore had high risks for NPC; only the Cantonese had a risk significantly higher than that for the rest of the Chinese population. NPC incidence rates for men and women of the other major racial groups in Singapore were 4.7 and 0.6 for Malays and 0.9 and 0.0 for Indians, respectively. Differences in the use and preparation of food items may be relevant to these patterns of cancer incidence. (21 refs)

- 79-2958 Epidemiological Data on Thyroid Cancer.** (Hun) Balazs, G. (I. sz. Sebeszeti Klinika, Debreceni Orvostudományi Egyetem, Debrecen, Hungary); Lukacs, G.; Csaky, G.; Arday, G. *Magy Onkol* 23(1): 63-67; 1979.

Epidemiological data on thyroid carcinoma are presented on the basis of 142 cases diagnosed over a 23-yr-period in Hungary. Of the 142 patients, 134 lived in a flatland endemic

goiter region (Debrecen). The histological diagnosis was available for 104 cases: differentiated carcinoma was found in 74%, undifferentiated carcinoma in 20%, and sarcoma in 6%. By comparison, differentiated carcinoma was diagnosed in 58% of 149 cases in a mountainous endemic goiter region (Innsbruck), undifferentiated carcinoma in 19%, and sarcoma in 23%. In a series of 224 cases in Vienna (nonendemic goiter region), the incidence of differentiated carcinoma, undifferentiated carcinoma, and sarcoma was 57%, 20% and 23%, respectively. Twenty-nine percent of the Debrecen patients were in the age group 0-20 yr, compared with 2% of the Innsbruck cases and 1.2% of the Vienna cases. The high incidence of struma nodosa in the Debrecen endemic goiter region suggests a relationship between this disease and thyroid carcinoma. (13 refs)

79-2959 Screening Program for Radiation-associated Thyroid Carcinoma (Meeting Abstract). (Eng) Shimaoka, K. (Roswell Park Memorial Inst., Buffalo, NY, 14263); Getaz, E. P.; Razack, M. S.; Rao, U.; Norman, M.; Wallace, H. J.; Shedd, D. P. *Proc Am Assoc Cancer Res* 20: 19; 1979. (no refs)

79-2960 Changing Incidence of Thyroid Cancer. (Eng) Weiss, W. (Div. Occupational Medicine, Dept. Medicine, Hahnemann Medical Coll. and Hosp., 6401 New Coll. Building, 230 N. Broad St., Philadelphia, PA, 19102). *J Natl Cancer Inst* 62(5): 1137-1142; 1979.

The incidence of thyroid cancer was examined temporally and geographically by age and sex from data provided by tumor registries in the US and abroad. The temporal trends in Connecticut showed an increase in annual incidence after 1945, with an especially sudden increase in incidence in women. The increase occurred predominantly in older men and younger women. The increase in young women was confirmed by cohort analysis. The rates rose with age in both sexes, but women have recently developed a secondary peak in the fourth decade of life. The same phenomenon was observed in other US data, but not as clearly in data from 10 foreign registries. These observations are consistent with the hypothesis that x-radiation therapy for benign conditions of the head and neck in childhood is a factor in the increased incidence of thyroid cancer in US women, but some other etiologic or modifying factor should be sought to explain the increased incidence in US men. (23 refs)

79-2961 Incidence of Thyroid and Other Tumors Associated with Radiation Therapy Exposure in Childhood (Meeting Abstract). (Eng) Colman, M. (Div. Radiation Oncology, Dept. Radiological Sciences, Univ. California, Irvine, CA, 92717); Kirsch, M.; Creditor, M. *Int J Radiat Oncol Biol Phys* 4(Suppl 2): 163; 1978. (no refs)

79-2962 Risk Factors for Brain Tumors in Children. (Eng) Gold, E. (Dept. Epidemiology, Johns Hopkins Sch. Hygiene and Public Health, 615 N. Wolfe St., Baltimore, MD, 21205); Gordis, L.; Tonascia, J.; Szklo, M. *Am J Epidemiol* 109(3): 309-319; 1979.

A case-control study was conducted in 15 hospitals in the Baltimore, Maryland area to determine possible etiologic factors associated with brain tumors in children. Eighty-four children with brain tumors diagnosed during 1965-1975 were compared with normal children and with children with other malignancies. Parents of these children were interviewed about a variety of possible etiologic factors, including exposure to chemical carcinogens. Children with brain tumors, as well as children with other cancers, had a greater tendency than normal children to have been first births and to have had higher birth weights. More children with brain tumors had a sibling with epilepsy or seizures than did normal children, and several of the mothers of children with brain tumors had themselves had epilepsy or a stroke at a relatively young age. There were no significant differences between the groups with regard to several maternal characteristics, including smoking during pregnancy and prior radiation exposure. However, the interview data indicated that more children with brain tumors and children with other cancers had been exposed to insecticides in the home than had normal children. Fewer children with brain tumors or with other cancers were reported to have had tonsillectomies than normal children. More of the children with brain tumors and those with other malignancies were reported to have been exposed to farm animals and to sick pets. This is one of the first case-control studies of the epidemiology of brain tumors in children, and the results suggest directions for future epidemiologic studies in this relatively uncharted field. (49 refs)

79-2963 Malignant Ethmoidonasal Tumors. Occupational Etiology of Adenocarcinomas in Woodworkers. (Fre) Gerard, A. (No affiliation given). *J Fr Otorhinolaryngol* 28(3): 200-201; 1979.

Sixty-five cases of malignant ethmoidonasal tumor were found in a hospital series. They included 32 adenocarcinomas, 9 epidermoid carcinomas, and 24 miscellaneous tumors. Twenty-seven of the 32 adenocarcinoma patients were woodworkers (cabinet-makers, joiners, carpenters, etc). The pathogenesis of ethmoidonasal adenocarcinomas in woodworkers is not yet clear. Tannins and hydrocarbons, especially benzo(a)pyrene, encountered during the woodworking process are suspected. In another series of 449 ethmoidonasal tumors, 33.18% occurred in woodworkers, and 92.61% of the woodworkers had adenocarcinoma. The findings indicate that ethmoidonasal adenocarcinoma should be recognized as an occupational disease. The length of employment in woodworking should be limited to 15 yr. (no refs)

79-2964 Some Environmental and Bodily Characteristics of Melanoma Patients. A Case-Control

Study. (Eng) Klepp, O. (The Norwegian Radium Hosp., Montebello, Oslo 3, Norway); Magnus, K. *Int J Cancer* 23(4): 482-486; 1979.

Possible risk factors for malignant melanoma were investigated in a case-control study of 78 Norwegian patients with malignant melanoma and 131 controls with malignant lymphoma, testicular cancer, or bone and soft tissue sarcoma. Cases did not differ significantly from controls with respect to smoking habits, participation in outdoor or indoor sports, sauna habits, hair or eye color, leisure or occupational time spent outdoors, sunbathing, use of UV-lamps, or degree of exposure of different body parts to sunlight. A higher percentage (19.2%) of melanoma patients had been to Southern Europe for sunbathing within the previous 5 yr than had controls (9.2%); the difference was of borderline statistical significance. Significantly more melanoma patients than controls reported very low tolerance to sun exposure as contrasted with very high tolerance, and significantly more melanoma patients than controls reported having freckles or freckling easily. Significantly more male melanoma patients reported using sun lotion during solar irradiation than male controls; no difference was seen among females. (10 refs)

79-2965 Offspring of Patients Treated for Cancer in Childhood. (Eng) Li, F. P. (Div. Epidemiology and Biostatistics, Sidney Farber Cancer Inst., 44 Binney St., Boston, MA, 02115); Fine, W.; Jaffe, N.; Holmes, G. E.; Holmes, F. F. *J Natl Cancer Inst* 62(5): 1193-1197; 1979.

The offspring of a series of survivors of childhood cancer were examined for inherited traits associated with development of the parental tumor and for mutagenic effects of preconception exposures to radiotherapy and chemotherapy. Patients with cancer diagnosed between ages 0 and 17 yr were identified from tumor registries of the Sidney Farber Cancer Institute (Boston, MA) and the Kansas University Medical Center (Kansas City, KS). For 146 patients (84 women and 62 men), 293 pregnancies were reported after cessation of treatment for diverse neoplasms. The outcomes of 286 completed pregnancies were as follows: 242 live births (1 set of twins), 1 stillbirth, 25 spontaneous abortions, and 19 therapeutic abortions. Seven live-born infants died during the first 2 yr of life, a frequency in accord with expectation. Two offspring have developed cancer. One girl and her father had bilateral hereditary retinoblastoma. A second girl developed acute myelocytic leukemia; her mother had received radiotherapy during childhood for a brain tumor. Compared with their cousins and with published figures for the general population, the study progeny had no excess of congenital anomalies or other diseases. Chromosome and immunoglobulin studies of a few offspring did not reveal damage from preconception exposure to cancer chemotherapy and radiotherapy. Findings indicated that large collaborative studies are needed to monitor the offspring of childhood cancer survivors for inherited traits associated with the parental tumors and for mutagenic effects of therapy, particularly intense multimodality treatments. (50 refs)

79-2966 Mouthwash and Oral Cancer: Carcinogen or Coincidence? (Eng) Weaver, A. (Dept. Surgery, Wayne State Univ., Detroit, MI); Fleming, S. M.; Smith, D. B. *J Oral Surg* 37(4): 250-253; 1979.

Two hundred patients with squamous cell cancer of the head and neck were compared with patients from a general surgery group on the use of tobacco, alcoholic beverages, and mouthwash. Patients with cancer of the head or neck used significantly greater quantities of tobacco and alcoholic beverages than the control group. Ten of the 11 nonsmoking and non-drinking patients had used mouthwash at least twice daily for > 20 yr. Only two patients diluted the mouthwash, and nine used a brand containing 25% alcohol. Another six patients used alcoholic beverages and/or tobacco only occasionally, but all used mouthwash excessively. Two case histories are presented, both of which implicate mouthwash as a possible etiological agent in persons susceptible to squamous cell cancer. It is suggested that a history of mouthwash use be determined for all patients with premalignant or malignant lesions of the oral cavity to ascertain whether the use of mouthwash is coincidental to the development of oral cancer or is carcinogenic. (8 refs)

79-2967 Risk of Cutaneous Carcinoma in Patients Treated with Oral Methoxsalen Photochemotherapy for Psoriasis. (Eng) Stern, R. S. (Dept. Dermatology, Beth Israel Hosp., Boston, MA, 02215); Thibodeau, L. A.; Kleinerman, R. A.; Parrish, J. A.; Fitzpatrick, T. B. *N Engl J Med* 300(15): 809-813; 1979.

A 2.1-yr prospective study of 1,373 patients given po 8-methoxypsoralen photochemotherapy for psoriasis revealed 30 patients with a total of 48 basal cell and squamous cell carcinomas. The incidence of cutaneous carcinoma was 2.63 (95% confidence limits = 1.91-3.90) times that expected for an age-, sex-, and geographically matched population. Relative risk to patients with a history of ionizing radiation was 3.68 (99% confidence limits, 2.42-8.69). Patients with a previous cutaneous carcinoma had a relative risk of 10.22 (99% confidence limits, 4.78-37.08). A higher than expected proportion of squamous cell carcinomas and an excess of squamous cell carcinomas in areas not exposed to sun were seen. New patients with known histories of ionizing radiation exposure or of skin tumors should be given 8-methoxypsoralen photochemotherapy only if they understand the risks and have disabling psoriasis untreatable by other means. (30 refs)

79-2968 Squamous Cell Skin Cancer in the North-west of England, 1967-69, and Its Relation to Occupation. (Eng) Whitaker, C. J. (Dept. Occupational Health, Univ. Manchester, Stopford Building, Oxford Road, Manchester M13 9PT, England); Lee, W. R.; Downes, J. E. *Br J Ind Med* 36(1): 43-51; 1979.

During 1967-1969, 781 cases of squamous cell carcinoma of

the skin were reported to the Manchester Regional Cancer Registry, Manchester, England. The male:female ratios were significantly different ($p < 0.001$) among the skin cancer sites. The age-specific incidence rates were significantly different ($p < 0.001$) between the sexes for the 5-yr age groups ≥ 55 yr. Full occupational histories were obtained for 598 patients; a further 148 patients gave one main occupation only, and the remaining 35 patients were untraced. The numbers of patients observed in broad occupational groups were compared with the numbers expected using the 1931 and 1951 censuses. For all skin cancer sites combined, male farmers and textile workers had highly significant excesses of 150% and 135%, respectively. The corresponding excesses for women were 30% for textile workers and they varied from 1,140% to 590% for farmers, but only for the farmers were the excesses highly significant. Male metal workers also showed excesses of 38% and 23% that were of borderline significance. The association between occupation and individual skin cancer sites was then considered. For men there were excesses in the arm for chemical workers, paper/printing workers, and fishermen and in the ears for builders, but these excesses were of borderline significance. There was a significant difference ($p < 0.05$) in the proportion of male patients with atopic skin conditions in each cancer site. However, this was not found for the female patients. For both male and female patients, no significant associations were found between the skin site and either eye color, residence in the tropics, or smoking habit. (23 refs)

- 79-2969 Risk Factors of Lip Cancer: A Critical Evaluation Based on Epidemiological Comparisons.** (Eng) Lindqvist, C. (Finnish Cancer Registry, Liisankatu 21 B, 00170 Helsinki 17, Finland). *Am J Public Health* 69(3): 256-260; 1979.

The incidences of cancer of the lip, head and neck skin, and oral cavity in Finland during 1953 through 1973 were investigated. The mean annual number of new cases of lip cancer was 151 for men and 14 for women; the incidences of skin and intraoral cancers were lower, especially for men. The incidences of lip and skin cancers and, to a lesser extent, intraoral cancer decreased during the 1960's. The incidences of all three cancers increased with age, but unlike the other two, lip cancer showed a marked decrease in incidence in the oldest age groups. The risk of contracting lip cancer was higher in rural than urban areas and higher in the northern and eastern parts of the country than in the south. The risk of skin and intraoral cancers was higher in urban than rural areas. The incidence of lip cancer was negatively correlated with those of skin, colon, and prostate cancers and positively correlated with those of lung, laryngeal, and stomach cancers (the latter 2 in men only). The geographic correlation between the incidence of lip cancer in men and median income was negative, whereas that between the incidence of lip cancer in men and the proportion of those employed in agriculture was positive. The data suggest that lip cancer is etiologically related to upper gastrointestinal and respiratory tract

cancers, but not to head and neck skin cancer. The strong positive association between lip and lung cancer suggests that tobacco smoking is a major common risk factor. (27 refs)

- 79-2970 Solar Keratosis, Pterygium, and Squamous Cell Carcinoma of the Conjunctiva in Malawi.** (Eng) Clear, A. S. (Geographical Pathology Unit., Dept. Morbid Anatomy, St. Thomas's Hosp. Medical Sch., London SE1, England); Chirambo, M. C.; Hutt, M. S. *Br J Ophthalmol* 63(2): 102-109; 1979.

When the histological features of 234 conjunctival biopsies from Africans in Malawi were reexamined, 167 lesions were reclassified as solar keratosis, and all but 1 contained an epithelial abnormality. These abnormalities were graded as hyperplasia or mild dysplasia (126 lesions), moderate dysplasia (19), or marked dysplasia or carcinoma in situ (21). Of the remaining 67 lesions, 54 were classified as invasive carcinomas but 13 could not be classified definitely; several of the latter were considered to be microinvasive carcinomas. Nearly all lesions were associated with solar damage to subepithelial connective tissues. The findings suggest that these lesions represent a continuous spectrum of epithelial abnormality from slight acanthosis and hyperkeratosis through mild and moderate dysplasia to severe dysplasia and carcinoma in situ. The present pathological findings, together with previous epidemiological findings, suggest that UV radiation is the major factor in the development of solar keratosis (pterygium) and, in consequence, the high incidence of squamous cell carcinoma of the conjunctiva in the tropics. (26 refs)

- 79-2971 Development of Second Malignancies in Patients Treated with Radiation (Meeting Abstract).** (Eng) Youssef, E. (Dept. Radiation Therapy, Methodist Hosp., Brooklyn, NY); Rafta, S.; Gillies, J.; Walton, R.; Selim, H. *Int J Radiat Oncol Biol Phys* 4(Suppl 2): 165; 1978. (no refs)

See also:

- *(Rev.): 79-2405, 79-2406, 79-2410, 79-2413, 79-2414, 79-2415, 79-2416, 79-2417, 79-2424, 79-2425, 79-2426, 79-2428, 79-2437, 79-2438, 79-2439, 79-2441, 79-2442, 79-2443, 79-2444, 79-2448, 79-2449, 79-2450, 79-2451, 79-2452, 79-2455, 79-2456, 79-2464, 79-2466, 79-2469.
*(Chem.): 79-2480, 79-2481, 79-2483, 79-2491, 79-2493, 79-2496, 79-2500, 79-2501, 79-2502, 79-2508, 79-2529, 79-2558, 79-2584, 79-2587, 79-2605, 79-2623, 79-2661, 79-2662.
*(Phys.): 79-2664, 79-2671, 79-2679, 79-2688.
*(Viral): 79-2808.
*(Path.): 79-2831, 79-2874, 79-2883, 79-2885.

MISCELLANEOUS

- 79-2972 Glycoprotein-containing Factor that Mediates Contact Inhibition of Growth.** (Eng) Lipkin, G. (Dept. Dermatology, New York Univ. Sch. Medicine, New York, NY, 10016); Knecht, M. E.; Rosenberg, M. *Ann NY Acad Sci* 312: 382-391; 1978.

A high-mol-wt glycoprotein-containing factor isolated from conditioned medium from a contact-inhibited (C-I) line of hamster melanocytes was studied for its effects on growth inhibition in cultures of malignant cells. The factor was added at 50 $\mu\text{g/ml}$ to subconfluent cultures of non-C-I RPMI 1846 hamster melanoma cells. At confluence, the treated cultures showed contact inhibition and a characteristic morphologic change from disoriented overgrowth of pleomorphic forms to well-oriented monolayers of fibroblastlike cells. Addition of partially purified melanocyte contact-inhibitory factor (MCIF) to subconfluent cultures of both murine (B16 and S91) and human melanomas produced identical morphologic and growth-inhibitory effects. The growth-inhibitory effects extended to a broad spectrum of transformed and benign cells of ectodermal, mesodermal, and endodermal origins. Maintenance of the C-I morphologic features of MCIF-treated melanoma cultures required intact cytoskeletal structures. In RPMI 1846 cells treated with MCIF, cyclic guanosine monophosphate levels decreased while cyclic AMP increased. An identical protein was detected by polyacrylamide gel electrophoresis in media from every C-I cell type examined but not in any of the non-C-I (malignant) cell types studied. The presence of this glycoprotein, therefore, appears to be correlated with the capacity for density-dependent growth regulation. (20 refs)

- 79-2973 Neoplastic Neuroepithelial Differentiation in an Experimental Transplantable Teratoma.** (Eng) Herman, M. M. (Dept. Pathology (Neuropathology), Stanford Univ. Sch. Medicine, Stanford, CA, 94305); Vandenberg, S. R. *30th Ann Symp Cancer Res* 93-109; 1978.

Neuroepithelial differentiation in the mouse teratoma OTT-6050 was characterized by morphological, biochemical, immunological, and in vitro methods. Ependymal, astrocytic, and neuronal differentiation was demonstrated in continuity with primitive neuroepithelial cells, and intracisternal A particles were found in stem cells and in the more differentiated cell types of OTT-6050. Compared with adult mouse brains, the teratomas showed increased cyclic AMP levels and increased activities of enzymes of the serotonergic, adrenergic, and cholinergic systems. Divergent neuroepithelial differentiation analogous to embryonic neurocytogenesis occurred in vitro within mitotically active cell populations as an early

event and proceeded without apparent tissue relationships to other germ layer derivatives. The divergent differentiation was characterized by astrocytes and neuroblasts. Limited neuroepithelial differentiation also occurred in organ cultures after 39 days in vitro. Brain-associated surface antigenic sites were demonstrated on the differentiating neuroepithelial tissues of OTT-6050. The mouse teratoma alkaline phosphatase was distinct from the kidney and placental isoenzymes, and one-dimensional electrophoresis demonstrated numerous qualitative similarities and quantitative differences between the fetal, neonatal, and adult brain nonhistone chromosomal proteins and those of the teratoma. (64 refs)

- 79-2974 Regulation of Differentiated Functions and Malignancy in Neuroblastoma Cells in Culture.** (Eng) Prasad, K. N. (Dept. Radiology, Univ. Colorado Medical Center, Denver, CO, 80262); Sinha, P. K. *30th Ann Symp Cancer Res* 111-141; 1978.

To determine the possible link between the expression of differentiated functions in cultured neuroblastoma cells and malignancy, the regulation of expression of each individual differentiated function was examined separately. Several conclusions were reached. Cyclic AMP (cAMP) appeared to be one of the important factors in the induction and regulation of several differentiated functions in mammalian nerve cells. A low level of cAMP in dividing nerve cells, which resulted from an increase in cAMP phosphodiesterase activity due to a mutation on the regulatory gene of this enzyme, was responsible for the expression of malignancy and abnormal differentiation. Although no one individual differentiated function seemed to be linked with malignancy, when several of the differentiated functions were expressed at max levels, tumorigenicity was abolished. Thus, the expression of malignancy and abnormal differentiation are apparently linked in neuroblastoma cells in culture. An increased level of cAMP-binding proteins provided one of the important intracellular mechanisms for protecting the formed cAMP from enzymatic hydrolysis during differentiation of neuroblastoma cells. Reduction in histone synthesis and in H_1 -histone phosphorylation may be an important biological signal for the dividing neuroblasts to 'turn-off' cell division. The absence of these events might be indicative of malignant changes. (57 refs)

- 79-2975 Studies of Human Prostatic Acid Phosphatase. II. Characterization with the Use of Anti-Human Prostatic Acid Phosphatase Serum and Application to**

Clinical Diagnosis of Prostatic Carcinoma. (Jpn) Sawada, H. (Gifu Coll. Pharmacy, 5-6-1 Mitahora-higashi, Gifu 502, Japan); Sasaki, E.; Asano, S. *Yakugaku Zasshi* 99(1): 83-89; 1979.

Rabbit antiserum monospecific to purified human prostatic acid phosphatase (PAPase) was used to characterize the enzyme and to determine the feasibility of its use in the clinical diagnosis of prostatic carcinoma. The Ouchterlony double-diffusion technique showed that the antiserum reacted with PAPase but not with RBC APase, serum platelets, or homogenates of brain, lung, liver, or kidney. When PAPase was treated with neuraminidase, it did not lose any of its activity and it reacted with the antiserum. PAPase lost 20%-50% of its activity when it was treated with 1 M urea, 0.01% sodium dodecyl sulfate (SDS), 0.25 M 2-mercaptoethanol (2-ME), or 0.2 M guanidine HCl (GH), and it lost all of its activity and did not react with the antiserum when it was treated with 4 M urea, 0.05% SDS, 1 M 2-ME, or 2 M GH. The antiserum protected the enzyme against heat and pH inactivation. When PAPase was coupled with the antiserum, the activity of the enzyme was stabilized at pH 4.6-5.8 at 56 C. PAPase levels were measured in the sera of 9 normal subjects (2 men, 7 women), 3 prostatic carcinoma patients, and 7 prostatic hypertrophy patients. The results were compared with the amount of PAPase activity inhibited by L-tartrate. Values of total PAPase activity and L-tartrate-inhibited activity were high in the sera of the prostatic carcinoma patients compared with values for the prostatic hypertrophy patients and normal subjects. Among the latter, values were similar for men and women. (35 refs)

79-2976 The Role of Phosphodiesterase in Cell Growth Control and Malignancy (Meeting Abstract). (Eng) Takemoto, D. F. (Univ. Southern California, Los Angeles, CA, 90007). *Diss Abstr Int [B]* 39(9): 4158-4159; 1979. (no refs)

79-2977 Further Studies on Tumorigenicity, Exclusion of HeLa Cell Contamination and Definition of Uniqueness by Polymorphic Enzyme Analysis of Cultured Human Tumor Cell Lines (Meeting Abstract). (Eng) Fogh, J. (Human Tumor Cell Lab., Sloan-Kettering Inst. Cancer Res., Rye, NY, 10580); Wright, W.; Daniels, W. P.; Tiso, J.; Orfeo, T. *In Vitro* 15(3): 197-198; 1979. (2 refs)

79-2978 Epidermal Transformation: The Production of Differentiated Cell Lines Under Improved Conditions of Culture (Meeting Abstract). (Eng) Miller, D. R. (Oak Ridge Natl. Lab., Oak Ridge, TN, 37830); Slaga, T. J.; Fischer, S. M. *In Vitro* 15(3): 195; 1979. (no refs)

79-2979 Neoplastic Potential of Cultured Insect Cells (Meeting Abstract). (Eng) Tsang, K. R. (Dept. Entomology, Fisheries and Wildlife, Univ. Minnesota, St. Paul, MN, 55108); Brooks, M. A. *In Vitro* 15(3): 196; 1979. (no refs)

79-2980 Heme Biosynthesis in Friend Erythroleukemia Cells: Control by Ferrochelatase. (Eng) Rutherford, T. (Nuffield Dept. Clinical Medicine, Radcliffe Infirmary, Oxford OX2 6HE, England); Thompson, G. G.; Moore, M. R. *Proc Natl Acad Sci USA* 76(2): 833-836; 1979.

The activities of the enzymes of heme biosynthesis (except protoporphyrin oxidase) were followed during the induction of Friend erythroleukemia cells in culture. All the enzyme activities increased after induction with dimethyl sulfoxide. The activities of the intermediate enzymes were much higher than those of δ -aminolevulinate synthase [ALS: succinyl-CoA:glycine C-succinyltransferase (decarboxylating)], the initial enzyme, or ferrochelatase (FC: protoheme ferriolase), the final enzyme of the pathway. FC activity was not detectable in the uninduced cells. ALS activity increased during the first 24 hr of induction; porphobilinogen deaminase activity began to increase after 48 hr and FC activity, after 72 hr. However, the induction of heme synthesis followed the same time course as that of FC activity, not that of ALS activity. The cellular growth medium was found to contain traces of protoporphyrin but not of other porphyrins. Thus, FC is shown to be rate-limiting for heme synthesis during early stages of Friend cell induction. A Friend cell variant (Fw), which is not inducible except in the presence of exogenous hemin, was also studied. All the enzymes of heme synthesis except FC were inducible by butyric acid. FC was not inducible by butyric acid or hemin plus butyric acid. These cells also excreted protoporphyrin. The failure to induce FC activity is believed to be the cause of, not a consequence of, the noninducibility of this cell line. (19 refs)

79-2981 The Cell Surface and the Control of Friend-Cell Differentiation. (Eng) Bernstein, A. (Ontario Cancer Inst., Univ. Toronto, Toronto, Ontario M4X 1K9, Canada); Mager, D. L.; MacDonald, M.; Letarte, M.; Loritz, F.; McCutcheon, M.; Miller, R. G.; Mak, T. W. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book A), 528 pp.; 249-260; 1978.

Recent experiments suggesting that membrane changes, both early and late, are an integral part of Friend cell differentiation are summarized. Ouabain, a specific inhibitor of Na^+/K^+ ATPase, induces Friend cell differentiation via its binding to this enzyme. Early changes in the transport of a number of compounds that require a functioning Na^+/K^+ ATPase occur during induction by a variety of different Friend cell inducers. The occurrence and magnitude of these early transport changes have a strong predictive value in

terms of the number of differentiated cells observed 5 days later. Growth of Friend cells in medium in which the relative concentrations of Na^+ and K^+ ions have been reversed induces differentiation in a significant proportion of cells. These results suggest that early inhibition of Na^+/K^+ ATPase is a common step in Friend cell differentiation. It decreases the transport of a number of Na^+ -dependent compounds and may affect other cellular processes that are dependent on Na^+/K^+ ATPase. Another important aspect of this model is that this early inhibition in the Na^+/K^+ ATPase activity is an integral part of the cellular changes that lead to later, inducer-independent events in Friend cell differentiation. (28 refs)

- 79-2982 Chromatin Changes and DNA Synthesis in Friend Erythroleukemia and HeLa Cells During Treatment with DMSO and n-Butyrate.** (Eng) Neumann, J. R. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA, 02139); Riggs, M. G.; Hagopian, H. K.; Whitaker, R. G.; Ingram, V. M. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book A), 528 pp.; 261-275; 1978.

Chromatin changes and DNA synthesis were studied in Friend erythroleukemia and HeLa cells during treatment of the cells with dimethyl sulfoxide (DMSO) and n-butyrate. The overall pattern of nuclear phosphoproteins was similar in uninduced and in DMSO-stimulated Friend cells. However, there was an overall decrease in protein synthesis and phosphorylation after 50 hr of DMSO treatment. H1 and H2A were the major phosphorylated histone species both in uninduced and induced erythroleukemia cells. H1 and H2A appeared to be the only histones affected by DMSO induction. The pattern of proteins associated with mono- and oligonucleosomes after 2 min of digestion of nuclei with micrococcal nuclease resembled that produced by the much longer digestion of uninduced cells, indicating that the DNA of DMSO-induced Friend cells is much more accessible to nuclease digestion. N-Butyrate induced a striking accumulation of acetylated histones in HeLa and Friend erythroleukemia cells. Histone acetylation was reversible when cells were shifted to control medium. The rate of incorporation of thymidine, presumably into DNA, was strongly inhibited by n-butyrate in HeLa cells. Both effects of n-butyrate were expressed quite early and had a similar time course, suggesting a close link between the two processes (acetylation of histones and inhibition of DNA synthesis). It is concluded that cytosol from n-butyrate-treated cells contains an inhibitor or dilutes factors needed by the nuclei for DNA synthesis. (59 refs)

- 79-2983 Induction of Murine Erythroleukemia Cells to Differentiate: Cell-cycle-related Events in Expression of Erythroid Differentiation.** (Eng) Marks, P. A. (Cancer Center, Columbia Univ. Coll Physicians and Surgeons, New York, NY, 10032); Terada, M.; Fibach, E.; Ga-

zitt, Y.; Nudel, U.; Reuben, R.; Bank, A.; Rifkind, R. A. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book A), 528 pp.; 221-233; 1978.

The relationship between cell-cycle events and transition to Hb production was studied in Friend virus murine erythroleukemia cells (MELC). MELC cultured with the inducers dimethylsulfoxide (DMSO), butyric acid, or dimethylacetamide developed prolonged G_1 or a transient block in initiation of DNA synthesis. There was a decrease in the proportion of cells in S phase during the early period of culture with inducing agents. MELC cultured with inducers remained in G_1 for 6.5-8 hr, compared with 4 hr for cells cultured without inducer. There was evidence that structural changes in chromatin occurred in the course of induced MELC differentiation. A decrease in the rate of sedimentation of DNA on alkaline sucrose gradients was seen with DNA preparations from MELC cultured with either of the three inducers. An increased proportion of MELC could be induced to differentiate by UV radiation when suboptimal concentrations of DMSO were present in the media after irradiation. MELC cultured in 28 mM DMSO for 5 days showed up to 25% benzidine-reactive cells. Actinomycin D (1-2 nanograms/ml) induced >90% of MELC to differentiate after 5 days in the culture. Present evidence suggests that different inducing agents may have different primary sites of action. (21 refs)

- 79-2984 Phospholipid Metabolism of 3T3 Mouse Fibroblasts after Serum Stimulation and Through the G_1 and S Cell Cycle Phases: Incorporation and Disappearance of ^{32}P .** (Eng) Dubois, C. (Laboratoire de Biochimie I, Faculte de Medecine Saint-Antoine, 27, Rue Chaligny, 75571 Paris Cedex 12, France); Rampini, C. *Biochimie* 60(11/12): 1307-1313; 1978.

The phospholipid metabolism of 3T3 mouse fibroblasts was studied after serum stimulation of arrested cells. Cells were arrested by incubating them in serum-free medium. The phospholipids were analyzed by studying the incorporation and disappearance of ^{32}P into cellular phospholipids at different time intervals after the addition of serum. For phosphatidylethanolamine (PE) and phosphatidylcholine (PC), there was an early peak of incorporation in the G_1 phase of the cell cycle, 6 hr after serum addition. For phosphatidylinositol (PI), there was an intense initial increase of incorporation that continued through G_1 and S. Study of the disappearance of ^{32}P from the different phospholipids indicated that at the beginning of serum stimulation, an intense breakdown of PI occurred, that continued through G_1 and S. Except at the onset, the breakdown of PI was compensated for exactly by its synthesis. These two phenomena were closely linked. The breakdown-synthesis cycle, triggered by serum addition to arrested cells, may be linked to other membrane phenomena triggered by serum stimulation. Study of ^{32}P disappearance also indicated that synthesis of PE, probably from PC, occurred at the G_1 phase, 4 hr after serum addition and the beginning of the chase experiment. (24 refs)

- 79-2985 Heterotransplantation of Human Transitional Cell Carcinoma in Athymic Mice.** (Eng) Sufrin, G. (Roswell Park Mem. Inst., New York State Dept. Health, Buffalo, NY); McGarry, M. P.; Sandberg, A. A.; Murphy, G. P. *J Urol* 121(2): 159-161; 1979.

The growth of human transitional cell carcinoma in athymic nude mice and their normal heterozygote littermates was studied. Tumors from 20 patients were transplanted sc into 104 athymic mice. Successful takes were observed with 40% of the tumors and in 39% of the mice. No tumors grew in the normal heterozygotes. The stage of tumor, its histologic grade, its multicentricity and tendency to recur, and the subsequent clinical prognosis were not related to the success or failure of graft takes in the nude hosts. The transplanted tumors formed visible masses 24 hr after implantation, but showed critical periods of growth around 12 days and 20-25 days, when there was either an increase in size or regression. Fully developed xenografts appeared as well-encapsulated solid masses with cytologic and histologic appearances in full accordance with those of the primary tumor. There were no metastases. Chromosome analysis of the xenografts from two patients showed a human karyotype with no mouse chromosomes and a modal chromosome number of 48. (19 refs)

- 79-2986 Establishment of a New Human Chondrosarcomatous Cell Line.** (Eng) Uchida, T. (Dept. Pathology, Nihon Univ. Sch. Medicine, 30-1 Ooyaguchi-kamimachi, Itabashi-ku, Tokyo 173, Japan); Matsumoto, K.; Shimoda, T.; Shikata, T. *Acta Pathol Jpn* 29(1): 51-60; 1979.

A human osteosarcoma cell line (HuOS) was established from the tumor cells of a pulmonary metastasis of chondroblastic osteosarcoma in a 15-yr-old boy. The tumor cell line has been maintained in culture for > 19 mo. Injection of HuOS cells (1×10^7 cells im) into the thigh of seven nude mice produced a tumor at the injection site in all the mice after 1 mo. These tumors were chondrosarcomas, and no evidence of osteoid or bone formation was detected. The electron microscopic appearance of the HuOS cells was that of typical cartilaginous tumor cells with a prominent and dilated granular endoplasmic reticulum, a distinct Golgi apparatus, numerous dark and elongated mitochondria, numerous scattered ribosomes, a few lipid droplets, myelinlike bodies, and occasional small bundles of cytoplasmic filaments. The production of a cartilaginous matrix by the HuSO cells was not detected either histologically or electron microscopically. (18 refs)

- 79-2987 Bronchoscopy in the European Hamster (*Cricetus cricetus*).** (Eng) Reznik, G. (Tumor Pathology Branch, Carcinogenesis Testing Program, NCI, NIH, Bethesda, MD, 20014); Resnik-Schuller, H.; Dickhaus, S. *Lab Anim Sci* 29(1): 85-87; 1979.

The chronological development of induced lung cancer was studied in vivo in the European hamster by a modified pediatric bronchoscope that yielded excellent biopsy specimens for microscopy and cytology studies. (6 refs)

- 79-2988 α -Fetoprotein and Albumin Genes of Rats: No Evidence for Amplification-Deletion or Rearrangement in Rat Liver Carcinogenesis.** (Eng) Sala-Trepat, J. M. (Laboratoire d'Enzymologie, Centre National de la Recherche Scientifique, 91190 Gif-sur-Yvette, France); Sargent, T. D.; Sell, S.; Bonner, J. *Proc Natl Acad Sci USA* 76(2): 695-699; 1979.

Full-length radiolabeled albumin and α -fetoprotein (AFP) complementary DNA's (cDNA's) were synthesized from pure albumin and AFP messenger RNA (mRNA) preparations with the use of avian myeloblastosis virus reverse transcriptase (RNA-dependent DNA polymerase). The cDNA's were used to quantitate the number of albumin and AFP genes in different Sprague-Dawley or Buffalo rat tissues by two independent methods, both of which yielded similar results. First, the kinetics of the association of these cDNA's with nuclear DNA from rat liver, rat kidney, and Morris hepatoma 7777 under conditions of vast DNA excess indicated that the albumin and AFP mRNA's are transcribed from "nonrepetitive DNA." Second, saturation hybridization experiments in which a constant amount of rat liver DNA or Morris hepatoma 7777 was hybridized with increasing amounts of cDNA to albumin mRNA showed that there are one to two albumin genes per rat haploid genome. The number of AFP genes obtained in similar titration experiments was approx two to three. This was true whether rat liver DNA or hepatoma 7777 DNA was used in the reassociation experiments. When high-mol-wt DNA preparations from both these tissues were digested with the restriction endonuclease *EcoRI* and the fragments were transferred to a nitrocellulose filter, the albumin and AFP [32 P]cDNA probes hybridized to different sets of DNA fragments. However, each probe gave the same hybridization pattern whether Buffalo rat liver DNA or hepatoma 7777 DNA was utilized. (22 refs)

- 79-2989 Current Problems in the Choice of Animals for Toxicity Testing.** (Eng) Stevenson, D. E. (Shell Development Co., Westhollow Res. Center, P.O. Box 1380, Houston, TX, 77001). *J Toxicol Environ Health* 5(1): 9-15; 1979.

Animal models for toxicity studies are chosen more for reasons such as life-span, ease of handling, and economics than because of a comprehensive process of validation. There is a tendency to seek the answers to nonspecific questions and then to use those answers in situations in which they cannot lead to proper judgments. (5 refs)

79-2990 The Characterization of Two New Mouse Mammary Tumor Cell Lines (Meeting Abstract). (Eng) Zeller, N. K. (Univ. Maryland Baltimore Professional Sch., Baltimore, MD, 21201). *Diss Abstr Int [B]* 39(9): 4202-4203; 1979. (no refs)

79-2991 Lipid Vesicles as Vehicles for the Introduction of Virus and Nucleic Acids into Cells (Meeting Abstract). (Eng) Wilson, A. T. (State Univ. New York, Buffalo, NY). *Diss Abstr Int [B]* 39(9): 4201; 1979. (no refs)

79-2992 Regulation of Hematopoietic Differentiation and Proliferation by Colony-stimulating Factors. (Eng) Burgess, A. W. (Cancer Res. Inst., Walter and Eliza Hall Inst. Medical Res., Melbourne, Victoria 3050, Australia); Metcalf, D.; Russell, S. *Cold Spring Harbor Conf Cell Proliferation* Vol. 5(Book A), 528 pp.; 339-357; 1978.

Experiments to characterize the different molecular species of granulocyte-macrophage colony-stimulating factor (GM-CSF) present in mouse lung conditioned medium (MLCM) and the microheterogeneity associated with a particular species of GM-CSF are described. MLCM appeared to contain at least two mol wt species of GM-CSF, but most of the activity [ie, that binding to concanavalin A (Con A)-Sephadex] eluted from Sephadex G-100 as a single peak with a mol wt of 29,000. GM-CSF passing through Con A-Sephadex eluted as two peaks from Sephadex G-100, one at the same elution volume as the GM-CSF that specifically eluted from Con A-Sephadex and the other at higher mol wt. Microheterogeneity was present even in highly purified GM-CSF and could be detected by electrophoresis. The amino acid composition obtained after a 24-hr hydrolysis in the presence of p-toluene sulfonic acid indicated that there were twice as many acidic residues as basic ones. The hexosamine content in this initial analysis was only five to six residues per molecule. The level of incorporation of ³H-labeled amino acids was too low and too nonuniform to produce radiolabeled GM-CSF that would be suitable for sequencing studies. However, it appears to be possible to introduce ¹²⁵I into GM-CSF, but with rather low efficiency and with a considerable loss of biological activity. Thin-layer granulated gel bed isoelectric focusing of GM-CSF and ¹²⁵I-labeled GM-CSF gave an isoelectric point close to pH 4.9. CSF's with other cellular specificities were also analyzed with isoelectric focusing, and each appeared to have distinct isoelectric properties. Thus, the charge distribution on different CSF's appears to be sufficiently different to allow the separation of cell-specific CSF's by isoelectric focusing. (42 refs)

79-2993 Contact Reactions Influencing Cell Locomotion of a Mouse Sarcoma in Culture. (Eng) Abercrombie, M. (Strangeways Res. Lab., Worts' Causeway,

Cambridge CB1 4RN, England); Turner, A. A. *Med Biol* 56(6): 299-303; 1978.

Time-lapse cinematography was used to study contact reactions influencing cell locomotion of the mouse transplantable sarcoma S180 in culture. It is apparent from these studies that contact inhibition exists between S180 cells, although this phenomenon has little influence on their mode of spread, which is largely brought about by diffusive movement. The relatively high (though variable) speed and high rate of turning of S180 cells (which, from the viewpoint of diffusive movement, tend to cancel each other) combine with their compact form and poor mutual adhesion to make the style of movement of S180 cells strangely like that of macrophages. S180 cells show a severe deficiency of contact inhibition by normal fibroblasts, which permits them to invade a population of fibroblasts. However, the deficiency is by no means a total absence of contact inhibition. Compared with the spreading of S180 cells from a focus of high density over an area of substratum free of fibroblasts, spreading during invasion is likely to be slowed by obstruction at the fibroblast front and by diminished speed when on the fibroblasts and quickened by contact guidance from oriented fibroblasts. During invasion, the S180 cells may be largely using the collagen substratum between the fibroblasts for their locomotion. (7 refs)

79-2994 Successively Transplanted Canine Transmissible Sarcoma. (Eng) Koike, T. (Dept. Veterinary Surgery, Faculty of Veterinary Medicine, Hokkaido Univ., Kita-18-jo, Nishi-9-chome, Kita-ku, Sapporo 060, Japan); Kudo, T.; Otomo, K.; Sakai, T. *Gann* 70(1): 115-118; 1979.

Observations from the 21st to the 50th transplant generation of an allogeneic tumor cell line derived from a sarcoma of the external genitalia of a female dog are reported. A total of 170 dogs aged 1-8 mo were observed; the time span required to take the tumor line from generation 21 to 50 was 4 yr, 7 mo. An av of 16.3×10^7 viable tumor cells were inoculated at each transfer. A tumor developed within 4-5 days at the inoculation site in 100% of the dogs inoculated. The tumors regressed in 26 animals, in 17 dogs within 5 mo. Metastasis occurred in 22 dogs, in 15 cases involving the liver and sc tissue of the whole body. The histologic features and karyologic characteristics of the tumor cell line were not modified by the serial transfers. (17 refs)

79-2995 Is Breast Cancer a Result of Endocrine Targeting? (How Women Differ from Rats or Mice). (Eng) Warren, S. (Cancer Res. Inst., New England Deaconess Hosp., Boston, MA, 02215); Brown, C. E.; Chute, R. N. *Ann Clin Lab Sci* 9(2): 87-93; 1979.

An attempt was made to identify hormonal changes occurring in both the surgical castrate and its intact female partner

in parabiosed NEDH rats and to determine the incidence of breast carcinoma (BC) in the intact females. The rats were divided into five groups: (1) 4 pairs, females anastomosed to oophorectomized females; (2) 5 pairs, females anastomosed to castrated males; (3) 7 pairs and (4) 6 pairs, same as Groups 1 and 2, respectively, except that a kidney was removed from each partner; (5) 3 pairs, parabiosed intact females (controls). Of the total 22 female partners of castrated rats, 20 developed BC's. Most of the cancers were multiple, numbering up to six. All female partners in Groups 3 and 4 developed BC. None of the Group 5 rats developed BC. In all groups, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels exceeded those of their intact partners, whereas prolactin (PL) levels of the intact partners with BC were more than double those of the castrates. Progesterone (PG) and estradiol (ED) values also exceeded those of the castrate partners. However, all the castrates' sera showed some steroid contact, which is assumed to have resulted from adrenal activity and backflow from the higher levels in the intact partners. The sequence of events culminating in BC is thought to be as follows: castration of the male or female parabiont caused removal of estrogen or testosterone with loss of feedback control over the pituitary secretion of LH and FSH. Crossover of these two polypeptides continuously stimulated the intact partner's ovaries to oversecrete PG and ED in variable amounts. This in turn resulted in oversecretion of PL by the intact partner's pituitary. The interplay between the continuing replicative stimulation of breast epithelium by PL, ED, and PG ultimately terminated in BC. An attempt to weight various factors that may be involved in causing human and experimental (rat) BC suggests significant differences of a quantitative nature. (35 refs)

- 79-2996 Inverse Correlation Between Cell-Surface Adhesiveness and Malignancy in Mouse Fibroblastoid Cell Lines.** (Eng) Bumenik, J. (Inst. Molecular Genetics, Czechoslovak Acad. Sciences, 166 10 Prague 6, Czechoslovakia); Perlmann, P.; Fenyo, E. M.; Jandlova, T.; Suhajova, E.; Malkovsky, M. *Int J Cancer* 23(3): 392-396; 1979.

The correlation between tumorigenicity and cell-surface adhesiveness was investigated in eight mouse fibroblastoid cell lines. Three methylcholanthrene (MC)-induced fibroblastoid cell lines (MC 11, 13, and 14) showed similar tumorigenicities after sc injection into 3-mo-old syngeneic recipients. A fourth MC-derived line (MC 15) was significantly less tumorigenic than the other three. As determined by the latex particle adherence assay, the cell-surface adhesiveness of lines MC 11, 13, and 14 was 14.6%-16.7%, whereas that of MC 15 was significantly higher (46.1%). Among the MC 11, 13, and 14 lines, 24.6%-38.9% of the cells attached no latex particles, whereas only 4.6% of the MC 15 cells attached no particles. Approx 50% of all cell lines attached 1-25 particles. The same inverse correlation between tumorigenicity and cell-surface adhesiveness was observed in two lines derived from mouse L cell line (A9 and A9HT) and in two lines

derived from the Sendai virus-induced hybridization of A9HT cells with normal diploid mouse lymphocytes. (25 refs)

- 79-2997 Demonstration of Gamma-Glutamyl Transpeptidase in Normal and Malignant Melanocytes (Meeting Abstract).** (Eng) Hu, F. (Dept. Cutaneous Biology, Oregon Regional Primate Res. Center, Beaverton, OR); Buxman, M. M. *Clin Res* 27(2): 529A; 1979. (no refs)

- 79-2998 Quantitative Markers for Transformation of Cultured Rat Liver Epithelial Cells (Meeting Abstract).** (Eng) Shimada, T. (Naylor Dana Inst. Disease Prevention, Valhalla, NY, 10595); San, R. H.; Williams, G. M. *Proc Am Assoc Cancer Res* 20: 36; 1979. (no refs)

- 79-2999 Cell Culture for the Study of Epithelial Cells.** (Eng) Green, H. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA, 02139). *Natl Cancer Inst Monogr* (48): 259-262; 1978.

A cell culture system that allows the study of interacting systems is described. The system consists of epidermal keratinocytes and lethally irradiated fibroblasts and permits development of the essential elements of a stratified, squamous epithelium. When line XB teratoma keratinocytes are cultured with medium harvested from fibroblast cultures, they may proliferate or undergo terminal differentiation, depending on the culture conditions. Four independent keratinocyte lines were established in culture from tumors resulting from injection of teratoma PS A4 stem cells into nude mice. When inoculated with lethally irradiated 3T3 cells or with medium that had been in contact with a confluent monolayer of 3T3 cells, they formed colonies with high efficiency. When inoculated in the absence of 3T3 cells, the keratinocytes did not form colonies or they failed to grow through more than a few cell divisions. As with other keratinocytes, they were dependent on fibroblasts for proliferation but not for terminal differentiation. It is likely that other epithelial cell types might be cultivable under cell culture conditions with proper fibroblast support. (14 refs)

- 79-3000 Serum Lactate Dehydrogenase Isoenzymes in Early Malignancy.** (Eng) Ananthanarayanan, P. H. (Dept. Biochemistry, Jawaharlal Inst. Postgraduate Medical Education and Res., Pondicherry-605006, India); Ramakrishnan, S. *Indian J Med Res* 68: 459-465; 1978.

The isoenzyme patterns of lactic dehydrogenase (LDH) in sera from normal humans and from patients with nonmetastatic malignant disease, myocardial infarction (MI), and infectious hepatitis (IH) were studied by polyacrylamide gel electrophoresis. The LDH isoenzymes LD₁ and LD₂ were increased in MI and LD₄ and LD₅ were increased in IH. Total serum LDH activity was significantly increased above normal levels in 14 patients with esophageal cancer, 8 with carcinoma of the prostate, 6 with Hodgkin's disease, 3 with multiple myeloma, and 1 patient each with embryonal rhabdomyosarcoma, ovarian cancer, and neuroblastoma. In

all cases of esophageal and prostatic cancer and Hodgkin's disease, LD₁ and LD₂ were increased markedly, whereas only minimal increases in LD₃ and/or LD₄ were observed. LD₁ and LD₂ were decreased in multiple myeloma; LD₁, but not LD₂, was increased in embryonal rhabdomyosarcoma; and LD₁, LD₂, LD₃, and LD₄ were increased in neuroblastoma and ovarian cancer. Additional isoenzyme bands were observed in the sera from patients with embryonal tumors. The results indicate that serum LDH and the LDH isoenzyme pattern may be of value in diagnosing early malignant conditions. (14 refs)

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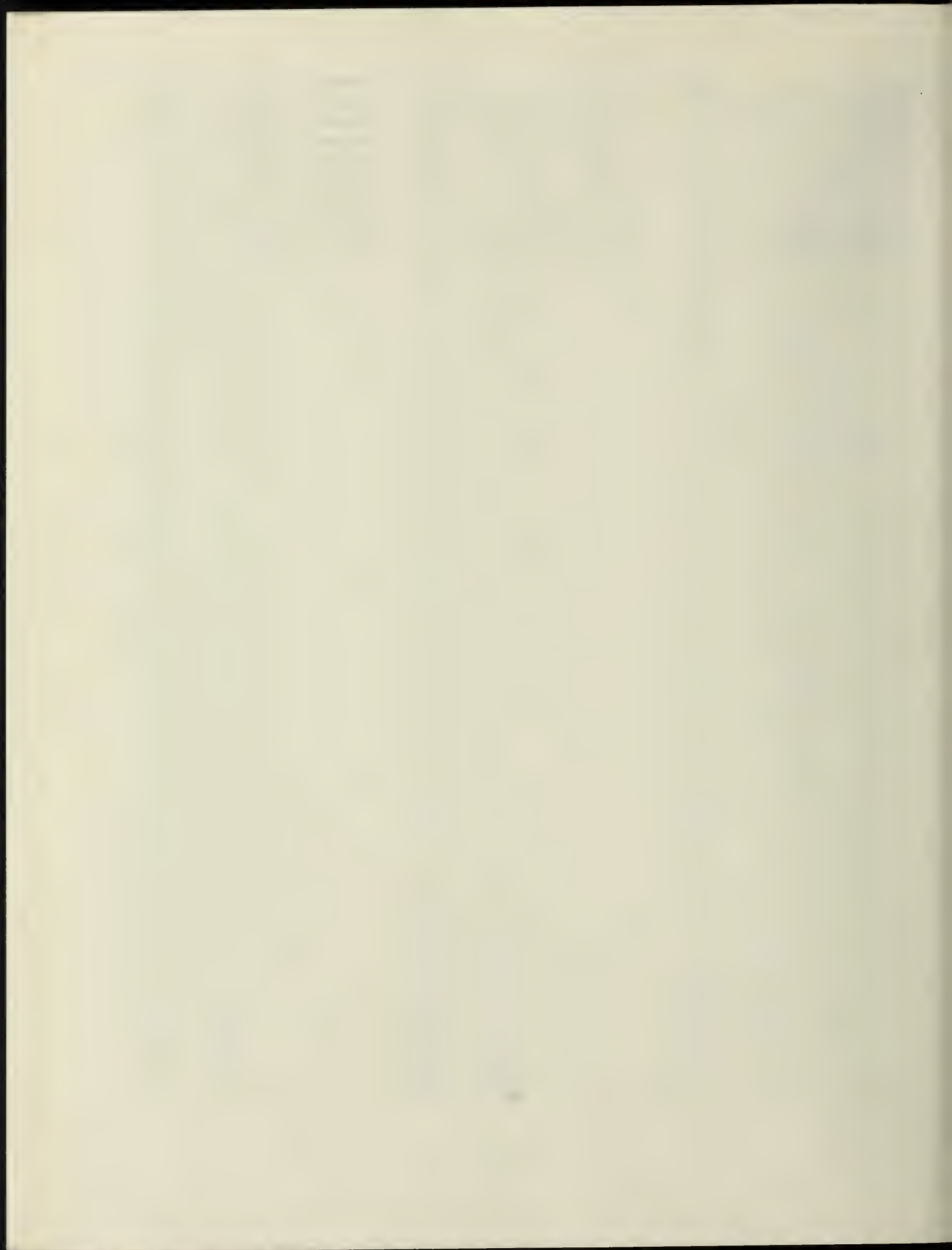
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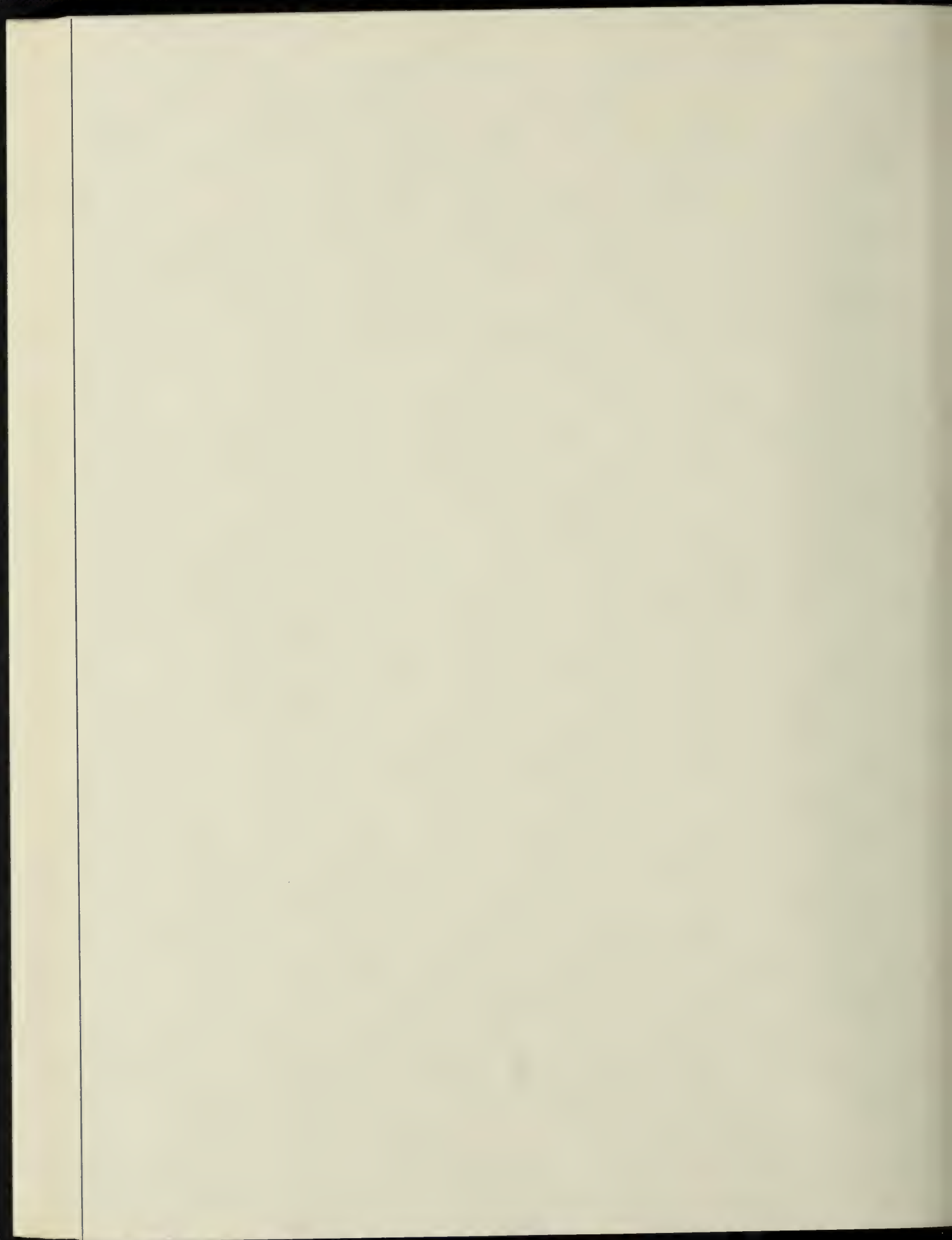
CARCINOGENESIS ABSTRACTS

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CARCINOGENESIS ABSTRACTS

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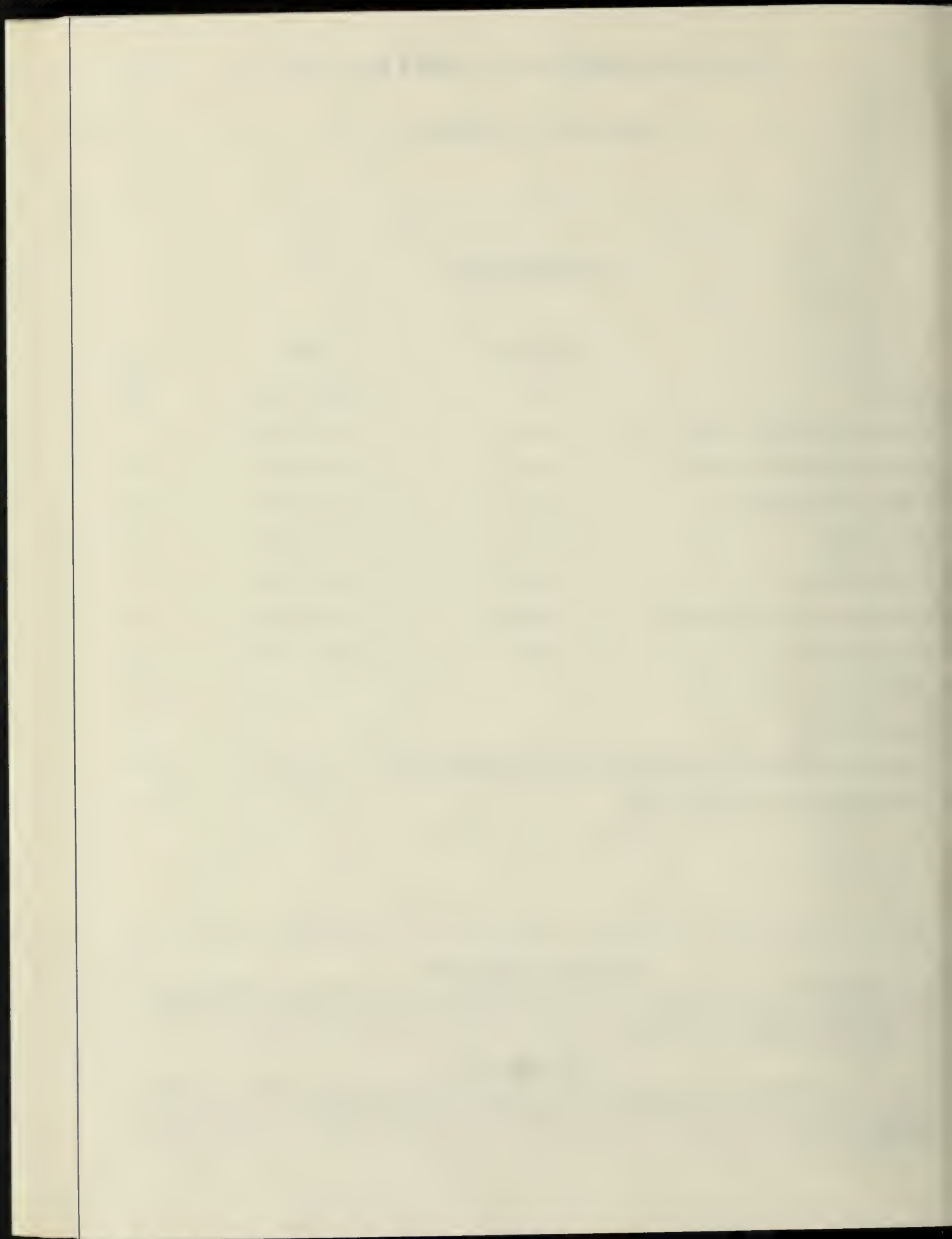
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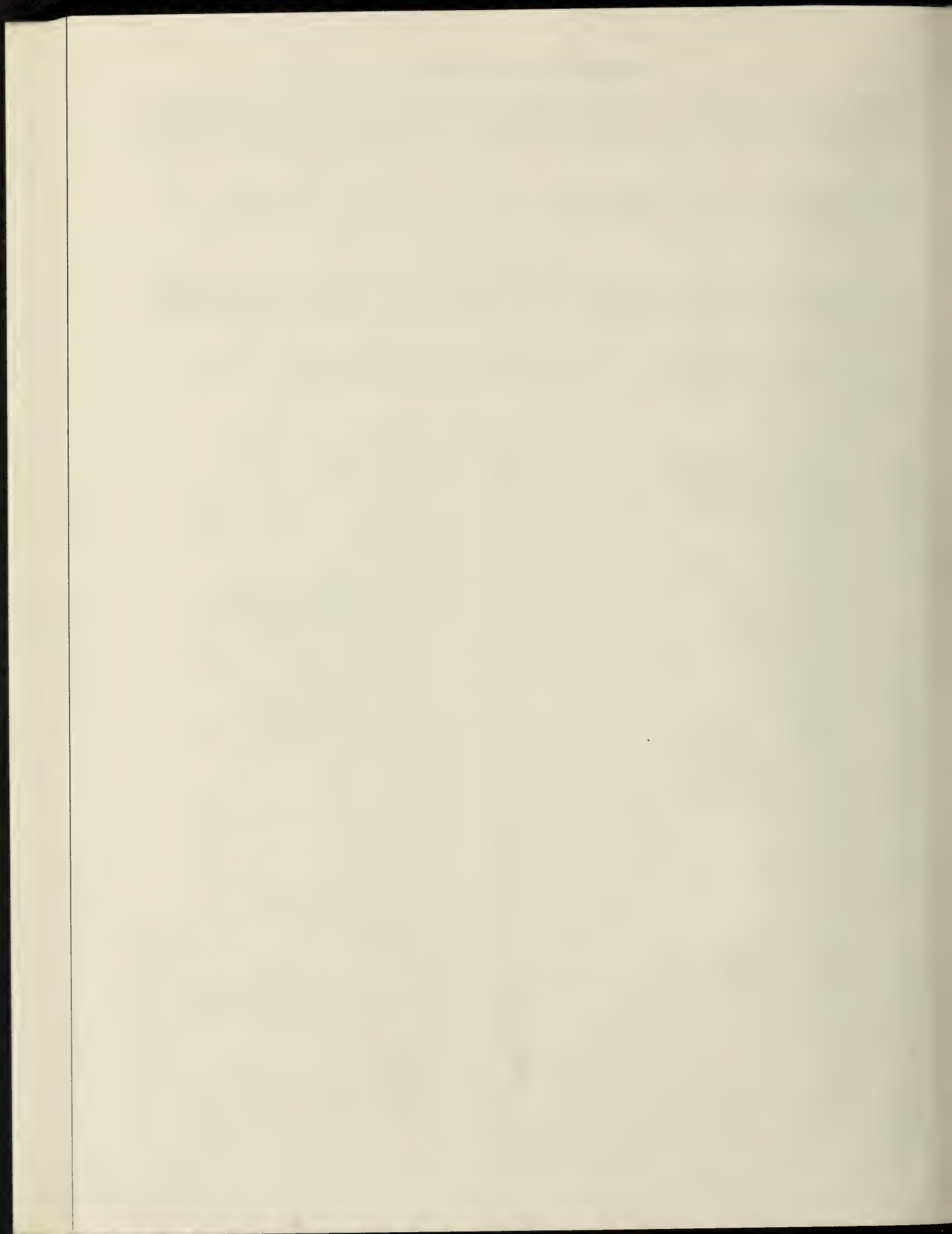
ABBREVIATIONS

JOURNAL names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations*.

LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED₅₀	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO ₂	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD ₅₀	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD₅₀	median lethal dose		
M	molar		
μM	micromolar		



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- 79-3001 Biological and Enzymatic Events in Chemical Carcinogenesis.** (Eng) Pitot, H. C. (McArdle Lab. Cancer Res., Dept. Oncology, Medical Sch., Univ. Wisconsin, Madison, WI 53706). *Annu Rev Med* 30: 25-39; 1979.

The biological and enzymatic events in chemical carcinogenesis are reviewed. Two stages, initiation and promotion, were identified in the carcinogenesis of squamous epithelium. Later, two distinct phases were also identified in rodent hepatocarcinogenesis: the appearance of hyperplastic nodules followed by the appearance of hepatocellular carcinomas after phenobarbital promotion. Hepatic adenomas and focal nodular hyperplasia in humans may be analogous to the hyperplastic nodules in the rat. Hyperplastic alveolar nodules and, possibly, mammary ductal hyperplasias appear to be intermediate stages in rodent mammary carcinogenesis, and preneoplastic lesions in the human breast may be analogous to these lesions. Two-stage carcinogenesis also appears to occur in colon, bladder, and pancreatic tumors. It appears that virtually all chemical carcinogens that are not themselves the ultimate forms undergo reactions to ultimate forms with or without a proximate intermediate form. The enzymatic pathways involved are primarily those associated with microsomal enzymes and the cytochrome P-450 system. Present knowledge of molecular biology strongly suggests that the critical site of action of the ultimate forms of chemical carcinogens is DNA. Promotion does not appear to involve any interaction with DNA, and a likely hypothesis is that promoting agents act as gene activators. (87 refs)

- 79-3002 Specific Genetic Loci as Targets of Carcinogens and Tumor Promoters in Mammals.** (Eng) O'Brien, S. J. (Immunology Section, Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014); Rice, M. C. *J Environ Pathol Toxicol* 2(4): 1055-1068; 1979.

The various classes of cellular genes that are associated with neoplasia are reviewed, with emphasis on their candidacy as subchromosomal targets of carcinogens. The methodology of the detection and mode of action of these genes is discussed as they relate to transformation. Although few carcinogen tests at present are aimed at single genes, it is felt that in the future they may and should be, since a strictly defined carcinogen-testing protocol would ultimately be easier to interpret for purposes of assigning human risk. The second part of this report is concerned with the question of inbred vs outbred mice as a model system for human carcinogenesis. Since outbred mice more closely approximate the polymorphic gene pool

of humans, these animals might be a better test population for carcinogens than inbred mice. Inbred mice are more likely to be fixed for genes that aggravate or abrogate carcinogenic action, leading to false results. The question of how inbred the supply-house-maintained populations of outbred mice become after decades of breeding in a controlled laboratory situation is addressed. A preliminary estimate of the nature and extent of genic variability of Swiss-Webster mice, compared with those of wild mouse and human populations, is given. (61 refs)

- 79-3003 The Predictive Value of Rodent Carcinogenicity Tests in the Evaluation of Human Risks.** (Eng) Tomatis, L. (Unit Chemical Carcinogenesis, International Agency for Res. on Cancer, 69372 Lyon Cedex 2, France). *Annu Rev Pharmacol Toxicol* 19: 511-530; 1979.

The use of rodent carcinogenicity tests to evaluate human cancer risks is reviewed. At least 26 chemical or industrial processes have proved or strongly suspected associations with cancer in humans, and data on the chemicals/processes are tabulated. For 5 processes in which the carcinogenic agent is not known, direct correlations between human and experimental animal (EA) data cannot be made; 4 compounds have not been adequately tested for carcinogenicity; for 15 chemicals, however, a correlation can be made between human and experimental data. These 15 compounds have the same target organ in humans as in at least one of the animal species tested. Multiple target organs were found more often in EA's than in humans. The major criticisms of experimental procedures have been that the route of administration does not correspond to the route by which humans are exposed; the doses sometimes greatly exceed the possible level of human exposure; and the experimental model is too sensitive. Results from a recent survey indicate that the sc route is as reliable as any other in predicting the carcinogenicity of a chemical given by other routes. Whenever a chemical was found to cause cancer in humans, it was because the incidence was exaggeratedly high or the type of tumor induced was rare. Thus, evidence of carcinogenicity in humans is obtained in situations that do not differ greatly from experimental situations, in which the sensitivity of the experimental model is maximized. According to revised International Agency for Research on Cancer (IARC) criteria, in the presence of adequate experimental data and in the absence of adequate human data, chemicals for which there is 'sufficient evidence' of carcinogenicity in EA's should be regarded for practical purposes as if they were carcinogenic in humans. Chemicals evaluated in IARC monographs 1-17 for which there is sufficient evidence of carcinogenicity in EA's are listed. (65 refs)

- 79-3004 A Critical Review: Will the Ames Salmonella Assay System be Used as A Screen for Presumptive Carcinogens?** (Eng) Jacobs, M. (American Inst. Physics, 335 East 45th St., New York, NY 10017). *J Environ Pathol Toxicol* 2(4): 1205-1217; 1979.

The use of the Ames *Salmonella typhimurium* assay as a screen for presumptive carcinogens is reviewed. It should be possible to reduce the incidence of human cancer appreciably by determining which substances in the environment are carcinogenic and then controlling their emission. The Ames test provides a quick, cheap technique by which manufacturers can identify at an early stage chemicals likely to be hazardous. Although the mutation origin of cancers remains an unproved hypothesis, mutagenesis in the Ames assay correlates well with the capacity to induce cancers in laboratory animals. A mutagenic activity ratio >2.5 indicates that the substance has a 95% probability of causing tumors in laboratory animals, whereas an activity ratio of <2.0 indicates that the substance has an 83% probability of being nontumorigenic in laboratory animals. After identifying presumptive carcinogens by the Ames test, one could then analyze human urine for their presence. Analyses of this type might identify individuals who do or do not metabolize carcinogens to the active compounds. The ultimate question is how the regulatory agencies are going to handle the Ames test. There is a growing consensus that a battery of short-term tests is needed and that multiple positive or negative tests would be necessary to define a chemical as a potential hazard or as non-mutagenic. Thus, to what extent the Ames test will be used as a basis for regulatory action is still uncertain. (11 refs)

- 79-3005 Statistical Issues in Interpretation of Chronic Bioassay Tests for Carcinogenicity.** (Eng) Gart, J. J. (Mathematical Statistics and Applied Mathematics Section, Biometry Branch, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014); Chu, K. C.; Tarone, R. E. *J Natl Cancer Inst* 62(4): 957-974; 1979.

The interpretation of chronic bioassay tests for carcinogenicity requires that the data be appropriately recorded. A "case history" for each animal links the pathology data for each organ of each animal to the length of its life. This information can be used in the interpretation of tumor incidences in light of the survival information. The role of historical controls was surveyed, and the use of significance tests in a multidisciplinary approach to the assessment of the pattern of tumor response was suggested. Multiple comparison methods valid for the interpretation of continuous (or measurement) data do not apply to the discrete data analyses used in these studies. The ideas and methods of these studies were applied to an animal study of chloroform. (31 refs)

- 79-3006 Cholesterol and Colon Cancer (Letter to Editor).** (Eng) Oliver, M. F. (Dept. Cardiology, Royal Infirmary, Edinburgh EH3 9YW, Scotland). *Lancet* 1(8122): 931-932; 1979.

The possibility that agents used to treat hypercholesterolemia may be related to the development of colorectal or other visceral cancers is discussed briefly. This possibility has been suggested by the results of small-scale studies in which patients with hypercholesterolemia were treated with clofibrate or with a polyunsaturated fat diet. (7 refs)

- 79-3007 What You Should Know about Estrogens or the Perils of Pauline.** (Eng) Landau, R. L. (Dept. Medicine, Univ. Chicago, 950 E. 59th St., Chicago, IL 60637). *JAMA* 241(1): 47-51; 1979.

The importance of weighing the benefits and risks of estrogen compounds for estrogen replacement therapy is pointed out. Although carefully executed retrospective studies have indicated that the sharply rising incidence of endometrial cancer is linked to the increased use of estrogens in postmenopausal women, it appears that the estrogen dose must be large, continuous, and administered over a long period of time. Smaller doses and cyclic administration apparently reduce cancer risk substantially. (20 refs)

- 79-3008 N-Nitroso Alkylating Agents: Formation and Persistence of Alkyl Derivatives in Mammalian Nucleic Acids as Contributing Factors in Carcinogenesis.** (Eng) Singer, B. (Virus Lab., Univ. California, Wendell M. Stanley Hall, Berkeley, CA 94720). *J Natl Cancer Inst* 62(6): 1329-1339; 1979.

The possibility that differences in the carcinogenicity of alkylating agents are due to differences in their chemical reactions with nucleic acids is discussed. Ethylnitrosourea reacts primarily with oxygens on RNA or DNA, whereas diethyl sulfate reacts almost exclusively with nitrogens. The oxygens are equally reactive in single- and double-stranded nucleic acids, and a good correlation appears to exist between oxygen affinity and the reported carcinogenicity of alkylating agents. When cells or animals were treated with a variety of alkylating agents, persistence of O⁶-alkyl guanine correlated with tumorigenic potential or UV repair deficiency. The available data suggest that the extent of reaction sites of DNA depends only on the alkylating agent used. Although a variety of metabolically activated carcinogens are ultimate alkylating agents, their sites of reaction in vivo generally differ from those of simple, direct-acting alkylating agents. The interaction of ultimate carcinogens with nucleic acids appears to be complex, and mutagenic reactions may play an important role if DNA

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modification is the initiating step in carcinogenesis. Experiments on mutations resulting from alkylation of nucleosides indicated that no predictions of base pairing can be made on the basis of chemical structure. It is likely that a given specific group of cells becomes malignant as a result of multiple events, most of which also occur in cells that do not become transformed. With respect to the persistence of specific alkyl derivatives formed in vivo, the removal of any alkyl base is not first order but is at least biphasic. Of the O-alkyl products, only the alkyl phosphotriesters are apparently completely stable in vivo. The alkyl bases are undoubtedly removed in vivo at a rate considerably exceeding that in vitro. (91 refs)

- 79-3009 Some Biochemical Aspects of N-Nitroso Compounds. (Eng) Digenis, G. A. (Div. Medicinal Chemistry, Coll. Pharmacy, Univ. Kentucky, Lexington, KY 40506); Issidorides, C. H. *Bioorg Chem* 8(1): 97-137; 1979.

The occurrence, carcinogenicity, chemistry, formation, inhibition, chemical reactivity, determination, and metabolism of the N-nitroso compounds are reviewed. Most chemical carcinogens produced alkylating or arylating species. The main site of base alkylation in nucleic acids by nitroso compounds is the N-7 position of guanine, but there is no correlation between alkylation at this position and carcinogenicity or mutagenicity. A much better correlation exists with alkylation at the O-6 position of guanine. Of all the chemical carcinogens, only the nitrosamides and some alkylating agents are chemically reactive and do not require enzymic activation to exert their toxic effects. The nitrosamines persist in the body unchanged for much longer periods and require metabolic activation. Thus, the nitrosamines produce cancer only in organs that are capable of metabolizing them. The simplest and some of the higher dialkylnitrosamines are metabolized predominantly in the liver and are activated by α -hydroxylation. The higher dialkylnitrosamines can also be activated by other pathways. It is possible that sulfate or phosphate conjugates of β -hydroxyalkylnitrosamines could serve as direct-acting in vivo alkylating agents. The diversity of the metabolic activation of the higher dialkylnitrosamines is further evidenced by the occurrence of ω - and (ω -1)-hydroxylations. The multiple carcinogenic effects on different organs may be due to the varied metabolic activation of the nitrosamines. There is also evidence that metabolic pathways other than those leading to the formation of alkylating intermediates may exist for N-nitroso compounds. (174 refs)

- 79-3010 Nitrosamines in Cosmetics. (Eng) Anderson, G. A. (Dow Chemical Co., Midland, MI). *Cosmetics Toiletries* 94(5): 65-68; 1979.

The problem of nitrosodiethanolamine (NDEA) contamination in cosmetics is discussed. Analysis of production grade samples of triethanolamine for NDEA contamination gave negative results. Possible sources of nitrite contamination, ie, the drums, cosmetic ingredients, water, and air, are being investigated. (22 refs)

- 79-3011 Food. (Eng) Yeransian, J. A. (General Foods Technical Center, White Plains, NY 10625); Sloman, K. G.; Foltz, A. K. *Anal Chem* 51(5): 105R-134R; 1979.

An extensive literature review concerning advances made in the analysis of foods between 1976 and 1978 is presented. Specific topics include additives; adulteration, contamination, and decomposition; carbohydrates; color; enzymes; fats, oils, and fatty acids; flavors and volatile compounds; organic acids; nitrogen; and vitamins. (1,147 refs)

- 79-3012 Identifying Environmental Chemicals Causing Mutations and Cancer. (Eng) Ames, B. N. (Dept. Biochemistry, Univ. California, Berkeley, CA 94720). *Science* 204(4393): 587-593; 1979.

The identification of environmental chemicals that cause mutations and cancer is reviewed. Damage to DNA appears to be the major cause of most cancers and genetic birth defects, and it may contribute to aging and heart disease. Many of these agents are natural chemicals present in the human diet as complex mixtures. Because of the time, expense, and difficulty of dealing with complex mixtures, existing animal tests and human epidemiology studies are inadequate for identifying the environmental chemicals that cause this damage. Over 50,000 chemicals are currently used in significant quantities, and approx 1,000 new chemicals are introduced each year. The *Salmonella* assay and other short-term assays that have been developed for determining the mutagenicity of chemicals or their ability to interact with DNA should prove useful in detecting chemical mutagens. A very high percentage of carcinogens tested in the *Salmonella* assay are mutagens, and most mutagens appear to be carcinogens. Among the mutagens subsequently found to be carcinogens are furylfuramide, ethylene dichloride, ethylene dibromide, tris(2,3-dibromopropyl)phosphate, and oxidative-type hair dyes. Much of the cancer occurring today appears likely to be due to the ingestion of natural carcinogens in the diet. Since each short-term test detects a few carcinogens that the others do not, a battery of short-term tests is now favored by many investigators. A knowledge of carcinogenic potency would be an important aid in assessing human risk. The short-term tests could be used to establish priorities among the many mutagens that have never been tested in animal cancer models and to which there is significant human exposure. (57 refs)

- 79-3013 Deadly Legacy: Dioxin and the Vietnam Veteran.** (Eng) Thomasson, W. A. *Bull At Sci* 35(5): 15-19; 1979.

Dioxin, a contaminant of a herbicide [(2,4,5-trichlorophenoxy)acetic acid] widely used in Vietnam, apparently caused numbness and tingling in the hands and feet, depression, irritability, memory loss, and even cancer in veterans 8-12 yr after their last exposure. Some had fathered deformed children. Extremely small amounts of dioxin caused death in mice and rats, and rats fed a diet with as little as 5 parts per trillion dioxin developed tumors. The chemical also produced mutations in *Salmonella* and other bacteria. (13 refs)

- 79-3014 Lung Cancer due to Chemicals.** (Eng) Weiss, W. (Hahnemann Medical Coll. and Hosp., Philadelphia, PA). *Compr Ther* 5(2): 18-23; 1979.

Lung cancer due to exposure to coke oven fumes, arsenic, nickel, chromate, chloromethyl ethers, gas, and mustard gas is reviewed. In a study of coke oven workers, the relative risk of lung cancer was 6.9 for men with ≥ 5 yr of employment on top of ovens, 2.1 for men with ≥ 5 at the side of ovens, and 1.7 for men with < 5 yr exposure. In a group of 173 exposed and 1,809 unexposed arsenical workers, the relative risk was dose-related and elevated, reaching 7.0 for those exposed for > 8 yr to compounds containing an equivalent level of $1 \text{ mg/m}^3 \text{ As}$. In a study of smelter workers, there was an inverse relationship between carcinogenic dose of As and induction period. Studies of men exposed > 5 yr to Ni indicate that the elevated risk of nasal and lung cancer is limited to workers exposed prior to 1925. The form of Ni that is carcinogenic in humans is not known. Some studies of chromate carcinogenicity in the respiratory tract have shown very high relative risks. There are no adequate data for a dose-response relationship. The relative risk from exposure to bis(chloromethyl) ether and/or or chloromethyl methyl ether was 29.1 over a 10-yr observation period, and there was a strong dose-response relationship: for 20 chemical workers with heavy cumulative exposure, the relative risk was 77.4. There was an inverse relationship between cigarette smoking and lung cancer incidence in workers exposed to chloromethyl ethers. The relative risk of lung cancer following exposure to mustard gas was 36.7. The occurrence of bronchogenic carcinoma at a relatively early age (< 55 yr) should raise suspicion of exposure to a potent environmental carcinogen. (15 refs)

- 79-3015 Cancer Incidence (Letter to Editor).** (Eng) Wilson, R. (Energy and Environmental Policy Center, Harvard Univ., Cambridge, MA 02138). *Science* 203(4387): 1293; 1979.

If there is a proportional relationship between cancer incidence and dose, then exposure of more people to fixed dose will produce either the same total number of cancers spread over more persons (if there is no threshold dose) or fewer total cancers (if there is a threshold dose). This argument is applied to the current popularity of self-service gas stations and the exposure of customers to the gasoline additive ethylene dibromide. (no refs)

- 79-3016 Dose Comparisons in the Effects of Radiation and of Chemical Pollutants.** (Eng) Glubrecht, H. (Institut für Biophysik der Universität Hannover, Hannover, W. Germany). *Atomkernenergie Kerntechnik Bd* 33(2): 126-129; 1979.

Present knowledge of the effects of chemical pollutants (particularly carcinogenesis, mutagenesis, and teratogenesis) is far behind knowledge concerning the effects of ionizing radiation. If the action of a chemical pollutant can be represented by a dose-response curve, the rad-equivalent of a given dose of the chemical can be expressed by the product of concentration and time. (no refs)

- 79-3017 Materials for Human Implantation.** (Eng) Rostoker, W. (Univ. Illinois, Chicago Circle, Chicago, IL 60680); Galante, J. O. *Trans Am Soc Mech Eng* 101(1): 2-14; 1979.

The characteristics of materials used in devices to maintain or restore body functions are reviewed. A long-term complication of the use of artificial materials in the human body is tumor development. Rodent studies show that the chemical reactivity, physical characteristics, geometry, and surface texture of the material may be important in this type of carcinogenesis. Differences in the carcinogenicity of various substances may be related to whether or not the implant can be entirely encapsulated in a fibrous sheath. Available data indicate that polyethylene particles shed from polyethylene prostheses in clinical use are not carcinogenic in humans or animals. (103 refs)

- 79-3018 Radiotherapeutic Agents: Properties, Dosimetry, and Radiobiologic Considerations.** (Eng) Saenger, E. L. (Radioisotope Lab., Cincinnati General Hosp., Cincinnati, OH 45267); Kereiakes, J. G.; Sodd, V. J.; David, R. *Semin Nucl Med* 9(2): 72-84; 1979.

The properties, dosimetry, and radiobiologic characteristics of radiotherapeutic agents are discussed. Various factors have tended to decrease interest in radionuclides for therapy in recent years. The radiation dose to target tissue depends on biologic (the time concentration

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of radioactivity within the target) and physical (the type and energy of the ionizing radiation in the radioactive decay) parameters. A formula for calculating radiation dose estimates to total body and specific organs for administered radionuclides is given. Radiobiologic considerations include the possibility of early deleterious effects from overdosage and chromosomal changes in circulating lymphocytes. Carcinogenesis, a possible late effect, has been minimal in patients receiving therapeutic levels of radioactive drugs. Genetic and developmental effects have also been negligible. Complications encountered more frequently have been leukemia after extensive therapy of thyroid carcinomas, and local fibrosis has developed after direct injection of radioactive colloids into tumor tissue. (55 refs)

79-3019 NAS Study on Radiation Takes the Middle Road. (Eng) Marshall, E. (No affiliation given). *Science* 204(4394): 711-714; 1979.

Most members of the committee on the Biological Effects of Ionizing Radiation have concluded that injury from and exposure to radiation decrease at the same rate. Six members, however, filed a dissenting opinion, claiming that the majority report was alarmist. This group believes that as exposure decreases, injuries taper off more rapidly. They also claim to have found errors in some of the data supporting the majority conclusion. (no refs)

79-3020 Biological Effects of ^{125}I . (Rus) Kalistratova, V. S.; Tishchenko, G. S. *Med Radiol (Mosk)* 24(4): 69-72; 1979.

Available literature on the biological effects of ^{125}I are reviewed, and the data are compared with those for ^{131}I . Administration of 200, 140, and 92 μCi ^{131}I to mice resulted in atrophy of the thyroid, whereas administration of 400 μCi ^{125}I did not. (25 refs)

79-3021 Purification and Assay of Murine Leukemia Viruses. (Eng) Sherr, C. J. (Lab. Viral Carcinogenesis, NCI, Bethesda, MD 20014); Todaro, G. J. *Methods Enzymol* 48: 412-424; 1979.

Techniques for growing murine leukemia viruses (MuLV) in tissue culture, methods for purifying virus in sufficient quantities for biochemical studies, and quantitative in vitro assays for viral replication are described. Among the latter is the supernatant reverse transcriptase assay, which depends on the packaging of RNA-dependent DNA polymerase activity in extracellular C-type viral particles. A linear correlation generally exists between the amount of reverse transcriptase activity and the number of infectious

particles. The most useful viral structural proteins for radioimmunoassays (RIA) are p30 and gp70; a representative RIA protocol for MuLV p30 is described. In the mixed culture cytopathogenicity test, XC cells infected with MuLV's undergo syncytium formation resulting in the appearance of plaques. The number of plaques is a direct function of the number of infectious viral particles. The Moloney sarcoma-positive, leukemia-negative strain of mouse sarcoma virus can transform susceptible cells from a variety of mammalian species. In the secondary focus-formation test, several nonproducer lines show plaque formation when infected by MuLV's, and the number of plaques is directly proportional to the infectious virus concentration. (34 refs)

79-3022 Latent Infection Caused by Herpesvirus. (Rus) Raichani, I. (Inst. Virology, Bratislava, Czechoslovakia); Santo, I. *Vopr Virusol* (2): 99-105; 1979.

Current data on the structure and replication of herpes simplex virus during nonproductive interaction with the host cell are reviewed. It is suggested that latent infection without virus release can undergo transition into persistent infection and vice versa. (72 refs)

79-3023 The Structure and Isomerization of Herpes Simplex Virus Genomes. (Eng) Roizman, B. (Marjorie B. Kovler Viral Oncology Lab., Univ. Chicago, Chicago, IL 60637). *Cell* 16(3): 481-494; 1979.

The structural properties of herpes simplex virus (HSV) DNA's are discussed. HSV-1 and HSV-2 DNA's are linear double-stranded molecules approx 100×10^6 in mol wt, and they have a base composition of 67 and 69 G + C mole %, respectively. The DNA's are fragmented upon denaturation with alkali. The sequence arrangement of HSV DNA consists of two covalently linked long and short components (designated L and S) that comprise 82% and 18% of the viral DNA, respectively. Each component consists of largely unique sequences bracketed by inverted repeats. The L and S components can invert relative to each other, so that DNA extracted from wild-type virions consists of four equimolar populations differing from each other solely in the orientation of the L and S components. The origin of the inversions is discussed in detail. Apparently, the four isomers of HSV-1 DNA are not functionally equivalent, although there is evidence that the terminal portions of the reiterated sequences in the L and S components are obligatorily identical. It is possible that HSV DNA has two sites for the initiation of DNA synthesis, one in the S and one in the L component. The DNA appears to be replicated by a rolling circle mechanism. A model of DNA replication is presented, and predictions based on it are evaluated. The hypothesis that HSV originates by the fusion of two genomes, each bracketed by its own inverted repeats, seems plausible. (59 refs)

- 79-3024 Burkitt's Lymphoma and Infectious Mononucleosis.** (Eng) Wright, D. H. (Univ. Southampton Medical Sch., Southampton SO9 4XY, England). *Compr Immunol* 4: pp. 391-424; 1978.

Evidence linking Epstein-Barr virus (EBV) with infectious mononucleosis (IM) and Burkitt's lymphoma (BL) is reviewed. The anatomical distribution of BL in most patients is strikingly different from that seen in other malignant lymphomas. For example, in Ugandan patients, one or more quadrants of the jaw is involved in 50% of the patients (max occurrence at age 3 yr), and 4/5 women with BL have ovarian tumors (invariably bilateral) and 4%-10% men have testicular tumors (bilateral in one-third of cases). The most striking morphologic feature of IM is the wild hyperplasia of lymphoreticular elements. EBV may be identified in cells by the isolation of virus particles; virus capsid antigen (VCA); EBV-determined membrane, nuclear, and early antigens; and soluble complement-fixing antigen. Most African BL cases are EBV genome-negative. Antibodies to EBV-determined antigens can be detected in many patients with IM and BL, as can cell-mediated reactivity against EBV-infected cells and antibody-dependent lymphocyte cytotoxicity. The excellent response to chemotherapy and the long-term remission seen in a high proportion of BL patients could be related to host immunologic reactivity to the tumor cells. Raised antibody levels to EBV-associated antigens are also encountered in many patients with Hodgkin's disease and approx 40% of patients with chronic lymphocytic leukemia. However, the tumor cells in BL are almost invariably EBV genome-positive, whereas those in other lymphomas (except for 1 case of immunoblastic lymphadenopathy) are EBV genome-negative. New World, but not Old World, monkeys are also susceptible to EBV infection; they may develop lymphoma, a syndrome similar to IM, or no apparent infection. Although EBV and malaria are associated with BL, the possibility of a third factor in the etiology of the disease or the possibility that EBV and/or malaria are not etiologic factors must be considered. (218 refs)

- 79-3025 Use of Nude Mice for Tumorigenicity Testing and Mass Propagation.** (Eng) Shin, S. (Dept. Genetics, Albert Einstein Coll. Medicine, Bronx, NY 10461). *Methods Enzymol* 48: 370-379; 1979.

A review of procedures involved in the use of nude mice for tumorigenicity testing and mass propagation includes the source, maintenance, and breeding of nude mice; cellular tumorigenicity testing; testing for mycoplasma infection; mass production of cells in nude mice; and reestablishment of cell cultures from tumors. (22 refs)

- 79-3026 Carcinogenesis Effects of Combined Treatment: Immunologic Factors.** (Eng) Pomeroy,

T. C. (Div. Radiation Therapy, Univ. Texas Health Science Center, San Antonio, TX 78284). *Front Radiat Ther Oncol* 13: 194-214; 1979.

The role of immunologic alterations due to combined modality therapy in the development of malignant disease is reviewed. Spontaneous tumor formation and metastasis were enhanced in mice treated with cortisone acetate (CA). Manipulation of the reticuloendothelial system by CA, selected chemotherapeutic agents, and a nonspecific reticuloendothelial stimulant resulted in atrophy of the reticuloendothelial system. The degree of immune suppression also depends on the specific antigenicity of the individual tumor cells. Immune suppression by CA can interfere with the effects of radiation in tumor control, and the distribution of varying concentrations of chemotherapeutic agents within the tissues in a radiation field may become a critical factor. It appears that cortisone immunosuppression reduces the number of lymphomas that develop. Whereas the increase in solid tumors in transplant patients can be explained by immunosuppression, the increased incidence of reticulum cell sarcoma by any form of immunosuppression, including steroids, cannot be fully explained or correlated with the present data. It appears that the major effect of immune suppression on tumor incidence is a marked shortening of the latent period for tumor development. (40 refs)

- 79-3027 Do Macrophages Destroy Nascent Tumors?** (Eng) Adams, D. O. (Dept. Pathology, Duke Univ. Medical Center, Durham, NC 27710); Snyderman, R. *J Natl Cancer Inst* 62(6): 1341-1345; 1979.

The proposition that macrophages (MP's) play a central role in immune surveillance is discussed. Abundant evidence exists that MP's destroy neoplasms in vivo if the MP's can be attracted in large numbers by T or B lymphocytes and activated. MP's can also selectively recognize and destroy neoplastic cells in vitro. They bind tumor cells to their surface more than they bind normal cells and to a greater extent than nonactivated MP's bind either normal or neoplastic cells. It is proposed that tissue MP's recognize small clones of newly developed tumor cells, cluster about them, and destroy the nascent tumor to effect surveillance. The extremely small burden of tumor cells is recognized and destroyed by the MP's without need of amplification by other components of the immune system. The available data support but do not controvert the hypothesis that MP's exert surveillance. The development of a neoplasm does not abrogate the idea of surveillance. Clinically apparent neoplasms may be those that have developed the means to subvert MP functions. Evidence exists that tumors and carcinogens directly depress the function of MP's, and the ability of chemical carcinogens and tumor promoters to suppress MP function may be necessary to their carcinogenic potential. (81 refs)

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- 79-3028 Cancer after Cardiac Transplantation (Letter to Editor).** (Eng) Goldsmith, A. E. (Natl. Cancer Cytology Center, Melville, NY); Ryan, G. F.; Joseph, A. B. *Br Med J* 1(6171): 1146-1147; 1979.

Various animal models in which malignant lymphoma develops as a late sequel of transplantations in the absence of exogenous immunosuppressants are discussed. Chronic antigenic stimulation appears to be a possible common factor. Only lymphoid neoplasms developed, despite the grafting of four different tissues—lymph node, mammary gland, skin, and bone marrow. Bone marrow was the only tissue that did not induce lymphoma in its recipients, even following exposure to a nonphysiological environment. This raises the question whether the T lymphocytes in the graft tissues are involved in the malignant processes in these animal models. (5 refs)

- 79-3029 Immunosuppression and Malignant Disease.** (Eng) Penn, I. (Dept. Surgery, Univ. Colorado Sch. Medicine and Veterans Admin. Hosp., Denver, CO 80220). *Compr Immunol* 4: pp. 223-237; 1978.

The markedly increased incidence of neoplasms in immunosuppressed organ transplant recipients is surveyed. Many of the tumors are low-grade lesions of the skin, lip, and uterine cervix, but almost 26% are highly lethal lymphomas, compared with a 3% incidence in the general population. The lymphomas occur at a young age (av 36 yr) and after a relatively short induction time following transplantation (av 23 mo). Hodgkin's disease is rare, the preponderant type of lymphoma being reticulum cell sarcoma. Many of these tumors have the morphologic features of antigen-activated lymphocytes and can be classified as 'immunoblastic sarcomas.' The CNS is involved in nearly half the organ homograft patients with lymphomas other than Kaposi's sarcoma, and, in many instances, the lesions are confined only to the brain and spinal cord. The homograft is involved by lymphoma in 21% of cases. The cause of the increased incidence of malignancies in organ homograft recipients is unclear, but a complex interplay of multiple factors is probably responsible. Depression of the immunosurveillance function of the lymphoreticular system or immunosuppressive agents alone are probably not responsible for the increased incidence of cancer. The possible role of viruses and chronic antigenic stimulation of the lymphoid system by foreign histocompatibility antigens of the homograft must be considered. (80 refs)

- 79-3030 Immunodeficiency Diseases and Malignancy.** (Eng) Spector, B. D. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455); Perry, G. S.; Good, R. A.; Kersey, J. H. *Compr Immunol* 4: pp. 203-222; 1978.

The relationship between immunodeficiency (ID) and malignancy is reviewed based on data from 227 cancer patients with diagnosed ID diseases. Seven ID states are represented: X-linked (Bruton's) hypogammaglobulinemia (HGG: 12 cases); IgA deficiency (13); IgM deficiency (7); variable ID (70); severe combined ID (11); ataxia-telangiectasia (79); and Wiskott-Aldrich syndrome (35). Lymphoreticular tumors represented 129/227 total malignancies, and they were the most prevalent neoplasms in 5/7 ID diseases; in the two exceptions, IgA deficiency and X-linked HGG, lymphoreticular tumors accounted for 31% and 33% of the total, respectively. Differences in malignancy patterns were found in the distribution of epithelial cancers and leukemias: with IgA deficiency, 54% of all cancers were epithelial, and leukemias comprised 58% of all reported cancers with X-linked HGG. The cancers showed unique sex- and age-related characteristics. Also, a surprisingly large number of these patients developed gastrointestinal malignancies, particularly stomach cancers. The distribution of cancers by age and primary site indirectly supports the conclusion that immunodeficient patients are at greatly increased risk for development of cancer, despite a shortened life-span (2-20 yr). Hypotheses of the mechanisms of oncogenesis involving the lymphoid system fall into at least two general categories: the immune system in persons with genetically induced ID's has an increased likelihood of establishing lines of malignant cells; and the immune system in these diseases has decreased ability to eliminate malignant cells as they develop. The possible role of immunologic surveillance mechanisms in the defence against malignant cells has become a matter of increasing skepticism. (76 refs)

- 79-3031 α -Chain Disease: A Possible Model for the Pathogenesis of Human Lymphomas.** (Eng) Seligmann, M. (Lab. Immunochemistry and Immunopathology (INSERM U 108), Res. Inst. on Blood Diseases, Hopital Saint-Louis, Paris 10, France); Rambaud, J. C. *Compr Immunol* 4: pp. 425-447; 1978.

Various aspects of α -chain disease (CD), a proliferative disorder of B lymphoid cells involving primarily the IgA secretory system, are reviewed, along with its possible use as a model for the pathogenesis of human lymphomas. CD appears to proceed in two stages: the early stage is characterized by a possible nonmalignant diffuse and extensive plasma cell infiltration; the later stage is characterized by overt malignancy and supervening immunoblastic lymphoma. The diagnosis of CD relies entirely on laboratory studies, including immunochemical analysis of serum proteins. CD involves the production of molecules consisting of incomplete heavy α chains and no L chains. The striking and unexpected electrophoretic heterogeneity of these presumably monoclonal proteins may be related to their high carbohydrate content and tendency to polymerize. CD may involve a primary internal

deletion followed and obscured by a secondary proteolysis of limited degree. Intestinal CD usually presents with a severe malabsorption syndrome characterized by chronic diarrhea, steatorrhea, abdominal pain, and vomiting. Hepatosplenomegaly and peripheral lymphadenopathy are rare at presentation. Tetany is observed frequently, and it may be the major presenting symptom. The intestinal lesions of CD usually predominate in the duodenum and jejunum. There is also a less common respiratory form of the disease. Most CD patients are 10-30 yr old, and the male:female ratio is 3:2. Most patients originated from and had been living in areas with a high degree of infestation by intestinal microorganisms and were exposed to poor hygiene conditions in low socioeconomic circumstances. Most diffuse 'Mediterranean lymphomas' probably represent the late malignant phase of CD. Environmental factors providing a local and protracted antigenic stimulation may play an important role in the pathogenesis of the disease. Predisposing genetic factors may also exist, and the postulated environmental antigenic stimulus might be associated with an underlying immunodeficiency. (55 refs)

- 79-3032 Ecotaxis, Ecotaxopathy, and Lymphoid Malignancy: Terms, Facts, and Predictions.** (Eng) de Sousa, M. (Sloan-Kettering Inst. Cancer Res., New York, NY 10021). *Compr Immunol* 4: pp. 325-359; 1978.

The relationship of ecotaxis and ecotaxopathy to lymphoid malignancy are reviewed. Ecotaxis is the capacity of mature lymphomyeloid cells to arrange themselves in microenvironments of the peripheral lymphoid organs. For both T and B cells, ecotaxis involves circulation from blood to lymph and includes passage through large nonlymphoid capillary filters in the lungs and liver and slow penetration through distinct territories of the peripheral lymphoid organs. The position occupied by a cell in a lymphoid organ reflects a series of interactions with obvious and underlying neighbor cells. Thus, the accumulation or proliferation of one malignant cell type exemplifies a defect in these interactions as well as a defect in the malignant population itself. Since the spleen is one of the major sequestration sites of abnormal circulating cells, splenectomy will predictably help improve cell maldistribution. Ecotaxopathy is abnormal ecotaxis and is due to failure of circulating cells to migrate and occupy their normal environment. Experimental and human lymphoid malignancies are discussed to illustrate how progressive depletion of one lymphoid cell population in its normal environment could lead to the development of malignancy among its usual neighbors. These modified cell interactions may offer an explanation for some anomalies of immunologic function that are often associated with lymphoid malignancy. Any attempt to investigate a disease of the lymphoid system is incomplete if only one compartment is analyzed. The quality of therapy reflects understanding of disease pathogenesis. If the pathogenesis of lymphoid malignancy

is associated with the failure of neighboring cells to control the traffic and proliferation of malignant cells, therapy designed to eliminate the accumulating cells may only be temporarily beneficial. (151 refs)

- 79-3033 Immunodeficiencies Associated with Chronic Lymphocytic Leukemia and Non-Hodgkin's Lymphomas.** (Eng) Gupta, S. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Good, R. A. *Compr Immunol* 4: 565-583; 1978.

The involvement of humoral immunity, cellular immunity, the phagocytic cell system, and amplifying systems in chronic lymphocytic leukemia and non-Hodgkin's lymphomas is discussed. Patients with non-Hodgkin's lymphomas often demonstrate an impairment of both cellular and humoral immunity that is usually minimal in early stages of localized disease, but becomes more pronounced with progression of the disease. Defects are usually more pronounced in patients with so-called reticulum cell sarcoma than in patients with lymphosarcoma. A depressed in vitro blastogenic response to mitogens is accompanied by abnormal glucose metabolism of lymphocytes. Reticuloendothelial phagocytic functions are generally intact. Familial studies demonstrate an apparent genetic relationship between immunologic abnormalities and susceptibility of lymphoid tissue to neoplasia. (135 refs)

- 79-3034 Experimental Models of Lymphoid Malignancies.** (Eng) Peterson, R. D. (Dept. Pediatrics, Univ. South Alabama Coll. Medicine, Mobile, AL 36617). *Compr Immunol* 4: pp. 81-127; 1978.

The experimental models of lymphoid malignancies are discussed. Thymic-type lymphomas are composed of apparently immature lymphocytes and are often characterized by abnormalities in chromosome number and morphology. The nonthymic lymphomas are often pleomorphic in cellular composition, and the reticulum cell sarcomas seem to preferentially involve the spleen, liver, and mesenteric lymph nodes. Lymphomas can be identified as belonging to the T- or B-cell category, although several experimental lymphoid malignancies do not show characteristics of either. Multigenic and nongenetic factors influence the process whereby a certain genetic configuration leads to a lymphoid malignancy; the malignancy may be incited by chemicals, viruses, radiation, or hormones. Tumor viruses may be acquired by an animal via vertical or horizontal transmission. Current knowledge of the molecular characteristics of these viruses exceeds knowledge of their relationship to tumors. The mechanism whereby radiation, or its associated oncornavirus, leads to lymphoma in experimental animals is also not known. There is some evidence that a virus is involved in the pathogenesis of chemically induced lymphomas. Various

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manipulations of the immune system have led to increased incidences of lymphoid malignancies, and evidence exists that hormones can significantly influence such malignancies. At least some lymphoid malignancies appear to have hormonal dependence. Both the thymus and bursa (in chickens) appear to have central roles in the development of lymphoid malignancies. Although immunological mechanisms can deter the development and survival of malignant cells, the immune response may accelerate tumor growth in some situations. (591 refs)

- 79-3035 The Pathology of Lymphoreticular Neoplasms.** (Eng) Lukes, R. J. (Dept. Pathology, Univ. Southern California Sch. Medicine, Los Angeles, CA); Parker, J. W. *Compr Immunol* 4: pp. 239-279; 1978.

The pathology of lymphoreticular neoplasms is reviewed. Malignant lymphomas are now acknowledged to be neoplasms of the immune system, and they are related to the T- and B-cell systems and alterations in lymphocyte transformation. The neoplasms can be classified into five major groups: U cell (undefined), T cell, B cell, histiocytic, and Hodgkin's disease (cell of uncertain origin). A multiparameter investigative approach to the redefinition of these disorders as related to the T- and B-cell systems is outlined. Studies of over 300 cases of non-Hodgkin's lymphomas and related leukemias revealed that the majority (approx 85%) marked as T cells (20%) or B cells (65%). The exceptions were some of the acute lymphocytic leukemias (ALL's) and a rare case of apparently true histiocytic neoplasm. The cytomorphology of the various types was predictive of their T- and B-cell nature, but the reliability of this approach was dependent on carefully collected and prepared histologic sections and an experienced observer. The data obtained, and those of other investigators, permitted the identification of several new homogeneous cytological types that are emerging as clinical-morphologic-immunologic entities. These include the convoluted T-cell lymphoma-leukemia that interrelates with T-cell ALL, the plasmacytoid lymphocytic lymphoma commonly associated with monoclonal gammopathies, and the cerebriform T-cell lymphoma associated with Sezary's syndrome and mycosis fungoides. (93 refs)

- 79-3036 Clonal Origin of Human Tumors.** (Eng) Fialkow, P. J. (Medical Genetics Section, Medical Service, Veterans Admin. Hosp., Seattle, WA 98195). *Annu Rev Med* 30: 135-143; 1979.

Studies of the neoplasms that arise in patients with mosaicism provide important insights into stem cell relationships and factors involved in the initiation and progression of the tumors. At present, genetic marker approaches to the study of tumor development are essentially limited to

neoplasms that either synthesize immunoglobulin or arise in glucose-6-phosphate dehydrogenase (G-6-PD) heterozygotes. G-6-PD studies in chronic myelocytic leukemia (CML), polycythemia vera (PV), and idiopathic myeloid metaplasia with myelofibrosis have shown that these disorders involve pluripotent hematopoietic stem cells, and they suggest that at the time of diagnosis, the disorders probably have a clonal origin. However, fibroblasts cultured from the marrows of such patients do not arise from the abnormal stem cell clones. Although there is no evidence for residual normal committed granulocyte stem cells in CML, there are normal, but suppressed, stem cells in PV. Most benign and malignant tumors have been found to have a clonal origin at the time of examination, but one case of colon carcinoma, some invasive carcinomas of the cervix, some spontaneous parathyroid adenomas and multiple neurofibromatosis appear to have a multicellular origin. The malignant putative viral disease Burkitt's lymphoma is of clonal origin, whereas the benign viral growth "veneral" wart is of multicellular origin. Virus-infected cells appear to give rise to a malignant clone only if one or more additional events occur. (20 refs)

- 79-3037 Psoriasis and Cancer.** (Eng) Shuster, S. (Dept. Dermatology, Univ. Newcastle upon Tyne, Newcastle upon Tyne NE1 4LP, England); Chapman, P. H.; Rawlins, M. D. *Br Med J* 1(6168): 941-942; 1979.

It is hypothesized that the low incidence of skin cancer in psoriasis patients is due to a decreased capacity of psoriatic skin to metabolize carcinogens. Although psoriasis is treated by the topical application of tars, which are carcinogenic, and with ultraviolet B radiation, which is a major cause of skin cancer in healthy people, no increase in skin cancer incidence has been reported in chronic psoriasis. Epidermis from psoriasis patients shows reduced basal and induced aryl hydrocarbon hydroxylase (AHH) activity, both in the lesions and in lesion-free skin. If the hypothesis is correct, it would provide strong support for the idea that epidermal AHH activity is a major determinant of the development of skin cancer. The hypothesis also raises the possibility that the incidence of systemic cancer may be lower in psoriasis patients if the impaired AHH activity in psoriatic skin is shared by other tissues. Since psoriasis is probably transmitted as a simple dominant trait, it may have persisted because it confers a genetic advantage; ie, the decreased AHH activity of psoriatic skin, which reduces the incidence of cancer of the skin and perhaps of other organs. (15 refs)

- 79-3038 Premalignant Lesions of the Stomach.** (Eng) Gedigk, P. (Inst. Pathology, Univ. Bonn, Bonn, W. Germany); Bechtelsheimer, H.; Mueller-Wallraf, R. *Isr J Med Sci* 15(4): 405-409; 1979.

The histopathogenesis of the three major types of gastric carcinoma—intestinal, diffuse, and indeterminate—is reviewed. The gastric mucosa responds to any sustained irritation or damage with a proliferation of glandular neck cells and, eventually, a progressive decrease in differentiation (metaplasia) and the formation of intestinal-type glands. The rapidly transforming cells of the glandular neck are the probable site of neoplastic mutation, leading to persistent histologic abnormality. Long-lasting mucosal damage leads to a replacement of normal resting glandular cells by actively proliferating ones. With increased and continuing proliferative activity, carcinomatous polypoid protrusions may develop. Intestinal transformation usually involves only one of the many branches of the pyloric glands. Continuous growth and lack of cellular loss by shedding may lead to the formation of cysts deep in the mucosa and, occasionally, the submucosa. Invasive carcinoma eventually develops from these cysts. The four early stages of intestinal carcinoma belong to the category of intestinal metaplasia. Stage 3 is a borderline cancerous lesion and Stage 4 represents a high probability of carcinoma; Stage 5 represents the classic intestinal type of gastric carcinoma. The initial lesion in the development of a diffuse carcinoma is an atypical transformation of the lower part of the glandular neck (glandular neck dysplasia). The signet ring cell subtype starts with the development of clusters of cells in the glandular neck; in the anaplastic subtype, the detaching carcinoma cells of the glandular neck dysplasia are undifferentiated. The genesis of the early stages of indeterminate gastric carcinoma is not known. (52 refs)

- 79-3039** Some Leads to the Etiology of Cancer of the Large Bowel. (Eng) Hill, M. J. (No affiliation given). *Surg Annu* 10: 135-149; 1978.

Large bowel cancer is common in Western countries and is relatively rare in developing countries. It appears to be related to diet, particularly to meat or fat. Evidence from population studies in various parts of the world and from a retrospective case-control study supports the hypothesis that the Western diet results in a high concentration of bile acids in the large bowel and that these acids are metabolized by the gut bacteria to carcinogens or cocarcinogens. (88 refs)

- 79-3040** Epidemiology of Primary Neoplasms of Lymphoid Tissues in Animals. (Eng) Hardy, W. D. (Lab. Veterinary Oncology, Memorial Sloan-Kettering Cancer Center, New York, NY 10021). *Compr Immunol* 4: pp. 129-180; 1978.

The occurrence of lymphoid tumors in animals is reviewed. The etiology of many animal lymphosarcomas (LSA's) is unknown, and the only etiological agents that have been discovered to date are viruses. The oncogenic RNA viruses

(oncornaviruses) can be transmitted vertically via the genes or horizontally by contagion. Avian and mammalian cells are resistant to superinfection with, and replication of their own endogenous oncornaviruses. Endogenous and exogenous oncornaviruses of cats and nonhuman primates can be distinguished serologically, and only the exogenous oncornaviruses induce LSA's in these species. Herpesviruses are the only DNA viruses associated with lymphoid tumors. They can be transmitted by contagion or transplacentally, but apparently not genetically. The epidemiology of naturally occurring LSA in mollusks, fish, amphibians, reptiles, chickens, mice, rats, hamsters, guinea pigs, cattle, sheep, horses, swine, dogs, cats, and primates is discussed. When possible, the incidence, clinical findings, tissues involved, etiology, epidemiology, seroepidemiology, prevention, and public health aspects of LSA's in each species are reviewed. Application of the findings to human LSA's is briefly considered. (250 refs)

- 79-3041** Epidemiology of Lymphoreticular Malignancies in Man. (Eng) Vianna, N. J. (Cancer Control Bureau, New York State Dept. Health, Albany, NY 12237). *Compr Immunol* 4: pp. 181-201; 1978.

Aspects of the epidemiology of lymphoreticular malignancies in humans are reviewed. International and regional variations in the incidence patterns of these diseases, particular Burkitt's lymphoma (BL), suggest that environmental factors may be important in their etiology. Lymphomas appear to be most prevalent among the more prosperous members of society and more common in urban than rural dwellers. In the US, men are affected more often than women, and marital status appears to influence the frequency of Hodgkin's disease (HD) specifically. Much lower rates were recorded among young married women than among young unmarried women or young men regardless of marital status. However, marital history had no apparent relevance to the frequency of HD among older population groups irrespective of their sex. The frequency and anatomical site of various lymphomas apparently differ geographically. Although Epstein-Barr virus and malarial parasites appear to play an etiologic role in BL, neither seems to be essential to the occurrence of the neoplasm. Demonstration of disease aggregates suggests that environmental factors, and possibly infectious factors, may contribute to the etiology of HD. Prior tonsillectomy may also be a predisposing factor. There is considerable evidence to indicate that immunoregulatory deficits contribute to the pathogenesis of autoimmunity, which in turn is coincidentally related to lymphoreticular malignancy. There is also limited evidence of a relationship between certain connective tissue disorders and various lymphoreticular malignancies. With the possible exception of multiple myeloma, the incidence of lymphoreticular malignancies is considerably lower in blacks than in whites. In view of the reported association between certain histocompatibility antigens and HD, consideration must be

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given to the fact that the frequency of some antigens differs markedly among whites and blacks. In one study, it was suggested that the incidence of HD in several ethnic groups correlates significantly with the gene frequency of HLA-A1 and 8 antigens. The HLA system or some closely related genetic system might modulate the effects of environmental factors, and a similar interaction might hold for other malignant lymphomas. (143 refs)

- 79-3042 **Epidemiology of the Leukaemias in Africa.** (Eng) Fleming, A. F. (Dept. Haematology, Ahmadu Bello Univ., Zaria, Nigeria). *Leuk Res* 3(2): 51-59; 1979.

The epidemiology of acute lymphatic, acute myeloid, chronic myeloid, and chronic lymphatic leukemia in Africans is reviewed. Conditions commonly seen in adult Africans that feature a great excess of mature lymphocytes include the tropical splenomegaly syndrome, gross lymphoid hyperplasia (possibly preceding the development of lymphoma), and chronic lymphatic leukemia. (58 refs)

- 79-3043 **Studies of Respiratory Cancer in High Risk Communities.** (Eng) Blot, W. J. (Environmental Epidemiology Branch, NCI, Landow Bldg., 3C07, Bethesda, MD 20014); Fraumeni, J. F. *J Occup Med* 21(4): 276-278; 1979.

The contribution of routinely collected cancer mortality data to the search for causes of respiratory cancer is reviewed. Between 1950 and 1969, the highest lung cancer mortality rates in the US were clustered in coastal areas of the south; rates were also high in metropolitan centers in the northeast and midwest. Since it is unlikely that smoking patterns caused all of the geographic variations in incidence, other factors, including occupational exposure, probably played some role. Mortality tended to be higher in counties with chemical, petroleum, paper manufacturing, and shipbuilding industries. Nasal tumors were significantly more frequent in areas with high proportions of furniture makers and petroleum refineries. These findings agree with similar findings in other countries. Case-control interview studies showed a relative risk associated with employment in shipyards of 1.6; the relative risk was increased to 2.4 among employees who were heavy smokers. Information supplied by next-of-kin was in good agreement with that supplied by the patient. Epidemiologic investigations of this type should help to identify the lung cancer risk factors prevailing in various parts of the US. (24 refs)

- 79-3044 **Non-toxic Goitre.** (Eng) Hennemann, G. (Erasmus Univ., Medical Faculty, Dykzigt

Hosp., Rotterdam, Netherlands). *Clin Endocrinol Metab* 8(1): 167-179; 1979.

The possible etiological factors, epidemiology, development, clinical features, thyroid function, and management of nontoxic goiter are reviewed. This condition may be defined as a sporadically occurring benign diffuse or multinodular enlargement of the thyroid of unknown etiology that is not associated with apparent abnormal hormone secretion. (62 refs)

- 79-3045 **Conclusions.** (Eng) Truswell, A. S. (No affiliation given); Asp, N. G.; James, W. P.; MacMahon, B. *Nutr Cancer* 1(2): 104-105; 1979.

Conclusions reached during a symposium on nutrition and cancer are summarized. Different dietary factors appear to be associated with cancer of the gastrointestinal tract, liver, and, possibly, the urinary bladder. There is also evidence for an indirect association between the level and type of nutrition and cancers of the breast and uterine endometrium. The level of nutrition and/or dietary fat probably predisposes to breast cancer by changing the hormone balance. Cancer of the large intestine is related to the Western diet and is correlated epidemiologically to total fat intake. Some epidemiological evidence suggests that beef consumption is correlated with large intestinal cancer; frying or broiling could produce a carcinogenic product, but the evidence is not firm. Evidence associating rectal cancer with beer consumption is inconclusive. The incidence of stomach cancer has decreased in all the industrial countries. Salt-cured and smoked foods could lead to this cancer as a result of nitrosamine formation in the stomach from nitrites. Vegetables and fruits appear to exert a protective effect, possibly because of the antioxidant effect of vitamin C. Moldy food contaminated with fungal toxins can be carcinogenic; this is unusual in countries with an efficient food industry. In general, avoidance of obesity, lower consumption of total fat, and a larger intake of fiber are recommended. (no refs)

- 79-3046 **The Cause of Species Differences in Mammary Tumorigenesis: Significance of Mammary Gland DNA Synthesis.** (Eng) Nagasawa, H. (Pharmacology Div., Natl. Cancer Center Res. Inst., Chuo-ku, Tokyo, Japan). *Med Hypotheses* 5(4): 499-509; 1979.

The causes of the species differences in mammary tumorigenesis are discussed. The incidence of mammary tumors varies from none in pigs and sheep to 100% in selected strains of mice, and there are also variations in the pattern of development and progression of the tumors. Development and progression are controlled by both genetic and environmental factors. Among the latter are hormones, mammary tumor virus (MTV) in some strains of mice, immune mechanisms, and diet. However, there is

no clear explanation for the observed species differences in mammary tumorigenesis. It appears that the longer the total period of low mammary gland DNA synthesis, the smaller the risk of mammary malignancy. It is hypothesized that species differences in mammary tumorigenesis may relate to intrinsic differences between species in the rate of normal mammary gland DNA synthesis under physiological conditions and/or in response to external stimuli. Differences in mammary gland DNA synthesis in response to pituitary grafting or prolactin in rats and mice might be partly due to MTV as well as to intrinsic species differences. (28 refs)

- 79-3047 Big ACTH and Bronchogenic Carcinoma.** (Eng) Yalow, R. S. (Solomon A. Berson Res. Lab., Veterans Admin. Hosp., Bronx, NY 10468). *Annu Rev Med* 30: 241-248; 1979.

The potential role of ACTH determinations in the diagnosis and management of patients with bronchogenic carcinoma is reviewed. 'Big' ACTH (larger and more acidic than the usual peptide hormone) is often a major component of plasma and tumor ACTH in patients with ectopic Cushing's disease. Its biological activity is <4% of its immunologic activity. ACTH is frequently detected in tissue samples of carcinoma primary to or metastatic from the lung but not in tumors metastatic to the lung. The concentration in adenocarcinoma is generally significantly lower than that in epidermoid carcinoma. ACTH is not detectable in normal human, dog, or rabbit lung, but it is found in some pathologic lung tissue, even in the absence of invasive carcinoma. If elevated values of plasma ACTH are derived from tumor ACTH, the tumor must be large, have a higher than minimally detectable hormone concentration, or be more active in secretion of the hormone than the pituitary. False-negative results can be obtained from lung tumors that do not meet these criteria. Also, in some patients, plasma ACTH apparently is not derived solely from tumor ACTH. False-positive results can result from the sporadic release of pituitary ACTH, even in normal subjects, and a significant fraction of false positives may be found among heavy smokers, particularly those with chronic obstructive pulmonary disease. The data suggest that determination of plasma ACTH in a mass screening program for early detection of lung carcinoma is of limited value, primarily because of this overlap between patients with invasive carcinoma and heavy smokers. (19 refs)

- 79-3048 Ectopic ACTH In Carcinoma of the Lung.** (Eng) Yalow, R. S. (Veterans Admin. Hosp., Bronx, NY 10468). *Prog Cancer Res Ther* 11: 209-216; 1979.

The use of radioimmunoassay to detect increases in plasma ACTH in patients with carcinoma of the lung is reviewed. ACTH is found in a high percentage of bronchial adenomas and carcinomas primary to or metastatic from

the lung, but not in other carcinomas metastatic to the lung. About one-third of pancreatic tumors also contain ACTH. The concentrations in lung adenocarcinomas are generally significantly less than those in squamous cell carcinomas. Although ACTH is not detectable in normal lung tissue, it is found in at least some pathologic lung tissue in the absence of invasive carcinoma; a significant number of these false-positive results might be found among heavy smokers. False negatives are most likely to be found among patients considered suitable for surgical resection rather than among those with more extensive or virulent disease. In one study, patients with extensive disease appeared to exhibit elevated ACTH levels more frequently than those with limited disease. In another study, 21/24 patients with untreated carcinoma of the lung had afternoon plasma ACTH concentrations >150 picogram/ml, the upper limit demonstrated in only 6% of the control group. Before determinations of ACTH can be used as part of a mass screening program for early detection of lung carcinoma, it will be necessary to determine that elevations are due to cancer and not simply to heavy smoking. The applicability of this procedure for differentiating between primary and metastatic disease and for monitoring the effects of surgical resection, chemotherapy, and radiotherapy has yet to be determined. (16 refs)

- 79-3049 Placental Proteins in Bronchogenic Carcinoma.** (Eng) Broder, L. E. (Comprehensive Cancer Center State of Florida, Univ. Miami, Miami, FL 33152); Rosen, S. W.; Weintraub, B. D.; Sussman, H. H.; Muggia, F. M.; Hansen, H. H.; Primack, A. *Prog Cancer Res Ther* 11: 217-225; 1979.

The use of placental proteins as cancer markers, especially in patients with bronchogenic carcinoma (BC) is reviewed. Human chorionic gonadotropin (hCG) has been found in the sera of 12% of a large series of cancer patients, 9% of 147 patients with BC, and in 4% of 201 patients with inoperable BC. hCG is composed of two dissimilar subunits, and inefficient combination and differential production of these subunits by clonal strains derived from the same tumor have been observed. Human placental lactogen (hPL) was found in approx 5% of 295 cancer patients and in 2% of the 201 inoperable BC patients. Placental alkaline phosphatase (PAP) was also found in 2% of the latter. The presence of each marker peptide (eg, hCG, hPL, and PAP) in the inoperable patients was independent of the other two, and the concentrations varied independently when more than one was found in a single patient. Serum marker concentrations have occasionally shown discordances during chemotherapy: marker concentrations fell despite increases in tumor size. The usefulness of these markers hinges on the fact that they are normally undetectable or present only in low concentrations in the plasma. Placental markers may prove useful in the location of primary neoplasms and in the identification of cerebral metastases. The biochemical basis for their expression by nonplacental neoplastic cells remains to be determined. (31 refs)

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- 79-3050 **Monitoring Environmental Exposures with Semen Assays.** (Eng) Gledhill, B. L. (Lawrence Livermore Lab.). *Energy Technol Rev* 7-16; 1979.

Analysis of animal and human semen based on conventional parameters (ie, sperm count, motility, and morphology) may be useful in assessing human spermatogenic injury induced by environmental agents. Data from studies in mice suggest that induced sperm abnormalities result from mutations of the genes that control spermatogenesis. The F_0 (examination of semen from exposed animals) and F_1 (examination of the offspring of exposed animals) tests in mice could be used to screen chemical and physical agents commonly or uniquely found in the workplace. Animal and human data suggest that lowered sperm count, reduced sperm motility, and increased sperm abnormalities are correlated with reduced fertility and the likelihood of spontaneous abortions and stillbirths. Flow cytometric techniques are being developed to determine induced changes in the shape and DNA content of human sperm as part of a screening procedure for monitoring human occupational exposure. The detection of clastogenic effects in human sperm will require improvements in resolution, but the technique looks promising. It may even be possible to automate the chore of visually scoring percent abnormal sperm through the microscope. (14 refs)

- 79-3051 **Dose Response to Cancerogenic and Mutagenic Treatments.** (Eng) Totter, J. R. (Inst. Energy Analysis, Oak Ridge Associated Univs., P.O. Box 117, Oak Ridge, TN 38730); Finamore, J. *Environ Int* 1(5): 233-244; 1978.

Thirty-seven dose-response curves obtained following exposure of animal and plant material to carcinogenic chemicals or low-LET (linear energy transfer) ionizing radiation were gathered from the literature. These curves, along with one obtained following exposure to a nutritional factor, were analyzed. The data show that there is no response that is unique to low-LET radiation nor is there one that is special for chemical carcinogens. Both responses can be depicted as a family of curves that is commonly used to describe biochemical reactions. Only two parameters need to be determined for each species to compare their respective responses to the agent (and, presumably, to extrapolate these data to very low doses). One of the parameters must be largely population dependent, ie, related to gene distribution, but the other is related to the nature of the agent and is probably little affected by the treatment. It is not clear how much alteration in response

may accompany marked differences in modes of administration. The dose-response data from the 38 experiments are displayed in a logarithmic plot on a single graph. Values of the two parameters and the doses and responses needed to construct the graph are tabulated. The mathematical treatment of the published data shows an unexpected universality of biological behavior that may be helpful in extrapolating experimental data to humans. (33 refs)

- 79-3052 **Ozonation of Mutagenic and Carcinogenic Alkylating Agents, Pesticides, Aflatoxin B_1 , and Benzidine in Water.** (Eng) Caulfield, M. J. (Lobund Lab., Dept. Microbiology, Univ. Notre Dame, Notre Dame, IN 46556); Burleson, G. R.; Pollard, M. *Cancer Res* 39(6, part 1): 2155-2159; 1979.

Kinetic studies of the effect of ozonation on the mutagenicity of selected chemicals in water were performed with the use of the *Salmonella*-microsome assay. The mutagenicity of certain pesticides, including bis(2-chloroethyl)amine and sodium azide, was rapidly inactivated by ozonation (0.5-1.5 min), but that of other alkylating agents, such as β -propiolactone, propanesultone, and N-methyl-N'-nitro-N-nitrosoguanidine was unaffected by ozone treatment. Ozone also rapidly inactivated the mutagenicity of aflatoxin B_1 (0.5 min). Three chemicals were converted to direct mutagens by ozone treatment. Under certain conditions, dimethylhydrazine could be converted to a mutagen that was stable for weeks. A similar chemical, 2-hydroxyethylhydrazine, was converted to an unstable mutagen that was inactive after 24 hr at room temperature. When benzidine was treated with ozone, there was a transient increase in mutagenicity that was lost after longer treatment with ozone. (26 refs)

- 79-3053 **5-Methylcytosine Content of Nuclear DNA During Chemical Hepatocarcinogenesis and in Carcinomas Which Result.** (Eng) Lapeyre, J. N. (Section Experimental Pathology, M. D. Anderson Hosp. and Tumor Inst., Univ. Texas System Cancer Center, Houston, TX 77030); Becker, F. F. *Biochem Biophys Res Commun* 87(3): 698-705; 1979.

To examine the relationship between DNA methylation and malignancy, measurements were made of 5-methylcytosine levels in the nuclear DNA of normal Sprague-Dawley rat liver and of premalignant and malignant hepatic tissues induced by diethylnitrosamine (DEN)

and acetylaminofluorene (AAF). DNA from premalignant nodules and primary hepatocellular carcinomas (HCC's) induced by AAF and from primary HCC's induced by DEN was undermethylated by 20%, 45%, and 32.5%, respectively. A 12.5% hypomethylation occurred during the DNA synthesis phase of hepatic regeneration. To better determine the effect of cell proliferation on methylation, DNA synthesis in 21-hr regenerating rat liver was determined and compared with that in two transplantable HCC lines. DNA synthesis, hence, the cell proliferation rate, was approx 33% and 39% greater in the 21-hr regenerating liver than in the two respective HCC lines. Despite this, the transplantable HCC's demonstrated significantly less 5-methylcytosine than the regenerating liver. There was a relative 49% and 59% hypomethylation in the transplantable HCC DNA. It is suggested that an aberration in endogenous DNA methylation may occur during neoplastic transformation. (45 refs)

79-3054 Initiation of Putative Preneoplastic Liver Lesions by Single Doses of Nonliver and Liver Carcinogens Plus Partial Hepatectomy (PH) (Meeting Abstract). (Eng) Tsuda, H. (Dept. Pathology, Univ. Toronto, Toronto M5S1A8, Canada); Farber, E. *Proc Am Assoc Cancer Res* 20: 15; 1979 (2 refs)

79-3055 Increased Epoxide Hydrase Activity Following Aflatoxin B₁, Diethylnitrosamine (DEN), 2-Acetylaminofluorene (2-AAF), Safrole and Azo Dye Administration, and in Hyperplastic Nodules and Hepatomas Induced by DEN and 2-AAF (Meeting Abstract). (Eng) Cameron, R. (Univ. Toronto, Toronto M5S 1A8, Canada); Lee, G.; Parker, N. B. *Proc Am Assoc Cancer Res* 20: 50; 1979 (2 refs)

79-3056 Inhibition of Carcinogens by Wheat Sprouts (Meeting Abstract). (Eng) Lai, C. N. (Dept. Biology, Univ. Texas System Cancer Center, 6723 Bertner Ave., Houston, TX 77030); Dabney, B. J.; Shaw, C. R. *Proc Am Assoc Cancer Res* 20: 11; 1979 (no refs)

79-3057 A New Screening Procedure for Carcinogenic Agents (Meeting Abstract). (Eng) Swierenga, S. H. (Natl. Health and Welfare Canada, Ottawa, Canada); Boynton, A. L.; Whitfield, J. F. *In Vitro* 15(3): 223; 1979 (no refs)

79-3058 Transformation of BALB/3T3 Cells by Residue Organics from Drinking Water

(Meeting Abstract). (Eng) Kurzepa, H. (Univ. Cincinnati Coll. Medicine, Cincinnati, OH 45267); Cole, M. S.; Lang, D. R. *In Vitro* 15(3): 224; 1979 (no refs)

79-3059 Interactions of Multiple Carcinogens in the Salmonella Mutagenesis Assay (AMES) (Meeting Abstract). (Eng) Saffiotti, U. (Lab. Experimental Pathology, DCCP, NCI, NIH, Bethesda, MD 20014); Donovan, P. J.; Rice, J. M. *Proc Am Assoc Cancer Res* 20: 191; 1979 (no refs)

79-3060 Sister Chromatid Exchange (SCE) in Human Diploid Fibroblasts Induced by Mutagens With and Without Rat Liver Microsomal Activation (Meeting Abstract). (Eng) Yang, D. P. (Wyeth Labs. Inc., Philadelphia, PA 19101); Graupensperger, F.; Minecci, L. C.; Rubin, B. A. *In Vitro* 15(3): 173; 1979 (no refs)

79-3061 Measurement of Chemical-induced DNA Repair Synthesis in Human Peripheral Blood Monocytes (Meeting Abstract). (Eng) Lake, R. S. (Children's Hosp. Medical Center of Akron, Akron, OH 44308); Shoemaker, R. H.; Igel, H. J. *Proc Am Assoc Cancer Res* 20: 58; 1979 (no refs)

79-3062 Neoplastic Transformation and Somatic Mutation Induced by ³H-Thymidine Incorporation (Meeting Abstract). (Eng) Lin, S. L. (Div. Biophysics, Johns Hopkins Univ., Baltimore, MD 21205); Klein, L.; Takii, M. *Proc Am Assoc Cancer Res* 20: 118; 1979 (no refs)

79-3063 Chromosome Lateral Asymmetry: A Sensitive Assay for Screening Teratogenic Agents. (Eng) Tucci, S. M. (Dept. Anatomy, Albany Medical Coll., Albany, NY); Skalko, R. G. *J Environ Pathol Toxicol* 2(3): 625-632; 1979.

The effects of in utero administration of three teratogens on the frequency of lateral asymmetry in mouse embryo chromosomes were determined. Pregnant albino ICR mice were administered single ip embryotoxic doses of bromodeoxyuridine (2,000 mg/kg), hydroxyurea (250 mg/kg), or mitomycin C (0.5-10.0 mg/kg) on day 10 of gestation. Embryos were removed 4 hr later, and a cell suspension was prepared and cultured in the presence of colcemid. Metaphase chromosome spreads were subjected to standard G-banding procedures, and the occurrence and frequency of lateral asymmetry (unequal banding of sister

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chromatids) was monitored. Embryotoxic levels of all three teratogens increased the number of asymmetries/karyotype. For mitomycin C, the increase was dose-dependent. (24 refs)

79-3064 Mutagens and Inhibitors of Mutagenesis in Pan-Fried Hamburger (Meeting Abstract). (Eng) Pariza, M. W. (Dept. Food Microbiology and Toxicology, Univ. Wisconsin, Madison, WI 53706); Ashoor, S. H.; Chu, F. S.; Lund, D. B. *Proc Am Assoc Cancer Res* 20: 39; 1979 (1 ref)

79-3065 Model System for Studying Formation of Mutagens During the Cooking of Meat (Meeting Abstract). (Eng) Spingarn, N. E. (Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595); Garvie, C. T.; Vuolo, L. L. *Proc Am Assoc Cancer Res* 20: 179; 1979 (no refs)

79-3066 Inhibition of Carcinogenesis by Feeding Diets Containing Soybeans (Meeting Abstract). (Eng) Troll, W. (Dept. Environmental Medicine, New York Univ. Medical Center, 550 First Ave., New York, NY 10016); Wiesner, R.; Belman, S.; Shellabarger, C. J. *Proc Am Assoc Cancer Res* 20: 265; 1979 (no refs)

79-3067 The Mitochondrial Activation of Sulfate and Arsenate and Their Role in Carcinogenesis. (Eng) Hadler, H. I. (Dept. Chemistry and Biochemistry, Southern Illinois Univ., Carbondale, IL 62901); Cook, G. L. *J Environ Pathol Toxicol* 2(3): 601-612; 1979.

A study demonstrating that sulfate and arsenate can substitute for phosphate in the transitory uncoupling of rat liver mitochondria induced by hydrazine when β -hydroxybutyrate is the substrate is presented. Although hydrazine (400 μ M), sulfate (1.5 mM), or arsenate (200 μ M) alone did not induce transitory uncoupling, hydrazine combined with either sulfate or arsenate did cause uncoupling; this effect was blocked by rutamycin. The inception of stimulated respiration by hydrazine and sulfate was slowed by 150 μ M AMP and completely blocked by ADP, ATP, pyrophosphate, and Mg^{2+} (all at 150 μ M). Benzene sulfonate (1.5-6.0 mM) did not uncouple mitochondria either alone or in combination with hydrazine (300-600 μ M), and EDTA (5-10 mM) did not produce an EDTA-dependent hydrazine uncoupling when tested with 300-600 μ M hydrazine. Since sulfate enhances the carcinogenicity of certain carcinogens, these results expand the experimental confluence between oxidative phosphorylation and chemical carcinogenesis and also explain at least in part the

"toxic" effects of sulfate. Arsenate may also enhance carcinogenicity. The data are compatible with epidemiological studies implicating some role in carcinogenesis for sulfate and arsenate. (35 refs)

79-3068 Detection of the Mutagenic Activity of Lead Chromate Using a Battery of Microbial Tests. (Eng) Nestmann, E. R. (Mutagenesis Section, Environmental and Occupational Toxicology Div., Dept. Natl. Health and Welfare, Ottawa, Ontario K1A 0L2, Canada); Matula, T. I.; Douglas, G. R.; Bora, K. C.; Kowbel, D. J. *Mutat Res* 66(4): 357-365; 1979.

The potential mutagenicity of the carcinogen lead chromate was tested in the *Escherichia coli* PolA⁺/PolA⁻ survival test; the *Salmonella*/microsome His⁺ reversion assay; the *E. coli* Trp⁺ reversion test as a plate assay; the *E. coli* Gal⁺ forward mutation test; and the *Saccharomyces cerevisiae* assay for mitotic recombination. Lead chromate was mutagenic in *Salmonella* and in *Saccharomyces* and was thus identified as a microbial mutagen by this battery. Metabolic activation by a rat liver homogenate mixture (S9) was not required for the manifestation of mutagenic activity. The most statistically significant, positive result was found with a supplementary assay, the *E. coli* fluctuation test. To determine whether the lead ion and/or the chromate ion was responsible for the mutagenicity, lead chloride and chromium trioxide (chromic acid) were also tested. In *E. coli* fluctuation tests, the ranges of max mutagenicity for chromium trioxide and lead chromate overlapped at the concentration 10^{-5} M, whereas lead chloride showed no mutagenicity and little lethality at concentrations up to 10^{-3} M. Thus, the chromate ion apparently is responsible for the mutagenicity of lead chromate. (16 refs)

79-3069 Morphometric Light and Electron Microscopic Studies of Histopathological Changes in Nasal Mucosa of Nickel Workers (Meeting Abstract). (Eng) Reith, A. (Norsk Hydro's Inst. Cancer Res., Montebello, Oslo 3, Norway); Boysen, M.; Solberg, L. A. *Proc Am Assoc Cancer Res* 20: 290; 1979 (no refs)

79-3070 Determination of Nickel in Urine and Plasma by Atomic Absorption Spectrophotometry. (Cze) Tichy, M. (Institut hygieny a epidemiologie, Centrum hygieny prace a nemoci z povolani, Srobarova 48, 100 42 Prague 10, Czechoslovakia); Horejsi, M. *Prac Lek* 30(9): 337-340; 1978.

Nickel levels in the urine and serum of healthy subjects not exposed to nickel were determined by atomic absorption

spectrophotometry. The urinary clearance averaged 22 $\mu\text{g}/1/24$ hr. The mean plasma level was 2.7 $\mu\text{g}/100$ ml. (21 refs)

79-3071 Effect of Induced Skin Cancer on the Concentrations of Some Trace Elements in the Mouse.

(Eng) Mangal, P. C. (Dept. Biophysics, Panjab Univ., Chandigarh-160014, India); Verma, K. B. *Indian J Med Res* 69: 290-295; 1979.

The concentrations of the trace elements Se, Cr, Fe, Co, Zn, Sc, Rb, Cd, Ag, and Sb in normal and malignant mouse skin were studied using a neutron activation technique without chemical separation. Skin cancer was induced in female Swiss mice by sc injection of 20-methylcholanthrene (0.4 mg). The concentrations of Cr, Ag, and to a lesser extent, Sb were higher and the concentrations of Se and, to a lesser extent, Fe, Zn, and Rb were lower in the tumor tissue than in normal skin. Co, Cd, and Sc levels were similar in both tissues. It is not clear whether the changes in the concentrations of certain trace elements were a cause or effect of neoplastic growth. (11 refs)

79-3072 Selenium and Cadmium Levels in the Kidneys of Patients with Cancer and Other Diseases

(Meeting Abstract). (Eng) Shamberger, R. J. (Cleveland Clinic Foundation, Cleveland, OH 44106). *Proc Am Assoc Cancer Res* 20: 18; 1979 (no refs)

79-3073 Reduction in Mutagenicity of Cigarette Smoke Condensate by Added Sugars. (Eng) Sato, S.

(Biochemistry Div., Natl. Cancer Center Res. Inst., 5-1-1 Tsukiji, Chuo-ku, Tokyo 104, Japan); Ohka, T.; Nagao, M.; Tsuji, K.; Kosuge, T. *Mutat Res* 60(2): 155-161; 1979.

The effects of adding glucose, fructose, galactose, sorbitol, sucrose, and lactose to high- and low-tar cigarettes on the mutagenicity of their smoke condensates (SC's) were studied using *Salmonella typhimurium* strains TA100 and TA98 with and without metabolic activation. Using TA98 with metabolic activation, the lowest mutagenicities observed after addition of the sugars per milligram of SC were 37% (high-tar cigarettes) and 22% (low-tar cigarettes) of that of the SC from untreated cigarettes. Although the sugars increased the total amounts of SC, they decreased the mutagenicities of the total condensates, the lowest values being 35% (high-tar cigarettes) and 36% (low-tar cigarettes) of that of the SC from untreated cigarettes. Using TA100 with metabolic activation, decreases in both specific and total mutagenicities of the SC's of high-tar cigarettes were observed with all the sugars tested except galactose and sucrose. Glucose, fructose, or sorbitol decreased the specific mutagenicity of low-tar cigarette

condensates, and glucose and fructose also reduced their total mutagenicity. The effects of the sugars were more marked with TA98 than with TA100, and of the sugars tested, fructose and sorbitol had the greatest effects. Without metabolic activation, the sugars had no effect on the mutagenicity of the SC's. (21 refs)

79-3074 Effect of Tobacco Smoke on the Metabolism of Rat Lung. (Eng) Hamosh, M. (Dept.

Physiology and Biophysics, Georgetown Univ. Sch. Medicine and Dentistry, Washington, DC 20007); Shechter, Y.; Hamosh, P. *Arch Environ Health* 34(1): 17-23; 1979.

The effect of cigarette smoke on the uptake and metabolism of ^3H -palmitate, ^3H -leucine, U^{14}C -glucose, and ^{14}C -glucosamine was studied using slices of rat lung. Male Sprague-Dawley rats were exposed daily for 30 days to University of Kentucky research cigarettes [brands 1A1 (25 mg tar, 0.3 mg nicotine) or 1R1 (30 mg tar, 2.2 mg nicotine)], in the Walton smoking machine on two schedules: one cigarette 2x/day or three cigarettes 2x/day (7 puffs cigarette). One group of rats served as intact controls, and another was introduced to the machine but was not exposed to smoke. All animals were wt-matched at the beginning of the experiment (180 ± 5 g); machine-control rats gained 25% less wt, 1A1-exposed rats 40% less, and 1R1-exposed rats 26%-40% less than controls. Exposure to tobacco smoke had no effect on palmitate uptake and synthesis of phospholipids. Exposure to 1A1 cigarettes caused a 25% increase in $^{14}\text{CO}_2$ production and a 30% higher incorporation of glucose into lipids. Exposure to 1R1 cigarettes had no effect on glucose metabolism. Protein synthesis was 30% lower in 1R1-exposed rats, and glycoprotein synthesis increased twofold in both 1A1- and 1R1 exposed rats. At present, there is no satisfactory explanation for these observations (43 refs)

79-3075 Immuno- and Lympho-proliferative Neoplasms Associated with Exposure to Asbestos (ETA) (Meeting Abstract). (Eng) Haidak, D. J.

(Georgetown Univ. Medical Center, Washington, DC 20007); Kagan, E.; Jacobson, R. J.; Yeung, K. Y.; Nachnani, G. H. *Proc Am Assoc Cancer Res* 20: 417; 1979 (1 ref)

79-3076 Cellular Immunity in Asbestosis. (Eng) Lange, A. (Dept. Occupational Diseases, Inst. Internal Diseases, Medical Sch., 50-367 Wroclaw, Poland); Smolik, R.; Chmielarczyk, W.; Garncarek, D.; Gielgier, Z.

Arch Immunol Ther Exp (Warsz) 26(1-6): 899-903; 1978.

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The involvement of lymphocyte malfunction in the pathogenesis of asbestosis was investigated in 271 asbestos workers. Asbestosis and positive antinuclear antibody (ANA) titers were encountered significantly more frequently among workers who responded in delayed-hypersensitivity skin tests to tuberculin and/or streptokinase-streptodornase (SK-SD) than among those who responded to neither antigen. The proportion of lymphocytes lacking RBC and complement receptors was higher in workers with asbestosis than in those without the disease and higher in ANA-positive subjects. Assay for migration inhibitory factor showed impairment in the generation of this factor following stimulation of lymphocytes with SK-SD, phytohemagglutinin (PHA), or purified protein derivatives of tuberculin in subjects with asbestosis. Study of lymphocyte transformation showed an impaired response to lower doses of PHA and concanavalin A in asbestosis-positive, ANA-positive, and asbestosis-negative, ANA-positive workers. The data suggest that impairment of cell-mediated immunity is closely linked to the pathogenesis of asbestosis, which is associated with a high risk of cancer. (12 refs)

79-3077 Establishment and Characterization of a Human Mesothelioma Cell Line (Meeting Abstract). (Eng) Ohnuma, T. (Mount Sinai Sch. Medicine, New York, NY 10029); Wolman, S. R.; Daum, S. M.; Holland, J. F. *Proc Am Assoc Cancer Res* 20: 162; 1979 (no refs)

79-3078 Discontinuities in Dose Response Curves from Toxicological Tests. (Eng) Anderson, R. L. (Procter & Gamble Co., Miami Valley Labs., Cincinnati, OH). *Household & Personal Products Industry* 16(4): 92,94; 1979.

Nonlinear dose-related shifts in urinary pH and increases in urinary calcium, magnesium, and phosphorus excretion occurred in rats fed sodium saccharin in doses of up to 7.5%. These and further observations suggested that saccharin influences the normal metabolic pathways for nitrogen excretion and disturbs the acid/base balance. It is possible that shifts in the cellular environment produce changes in urinary tract cells via physical forces and not via chemically induced changes in cellular constituents. (1 ref)

79-3079 Studies on the Mutagenic Properties of Bleaching Effluents. (Eng) Eriksson, K. E. (STFI, Box 5604, Swedish Forest Products Res. Lab., S-114 86 Stockholm, Sweden); Kolar, M. C.; Kringstad, K. *Svensk Papperstidning* 82(4): 95-104; 1979.

The existence of mutagenic substances in effluents from the bleaching of softwood and hardwood kraft and sulfite pulps was determined using the histidine-requiring mutant *Salmonella typhimurium* strain TA1535. Effluents from the first-stage bleaching of softwood and hardwood kraft and softwood sulfite pulp with chlorine or mixtures of chlorine and chlorine dioxide-containing compounds contained mutagens. All other bleaching effluents were non-mutagenic. The compounds responsible for the mutagenic effects were relatively unstable when stored under neutral conditions, and they were highly unstable when stored under alkaline conditions. The results do not allow an assessment of the risks involved in releasing the mutagenic effluents into rivers and lakes. (7 refs)

79-3080 Safety Evaluation of Toothpaste Containing Chloroform. III. Long-Term Study in Beagle Dogs. (Eng) Heywood, R. (Huntingdon Res. Centre, Huntingdon, England); Sortwell, R. J.; Noel, P. R.; Street, A. E.; Prentice, D. E.; Roe, F. J.; Wadsworth, P. F.; Worden, A. N.; Van Abbe, N. J. *J Environ Pathol Toxicol* 2(3): 835-851; 1979.

Groups of eight male and female beagle dogs were given 15 or 30 mg/kg/day of chloroform (CF) in a toothpaste base po (in gelatin capsules) on 6 days/wk for 7.5 yr, followed by a 20- to 24-wk recovery period. Groups of 16 males and females received vehicle only, and 7 dogs of each sex remained untreated. Eleven of the 96 dogs died during the study, only 2 of these being in the CF-treated groups. The only significant toxic response during treatment was a moderate rise in SGPT levels; they reached a peak in the sixth year of the study and probably corresponded to minimal liver damage. Few palpable growths were noted while the dogs were alive. Fatty cysts were seen in the liver of several dogs at postmortem; they could possibly be associated with CF treatment, but the distribution of nodular changes in the liver was not obviously dose-related. A small number of macroscopic and microscopic neoplasms were seen; one dog in each CF-treated group had a malignant tumor, but there were no tumors in the liver or kidney of any dog. Overall, exposure to CF in a toothpaste base was not associated with any effect on the incidence of any kind of neoplasm. From this and related studies in mice and rats, it is concluded that repeated exposure to 3.5% CF in toothpaste is unlikely to result in any hazard to human health. (21 refs)

79-3081 Safety Evaluation of Toothpaste Containing Chloroform. II. Long Term Studies in Rats. (Eng) Palmer, A. K. (Huntingdon Res. Centre, Huntingdon, England); Street, A. E.; Roe, F. J.; Worden, A. N.; Van Abbe, N. J. *J Environ Pathol Toxicol* 2(3): 821-833; 1979.

The results of a preliminary 13-wk po toxicity study of chloroform (CF) in a toothpaste base, conducted with the use of Sprague-Dawley rats, are reported, along with the results of two longer-term studies. Significant changes in serum enzyme and certain hematological parameters were seen at the higher dose levels (150 and 410 mg/kg/day) in the range-finding study. Intercurrent disease made it necessary to terminate the first long-term experiment prematurely after 1 yr. No evidence of serious toxicity was recorded. In the second long-term experiment, groups of 50 cesarian-derived specific pathogen-free Sprague-Dawley rats received 60 mg/kg/day CF in a toothpaste base or the vehicle only by gavage on 6 day/wk for 80 wk; they were observed for an additional 15 wk. CF-treated rats of both sexes survived longer than the controls, although both groups had a high incidence of nonneoplastic respiratory and renal disease. Female rats consistently had decreased plasma cholinesterase levels, shown to be related to activity against butyrylcholine but not acetyl- β -methylcholine. Tumors of various sites were seen in 39% of CF-treated rats of both sexes examined histologically, compared with 38% of vehicle controls. There were no treatment-related effects on the incidence of liver or kidney tumors. Malignant mammary tumors occurred in more treated than control rats, but the difference was not statistically significant. Thus, long-term CF treatment had no adverse effect on survival and did not significantly influence time of onset, malignancy, or location of tumors. (6 refs)

- 79-3082 Safety Evaluation of Toothpaste Containing Chloroform. I. Long-Term Studies in Mice.** (Eng) Roe, F. J. (Huntingdon Res. Centre, Huntingdon, England); Palmer, A. K.; Worden, A. N.; Van Abbe, N. J. *J Environ Pathol Toxicol* 2(3): 799-819; 1979.

Studies were performed to determine whether exposure of mice to chloroform (CF) at dose levels 113 and 400 times the level ingested by humans using toothpaste containing 3.5% CF, but less than those producing severe liver damage, predisposed to cancer of the liver or any other site. CF was administered to several strains of mice by gavage in a toothpaste base or in arachis oil, in doses of 17 or 60 mg/kg/day on 6 days/wk for 80 wk. Control groups were left untreated or given vehicle only. In general, there were more survivors in CF-treated groups than in the controls. In C57BL, CBA, and CF/1 males, treatment was associated with no adverse effect on the incidence of any type of neoplasm or any other parameter. In males but not females of strain ICI, and in doses of 60 mg/kg/day but not 17 mg/kg/day, exposure to CF in toothpaste was associated with an increased incidence of epithelial tumors of the kidney. This effect was more pronounced in mice given 60 mg/kg/day CF in arachis oil. This treatment was also associated with a higher incidence and severity of non-neoplastic renal disease. The mechanisms underlying the peculiar strain- and sex-specific susceptibility of ICI male mice remain obscure; spontaneous renal tumors were also

seen in vehicle control mice, and possible ways in which this tendency may be enhanced by CF treatment are discussed. At the dose levels tested, no adverse effect was seen in the liver and there was no increased incidence of liver tumors, even in the higher liver tumor-susceptible CBA strain. At the 17-mg/kg/day level no excess of renal tumors was seen, even in males of the susceptible ICI strain. (21 refs)

- 79-3083 Carcinogenicity of Hexachlorocyclohexane (BHC) in Pure Inbred Swiss Mice.** (Eng) Kashyap, S. K. (Natl. Inst. Occupational Health, Meghani Nagar, Ahmedabad-380016, India); Nigam, S. K.; Gupta, R. C.; Karnik, A. B.; Chatterjee, S. K. *J Environ Sci Health [B]* B14(3): 305-318; 1979.

The carcinogenicity of technical grade benzene hexachloride (BHC: 100 ppm in the diet for 80 wk, 10 mg/kg by po intubation for 80 wk, or 0.25 mg painted on the skin twice weekly for 80 wk) was investigated in inbred Swiss mice. The BHC-treated and control mice did not differ with respect to survival rate, body wt, or growth rate, although the control animals consumed more food throughout the experimental period. Signs of BHC intoxication included convulsions, circling behavior, drooping ears, and corneal opacity. The incidence of tumors was significantly higher among the mice given BHC po than among the controls and nonsignificantly higher among the mice painted with BHC. The incidence of liver tumors was higher among males than females, and females were more susceptible to lymphoreticular tumors. Tumors seldom occurred at more than one site. Most of the liver tumors in the BHC-treated groups were hepatocellular carcinomas, whereas the controls developed only hepatomas and hemangiomas. The incidence of lung tumors, primarily well-circumscribed adenomas, was low, and few tumors developed in other sites. The results point out the high incidence of liver and lymphoreticular tumors induced by BHC given po (with the diet or through intubation). (7 refs)

- 79-3084 Residues of Chlorinated Pesticides in Mother's Milk and in the Serum of Their Babies.** (Cro) Bazulic, D. (Odsjek za pesticide, Zavod za zastitu zdravlja SRH, Zagreb, Yugoslavia); Kipic, D.; Stampar-Plasaj, B.; Jeric, J.; Bujanovic, V.; Juzbasic, N. *Arh Hig Rada Toksikol* 29(2): 125-128; 1978.

Serum and milk samples from 27 nursing mothers and serum samples from their babies were analyzed for organochlorine pesticide residues. α -Benzene hexachloride (α -BHC: 77.8 ppb), β -BHC (150 ppb), 1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene (p,p'-DDE: 1,537 ppb), 1,1,1-trichloro-2-(o-chlorophenyl)2-p-chlorophenylethane (o,p'-DDT: 42.8 ppb), 1,1-dichloro-2,2-bis(p-

chlorophenyl) ethane (p,p'-DDD: 59.6 ppb), and 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (p,p'-DDT: 256 ppb) were found in the milk samples. The corresponding concentration in maternal sera were 6.8 ppb and α -BHC, 13.1 ppb for β -BHC, 13.8 ppb for p,p'-DDE, 10.7 ppb for o,p'-DDT, 5.5 ppb for p,p'-DDD, and 34.4 ppb for p,p'-DDT. The ratio of the serum concentrations in the mother to that in the baby was 0.95 for α -BHC, 0.64 for β -BHC, 1.18 for p,p'-DDE, 0.31 for o,p'-DDT, 0.72 for p,p'-DDD, and 1.70 for p,p'-DDT. (5 refs)

79-3085 Carcinomas and Other Lesions of the Liver in Mice Ingesting Organochlorine Pesticides. (Eng) Reuber, M. D. (NCI Frederick Cancer Res. Center, Frederick, MD 21701). *Clin Toxicol* 13(2): 231-256; 1978.

The effects of po organochlorine pesticide treatment on the livers of mice were studied. Carcinomas of the liver, often multiple, were induced at high incidence rates in mice given dieldrin, aldrin, heptachlor, heptachlor epoxide, kepone, lindane, chloroform, chlordane, or carbon tetrachloride. Increased incidences of liver carcinomas were also seen in mice given chlorobenzilate, mirex, DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane], methoxychlor, or endrin. There were occasional metastases to the lungs; however, a careful search for metastases was generally not carried out. Well-, moderately well-, and poorly differentiated hepatocellular and cholangiocellular carcinomas were observed, the metastatic tumors being predominantly hepatocellular carcinomas. Hemangioendothelial sarcomas, leiomyosarcomas, and reticulum cell sarcomas were rarely observed. Focal cirrhosis was occasionally seen in mice given heptachlor or heptachlor epoxide, and it was severe in animals given large doses of carbon tetrachloride or chloroform. Hepatic vein thrombi were occasionally present in mice given heptachlor, heptachlor epoxide, dieldrin, aldrin, or chlorobenzilate, and focal hepatic necrosis was sometimes seen. (78 refs)

79-3086 Vinyl Chloride Associated Hepatic Angiosarcoma Chemotherapy. (Eng) Dannaher, C. (Div. Hematology-Oncology, Univ. Louisville, Louisville, KY 40202); Yam, L. T.; Tamburro, C. *Proc Am Assoc Cancer Res* 20: 358; 1979. (0 refs)

79-3087 Carcinogenicity and Chemical Reaction with Guanine of 1-Chloropropene and Two of Its Potential Metabolites. (Eng) Goldschmidt, B. M. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York,

NY 10016); Van Duuren, B. L.; Goldstein, R. C.; Smith, A. C. *Proc Am Assoc Cancer Res* 20: 91; 1979. (0 refs)

79-3088 The Mutagenicity of Halogenated Alkanols and Their Phosphoric Acid Esters for *Salmonella typhimurium*. (Eng) Nakamura, A. (Natl. Inst. Hygienic Sciences, Kamiyoga 1-18-1 Setagaya, Tokyo); Tateno, N.; Kojima, S.; Kaniwa, M.; Kawamura, T. *Mutat Res* 66(4): 373-380; 1979.

Nine halogenated alkanols, nine corresponding tris(haloalkyl)phosphates, and two bis(2,3-dibromopropyl)phosphate salts were evaluated for mutagenicity toward *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538, with and without a rat liver microsome metabolic activation system (S9 mix). Most of the test samples were mutagenic toward TA100 and TA1535, but not TA98, TA1537, and TA1538. In general, the mutagenic activities of the phosphates obtained in the presence of the S9 mix were greater than those obtained in the absence of the mix. Among the phosphates, several structure-activity relationships were found: (1) the bromoalkyl derivatives were more mutagenic than the corresponding chloroalkyl derivatives, (2) the β -haloethyl derivatives were more mutagenic than the γ -halopropyl derivatives, (3) the phosphates having adjacent β - and γ -halogen atoms in the alkyl moiety, [eg, tris(2,3-dibromopropyl)phosphate (tris-DBPP)] were particularly potent mutagens, and (4) the branched carbon chain reduced mutagenicity in spite of the presence of β -halogen atoms [eg, tris(1-bromomethyl-2-bromoethyl) phosphate]. However, these relations did not necessarily apply to the halogenated alkanols. It is concluded that the metabolic activation pathway via haloalkanol to mutagens must not be common to all of the tris-DBPP-like phosphates. (12 refs)

79-3089 Characterization of the Mutagenic Ethidium Azide Interactions with Deoxyribonucleic Acids. (Eng) Graves, D. E. (Lab. Molecular Biology, Univ. Alabama, Birmingham, AL 35294); Watkins, C. L.; Yielding, L. W.; Yielding, K. L. *Fed Proc* 38(3, part 1): 425; 1979. (0 refs)

79-3090 Binding of the Carcinogen Ethylene Dibromide to Chromosomal Constituents of Forestomach and Liver. (Eng) Banerjee, S. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016); Van Duuren, B. L. *Proc Am Assoc Cancer Res* 20: 85; 1979. (0 refs)

79-3091 Interaction of Potential Activated Intermediates of the Carcinogen Ethylene Dibromide with Protein and DNA In Vitro. (Eng) Kline, S. A. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016); Banerjee, S.; Van Duuren, B. L. *Proc Am Assoc Cancer Res* 20: 86; 1979 (0 refs)

79-3092 The Role of Peritoneal Cells (PC) and Bacterial Lipopolysaccharide (LPS) in the Growth of Murine Plasmacytoma (PCT). Platica, M. (Res. Inst., Hosp. Joint Diseases, Mt. Sinai Sch. Medicine, New York, NY); Bojke, C.; Hollander, V. P. *Proc Am Assoc Cancer Res* 20: 21; 1979. (0 refs)

79-3093 Induction of Increased Frequencies of Sister Chromatid Exchange in V79 Cells by Butyric Acid. (Eng) Tai, C. C. (Biotech Res. Lab. Inc., Rockville, MD 20852); Ting, R. C. *In Vitro* 15(3): 172; 1979. (0 refs)

79-3094 S-Vinyl Homocysteine (Vinthionine): A Highly Mutagenic Analog of Ethionine (Meeting Abstract). (Eng) Leopold, W. R. (McArdle Lab. Cancer Res., Univ. Wisconsin Center Health Sciences, Madison, WI 53706); Miller, J. A.; Miller, E. C. *Proc Am Assoc Cancer Res* 20: 28; 1979 (1 ref)

79-3095 Effect of Chronic Ethanol Ingestion on Procarcinogen Activation by Intestinal, Lung and Hepatic Microsomes (Meeting Abstract). (Eng) Garro, A. J. (Mount Sinai Sch. Medicine, New York, NY 10029); Seitz, H. K.; Lieber, C. S. *Proc Am Assoc Cancer Res* 20: 73; 1979 (no refs)

79-3096 Influence of Ethanol on the Metabolism and Metabolic Capacity of Hamster Cheek Pouch Squamous Epithelium (Meeting Abstract). (Eng) McCoy, G. D. (Div. Molecular Biology and Pharmacology, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595); Tambone, P. C.; Wynder, E. L. *Proc Am Assoc Cancer Res* 20: 174; 1979 (no refs)

79-3097 Does Ethanol Induce Chromosome Damage? (Meeting Abstract). (Eng) Au, W. (Section

Cell Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Badr, F. M. *In Vitro* 15(3): 221; 1979 (no refs)

79-3098 Increased Mutation Frequency Due to Ethylene Oxide Adsorbed to Plastics (Meeting Abstract). (Eng) Brown, A. M. (Federal Drug Admin., Silver Spring, MD 20910); Bruch, C.; Jacobson, E.; Krell, K.; Page, B.; Thomas, M. *In Vitro* 15(3): 220-221; 1979 (no refs)

79-3099 Divergent Effects of Enteric Bypass, Enterectomy, and Colectomy on Azoxymethane-induced Carcinogenesis (Meeting Abstract). (Eng) Williamson, R. C. (Surgical Services, Massachusetts General Hosp., Boston, MA 02114); Bauer, F. L.; Ross, J. S.; Malt, R. A. *Proc Am Assoc Cancer Res* 20: 18; 1979 (no refs)

79-3100 In Vivo Metabolism of ¹⁴C-Labeled Methylazoxymethanol Acetate and its Partial Inhibition by Pyrazole (PZ) (Meeting Abstract). (Eng) Fiala, E. S. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595); Kulakis, C.; Weisburger, J. H. *Proc Am Assoc Cancer Res* 20: 20; 1979 (no refs)

79-3101 DNA Fragmentation in Some Organs of Rats and Mice Treated with Cycasin. (Eng) Cavanaugh, M. (Dept. Pharmacology, Univ. Genoa, I-16132 Genoa, Italy); Parodi, S.; Taningher, M.; Bolognesi, C.; Sciaba, L.; Brambilla, G. *Br J Cancer* 39(4): 383-390; 1979.

The effect of cycasin (50-400 mg/kg, po or ip) on the elution rate of DNA (an increase being indicative of DNA damage) from liver, lung, kidney, and colon mucosa was studied in male C57BL/6 mice and male Wistar rats. In rats killed 4 hr after po cycasin, dose-dependent DNA damage was found in the liver and, to a lesser extent, in the kidney and colon mucosa; the lung appeared insensitive to the drug. The max effect was seen by 4 hr for the liver, by 2-4 hr for the kidney, and by 4-6 hr for the colon mucosa. In mice killed 6 hr after po cycasin or 4 hr after ip cycasin, liver DNA showed a dose-dependent increase in elution rate, the max effect occurring by 6-12 hr. Cycasin had practically no effect on the DNA of lung, kidney, or colon mucosa. DNA repair up to 18 hr appeared to be incomplete in both rats and mice. No evidence of cellular necrosis was seen in any organ 24 hr after cycasin treatment. Human EUE cells exposed to 1 or 2 mg/ml cycasin for 1 hr showed

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increased DNA elution rates without single-strand breaks. The increase was amplified by metabolic activation. The data indicate that cycasin is a peculiar type of procarcinogen that depends on a glucosidase for its activation to the biologically active aglycone methylazoxymethanol. (20 refs)

79-3102 Inhibition of the Mutagenicity of Methylazoxymethanol Acetate (MAMOAc) by Lithocholic Acid (LA) (Meeting Abstract). (Eng) Kanagal-ingam, K. (Chemical Carcinogenesis Program, NCI-Frederick Cancer Res. Center, Frederick, MD 21701); Andrews, A. W. *Proc Am Assoc Cancer Res* 20: 128; 1979 (no refs)

79-3103 Carcinogenic Effects of Cycasin and Methylazoxymethanol (MAM)-Acetate in Non-human Primates (Meeting Abstract). (Eng) Adamson, R. H. (NCI, Bethesda, MD 20014); Sieber, S. M.; Gargus, J. L. *Proc Am Assoc Cancer Res* 20: 218; 1979 (no refs)

79-3104 DNA Repair is Reduced in Lungs of Mice Exposed to Cigarette Smoke (Meeting Abstract). (Eng) Rasmussen, R. E. (Dept. Community and Environmental Medicine, Univ. California, Irvine, CA 92717); Boyd, C. H.; Kouri, R. E. *Proc Am Assoc Cancer Res* 20: 18; 1979 (no refs)

79-3105 Role of DNA Structure in Carcinogen-DNA Interaction (Meeting Abstract). (Eng) Sarma, S. R. (Dept. Pathology, Univ. Toronto, Toronto, Ontario M5S 1A8, Canada); Rao, P. M.; Sarma, D. S. *Proc Am Assoc Cancer Res* 20: 52; 1979 (no refs)

79-3106 Carcinogen-induced Unscheduled DNA Synthesis (UDS) in Primary Cultures of Adult Rat Hepatocytes (Meeting Abstract). (Eng) Sirica, A. E. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706); Hwang, C. G.; Sattler, G. L. *Proc Am Assoc Cancer Res* 20: 143; 1979 (1 ref)

79-3107 Human Liver Apurinic Deoxyribonucleic Acid (DNA) Endonuclease (Meeting Abstract). (Eng) Traina, V. (Microbiology Dept., Tulane Medical Sch., New Orleans, LA 70112); Liu, L.; Springgate, C. *Proc Am Assoc Cancer Res* 20: 9; 1979 (no refs)

79-3108 In Vitro Assay of Nuclear and Mitochondrial Hydroxyurea-resistant DNA Synthesis After In Vivo Exposure to Methylmercury (Meeting Abstract). (Eng) Miller, C. T. (Food Toxicology Div., Health Protection Branch, Health and Welfare Canada, Tunney's Pasture, Ottawa K1A 0L2, Canada). *Proc Am Assoc Cancer Res* 20: 290; 1979 (no refs)

79-3109 Potentiating Effect of Methyl Methane Sulfonate on Friend Virus Leukemogenesis In Vivo (Meeting Abstract). (Eng) Raikow, R. B. (Allegheny General Hosp., 320 East North Ave., Pittsburgh, PA 15212). *Proc Am Assoc Cancer Res* 20: 86; 1979 (1 ref)

79-3110 Ploidy Dependence of Induced Frequency in Somatic Mutation and Neoplastic Transformation (Meeting Abstract). (Eng) Morry, D. (Div. Biophysics, Johns Hopkins Univ., Baltimore, MD 21205). *Proc Am Assoc Cancer Res* 20: 184; 1979 (no refs)

79-3111 Forward Mutation Assays Using Bacteria and 8-Azaguanine. (Eng) Castellino, S. (Gruppo Mutagenesi Ambientale, Montedison S.p.A., Divisione Ricerche e Sviluppo e Istituto Ricerche, Carlo Erba, Milan, Italy); Lorenzetti, R.; Ravenna, L.; de Carneri, I. *Ecotox-icol Environ Saf* 2(3/4): 277-299; 1978.

The incidence of forward mutations employing 8-azaguanine-resistance and reverse mutations using histidine independence was compared in several strains of *Salmonella typhimurium*. The test compounds, which cause missense or frameshift mutations in the indicator strains, were N-methyl-N'-nitro-N-nitrosoguanidine, ethyl methanesulfonate, methyl methanesulfonate, β -propiolactone, streptozotocin, hycanthone, 9-aminoacridine, and 2-nitrofluorene. All eight compounds gave positive results with *S. typhimurium* strain TA1535 in the forward mutation assay. Six compounds were also positive with TA98 and TA100; 9-aminoacridine and 2-nitrofluorene were the exceptions. The presence of the R factor in strains TA98 and TA100 may prevent these two compounds from being mutagenic in this assay. All eight compounds gave positive results in the reverse mutation tests in strains with genetic alterations specific for the action of each compound. Selection for histidine independence generally produced higher mutation frequencies than selection for 8-azaguanine resistance, but it required a minimum of three strains for the eight compounds. It is concluded that neither of the methods has marked advantages over the other in detecting mutagenicity. (8 refs)

- 79-3112 Effect of Cell Growth Rate and Dose Fractionation on Chemically-induced Ouabain-resistant Mutations in Chinese Hamster V79 Cells.** (Eng) Lankas, G. R. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439). *Mutat Res* 60(2): 189-196; 1979.

The effect of reducing the cell growth rate on the number of ouabain-resistant mutants induced by N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) was studied using Chinese hamster V79 cells. Cells grown in medium with 10% fetal calf serum (FCS) grew faster than those grown in medium with 2% FCS. The reduced growth in 2% FCS was greater after treatment of the cells with 0.5 µg/ml MNNG than after treatment with 1.5 µg/ml. At 0.5 µg/ml MNNG, cell survival was greater and the mutation frequency lower in the slowly growing cells than in the faster growing cells. At 1.5 µg/ml MNNG, growth rate reduction did not affect cell survival or mutation frequency significantly. Two consecutive 0.75 µg/ml doses of MNNG were as toxic as 1.5 µg/ml given for half the time (1 hr), but the number of mutants induced by the split dose was significantly greater than the number induced by the single 1.5-µg/ml dose. There was an exponential decline in the percent of surviving cells and an exponential increase in mutation frequency as a function of duration of exposure to 48 µg/ml methylazoxymethanol acetate. The results demonstrate that growth rate affects the mutation frequency in V79 cells treated with low doses of chemical mutagens. (17 refs)

- 79-3113 Inhibition by Monofunctional Alkylating Agents of DNA Synthesis in Mutable and Transformable Cultured Mammalian Cells (Meeting Abstract).** (Eng) Peterson, A. R. (Univ. Southern California Comprehensive Cancer Center, Los Angeles, CA 90033); Altenbach, S.; Groom, D. A.; Heidelberger, C. *Proc Am Assoc Cancer Res* 20: 60; 1979 (no refs)

- 79-3114 Selectivity of the Excision of Alkylation Products in a Xeroderma Pigmentosum-derived Lymphoblastoid Line.** (Eng) Altamirano-Dimas, M. (Dept. Microbiology, Univ. Chicago, Chicago, IL 60637); Sklar, R.; Strauss, B. *Mutat Res* 60(2): 197-206; 1979.

The ability of Raji cells and human lymphoblastoid cells (XPA-3, derived from a group C xeroderma pigmentosum patient) to excise O⁶-methylguanine (OMG) and 3-methyladenine formed by reaction of the cells with N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) or methyl methanesulfonate (MMS) was studied. Both Raji and XPA-3 cells were competent in excision repair synthesis after treatment with MMS or MNNG. However, although Raji cells removed significant amounts of OMG from their DNA after treatment with MNNG, XPA-3 cells removed

relatively little. Both cell lines removed 3-methyladenine rapidly. The single gene *Escherichia coli* mutants *uvrA* and *uvrB*, which are UV-sensitive because of a defect in excision repair, were able to excise MNNG-induced OMG adducts, indicating the excision of this compound was not due to operation of the UV endonuclease system. Treatment of XPA-3 cells with MNNG resulted in numerous single-strand breaks in the DNA. The results support the hypothesis that base excision/apurinic repair and nucleotide excision proceed independently. It is predicted that xeroderma lines should be more sensitive than normal cells to the mutagenic effect of compounds such as MNNG, which produce considerable amounts of OMG. (24 refs)

- 79-3115 Decreased Loss of N⁵-Methylguanine and O⁶-Methylguanine During the S Phase in Synchronized 10T1/2 Cells (Meeting Abstract).** (Eng) Smith, G. J. (Univ. North Carolina, Chapel Hill, NC 27514); Kaufman, D. G.; Grisham, J. W. *In Vitro* 15(3): 224; 1979 (no refs)

- 79-3116 Promoting Effect of Cholesterol Metabolites and Bile Acids on Colon Carcinogenesis in Germfree (GF) and Conventional (Conv) Rats (Meeting Abstract).** (Eng) Reddy, B. S. (American Health Foundation, Valhalla, NY 10595); Watanabe, K. *Proc Am Assoc Cancer Res* 20: 125; 1979 (no refs)

- 79-3117 Studies on the Role of Basal Cells in the Histogenesis of Adenocarcinoma of Human Prostate (Meeting Abstract).** (Eng) Heatfield, B. M. (Universities Associated for Res. and Education in Pathology, Bethesda, MD 20014); Sanefuji, H.; Mostofi, F. K.; Trump, B. F. *Proc Am Assoc Cancer Res* 20: 129; 1979 (no refs)

- 79-3118 Glycolipids in Rat Duodenal Cells During Differentiation and Carcinogenesis (Meeting Abstract).** (Fre) Bouhours, J. F. (Laboratoire de Physiopathologie Digestive, Hopital Edouard-Herriot, F 69374 Lyon Cedex 2, France); Martin, M.; Bouhours, D. *Gastroenterol Clin Biol* 3(1): 78; 1979 (no refs)

- 79-3119 Malignant Transformation of Canine Cells In Vitro by a Chemical Carcinogen (Meeting Abstract).** (Eng) Rhim, J. S. (Microbiological Assoc., Inc., Bethesda, MD 20016); Arnstein, P. *Proc Am Assoc Cancer Res* 20: 5; 1979 (no refs)

79-3120 Multiple Hepatic Cysts Induced Experimentally by N-Methyl-N'-nitro-N-nitrosoguanidine in the Rat. (Fre) Justrabo, E. (Laboratoire d'Anatomie Pathologique, Faculte de Medecine, 21000 Dijon, France); Michiels, R.; Martin, M. S.; Martin, F. *Ann Gastroenterol Hepatol (Paris)* 15(1): 9-16; 1979.

The induction of multiple hepatic cysts by N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) was studied in 158 male and female BD IX, BN, Lewis, Wistar and Lewis (female) x BN (male) hybrid rats. Starting at age 6-8 wk, the animals received drinking water containing 83 mg/liter of MNNG for 7 mo. The surviving animals were sacrificed when the daily examination revealed hepatomegaly, ascites, wt loss, bloody stools, or bronchopneumopathy. One hundred and six gastric carcinomas and 84 intestinal tumors were found, and they were almost always associated with multiple hepatic cysts. The cyst induction rates were 100% in the BDIX, BN, and hybrid rats, 84% in the Lewis males, 94% in the Lewis and Wistar females, and 81.8% in the Wistar males. The cysts ranged from a few to 15 mm in diameter. They were filled with a lemon-yellow or turbid liquid, they had thin translucent walls, and they were closely clustered near the portal tracts. Ultrastructural study revealed the biliary nature of the cysts. The histological picture resembled that of polycystic disease in humans. (34 refs)

79-3121 Effect of the Carcinogen MNNG and Experimental Ulcer on Gastroduodenal Epithelial Proliferation in the Rat (Meeting Abstract). (Eng) Quimby, G. F. (Univ. Massachusetts Medical Sch., Worcester, MA); Eastwood, G. L. *Gastroenterology* 76(5, part 2): 1221; 1979 (no refs)

79-3122 Mutagenicity and Carcinogenicity of N-Methyl-N'-nitro-N-nitrosoguanidine. I. Induction of Chromosome Aberrations and Mitotic Anomalies in Chinese Hamster Ovary Cells. (Eng) Bemping, M. A. (Biomedical Res. Center, Norfolk State Coll., Norfolk, VA 23504). *J Environ Pathol Toxicol* 2(3): 633-656; 1979.

The spectrum of chromosome aberrations induced by N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) in Chinese hamster ovary (CHO) cells is reported. Cultured CHO cells were exposed to 0.25-2.0 µg/ml MNNG for 24 hr or 6 days. For cytological studies, CHO cells were treated for 24 hr and allowed a 24-hr recovery period prior to analysis; for cytotoxicity evaluations, cells were treated for 6 days followed by a 6-day recovery period. With respect to the cytotoxic effect of MNNG, there was a semilogarithmic decrease in the number of colonies formed with an increase in MNNG concentration. Cytologically, the chemical caused both structural and numerical chromosome anomalies,

and the incidence of each aberration type was influenced by MNNG concentration. Karyomorphological analysis of CHO cells treated for 24 hr and fixed after four cell cycles showed a varied chromosome constitution ranging from 18 to 54. Changes in chromosome structure arising from breaks and/or exchanges were of chromatid type. The distribution of breaks along the length of the chromosomes and among chromosomes showed that no chromosome segment or chromosome was preferentially susceptible to the clastogenic effects of MNNG. MNNG-induced chromosome rearrangements, such as ring configurations, exchanges, and dicentrics, exceeded such cytological errors as fragments and pulverizations. In a descending order of magnitude, the induced structural rearrangements were ring chromosomes, triradials, quadriradials, and dicentrics. (26 refs)

79-3123 Induction of Intestinal Metaplasia and Carcinoma in the Glandular Stomach of Rats by N-Alkyl-N'-nitro-N-nitrosoguanidines. (Eng) Matsukura, N. (Biochemistry Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan); Kawachi, T.; Sugimura, T.; Nakadate, M.; Hirota, T. *Gann* 70(2): 181-185; 1979.

The effects of 0.34 mM solutions of four homologs of N-alkyl-N'-nitro-N-nitrosoguanidine (N-alkyl-NNG) on gastric histology were studied in male Wistar rats that received one of the four compounds in the drinking water for 12 mo. The rats were killed 18 mo following initiation of the experiment. The incidence of intestinal metaplasia (IM) in the glandular stomachs of rats that received N-propyl-NNG, N-butyl-NNG, N-isobutyl-NNG, N-pentyl-NNG, or pure water was 100%, 50%, 44%, 17%, and 11%, respectively. The incidence of IM in the first group was significantly higher than that in the other groups ($0.001 < P < 0.05$). In all groups, the metaplastic glands were found most frequently in the proximal pyloric region. Adenomas and well-differentiated adenocarcinomas were found in the pyloric region of 2/7 surviving rats treated with N-propyl-NNG; no tumors occurred in any of the other groups. These findings support human studies showing that there is a correlation between the incidence of IM and well-differentiated adenocarcinoma. Thus, some human food carcinogens may cause IM and adenocarcinoma. (12 refs)

79-3124 Catalytic Effect of *p*-Nitrosophenol on the Nitrosation of Diethylamine. (Eng) Walker, E. A. (International Agency Res. Cancer, 69008 Lyon, France); Pignatelli, B.; Castegnaro, M. *J Agric Food Chem* 27(2): 393-396; 1979.

The kinetics of the nitrosation of diethylamine in water at 37 C was determined by gas chromatography. The reaction

was second-order with respect to nitrite, with a pH-independent rate constant of $0.87 \times 10^5 \text{ M}^{-2} \text{ sec}^{-1}$. The reaction catalyzed by p-nitrosophenol (p-NP) over a wide pH range was first-order with respect to nitrite, amine, and p-NP. A mechanism for this reaction is suggested. p-BP could be formed in the digestive tract by the reaction of phenol with nitrite ingested in saliva. Assuming complete nitrosation of the phenol, the rate of formation of nitrosodiethylamine under in vivo conditions could increase by a factor of about 140. Although this calculation is based on a model system and is an oversimplification, since there would be inhibiting factors as well as possible additional catalytic effects by other nitrosophenols, it illustrates a potential promoting effect on the in vivo formation of nitrosamines due to constituents in food. (25 refs)

79-3125 Dibenzofuran-free Aroclor 1254 as a Promoter of Diethylnitrosamine (DNA) Hepatocarcinogenesis in Rats (Meeting Abstract). (Eng) Preston, B. D. (Univ. Wisconsin, Madison, WI 53706); Van Miller, J. P.; Moore, R. W.; Allen, J. R. *Proc Am Assoc Cancer Res* 20: 153; 1979 (no refs)

79-3126 Induction of Nasopharyngeal Carcinoma in Rats by Nitroso Compounds. (Jpn) Cancer Research Group (Hunan Medical Coll., Hunan, People's Republic China). *Kexue Tongbao* 23(12): 756-760; 1978.

Rats weighing 100-150 g were divided into two groups. Group I received diethylnitrosamine intranasally, by sc injection in the back, or by rectal administration. Some rats in this group were also treated im with vitamin B₁₂ or po with 5% glucose. Group II rats received nitroso compounds intranasally or by sc injection. A high percentage of Group I rats developed nasopharyngeal carcinoma; some also had liver cancer. The vitamin B₁₂- and glucose-treated rats had a higher incidence of nasopharyngeal tumors but a lower incidence of liver tumors. Group II rats had a high incidence of nasopharyngeal carcinoma but a low incidence of liver cancer. (no refs)

79-3127 The Effects of Ethanol, Glucose and Sucrose on Nitrosation of Secondary Amines Following Alkalization of Reaction Mixture. (Eng) Yamamoto, M. (Dept. Food Additives, Natl. Inst. Hygienic Sciences, 18-1 Kamiyoga 1-chome, Setagaya-ku, Japan); Yamada, T.; Tanimura, A. *J Food Hyg Soc Jpn* 20(1): 15-20; 1979.

Nitrosodiethylamine (NDEA) formation was stimulated when diethylamine (DEA) was reacted with sodium nitrite at pH 3 in the presence of ethanol (E), glucose (G), or sucrose (S), and then the mixture was alkalinized with NaOH to terminate the reaction. E, G, and S had no effect on

NDEA formation when the reaction was terminated with sulfamic acid. It is postulated that the nitrous acid ester formed from sodium nitrite and E, G, or S in acidic medium nitrosates DEA in alkaline medium. (15 refs)

79-3128 An Experimental Approach for Evaluating Genetic and Epigenetic Contributions to Chemical Carcinogenesis (Meeting Abstract). (Eng) Reitz, R. H. (Toxicology Res., Dow Chemical Co., Midland, MI 48640); Schumann, A. M.; Watanabe, P. G.; Quast, J. F.; Gehring, P. J. *Proc Am Assoc Cancer Res* 20: 266; 1979 (1 ref)

79-3129 In Vivo Autoradiography with Labeled DEN as Related to Transplacental Carcinogenesis in Hamsters (Meeting Abstract). (Eng) Reznik-Schuller, H. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701). *Proc Am Assoc Cancer Res* 20: 110; 1979 (no refs)

79-3130 An Organ Culture System for Study of Carcinogenesis in the Rat Esophagus (Meeting Abstract). (Eng) Stoner, G. (Lab. Experimental Pathology, NCI, Bethesda, MD 20014); Autrup, H.; Pettis, W.; Harris, C. *Proc Am Assoc Cancer Res* 20: 64; 1979 (no refs)

79-3131 In Vivo Nitrosation of Dimethylamine Hydrochloride (DMA): Detection by an Intrahepatic Host-mediated Microbial Mutagenicity Assay (Meeting Abstract). (Eng) Edwards, G. S. (New England Inst. Life Sciences, Waltham, MA 02154); Whong, W. Z.; Speciner, N. D. *Proc Am Assoc Cancer Res* 20: 150; 1979 (no refs)

79-3132 DNA Methylation During Oxidative Dealkylation of Dimethylnitrosamine (DMN) by Liver Microsomes (Meeting Abstract). (Eng) Lotlikar, P. D. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA 19140); Smith, D. L.; Knight, R. C.; Magee, P. N. *Proc Am Assoc Cancer Res* 20: 124; 1979 (1 ref)

79-3133 Effect of Dimethylnitrosamine on Nuclear RNA Synthesis in Rat Liver and Kidney (Meeting Abstract). (Eng) Winicov, I. B. (Fels Res. Inst., Temple Univ. Sch. Medicine, Philadelphia, PA 19140). *Proc Am Assoc Cancer Res* 20: 41; 1979 (no refs)

- 79-3134 Identification of Methylated Bases in DNA Modified by Microsomal Activated Dimethylnitrosamine (Meeting Abstract). (Eng) Jensen, D. E. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA 19140); Lotlikar, P. D.; Magee, P. N. *Proc Am Assoc Cancer Res* 20: 121; 1979 (no refs)
- 79-3135 In Vivo Nitrosation of Secondary Amines at Low Levels (Meeting Abstract). (Eng) Krull, I. S. (New England Inst. Life Sciences, Waltham, MA 02154); Mills, K.; Goff, U.; Fine, D. H.; McDonald, J.; Iqbal, Z.; Epstein, S. S. *Proc Am Assoc Cancer Res* 20: 150; 1979 (no refs)
- 79-3136 Cytology of Fine Needle Aspiration Biopsy Material in Experimental Hepatocarcinogenesis: Morphological and Cytochemical Diagnosis of Preneoplastic Changes in the Rat (Meeting Abstract). (Ger) Boelsteri, U. (Institut für Toxikologie, ETH und Universität Zurich, Zurich, Switzerland); Zbinden, G. *Schweiz Med Wochenschr* 109(10): 367-368; 1979 (no refs)
- 79-3137 Species Differences in the Activation and Metabolism of Diallylnitrosamine (Meeting Abstract). (Eng) Grandjean, C. J. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105); Knepper, S. *Proc Am Assoc Cancer Res* 20: 172; 1979 (no refs)
- 79-3138 Methylation of Neuronal and Glial Macromolecules by Methylnitrosourea and Dimethylnitrosamine In Vivo (Meeting Abstract). (Eng) Hemminki, K. (Inst. Occupational Health, Haartmanink. 1, Helsinki 29, Finland); Savolainen, H. *Proc Am Assoc Cancer Res* 20: 76; 1979 (no refs)
- 79-3139 Quantitative Determination of Alkylated Purines in DNA Using a Highly Sensitive Non-radioactive Technique (Meeting Abstract). (Eng) Herron, D. C. (Univ. California, Irvine, CA 92717); Shank, R. C. *Proc Am Assoc Cancer Res* 20: 72; 1979 (no refs)
- 79-3140 Near Quantitative Production of Molecular Nitrogen From Metabolism of Dimethylnitrosamine. (Eng) Milstein, S. (Dept. Biochemistry, New York Univ. Dental Center, New York, NY 10010); Guttenplan, J. B. *Biochem Biophys Res Commun* 87(1): 337-342; 1979.
- The metabolism of dimethylnitrosamine (DMN) to molecular nitrogen was investigated in rat and mouse liver microsomes. As a positive control, N₂ production from N-methyl-N-nitrosourea (NMU) was studied. N₂ was produced stoichiometrically from the spontaneous decomposition of NMU in phosphate buffer and was readily detected by gas chromatography. N₂ production from DMN was about 70% that of formaldehyde when either rat or liver microsomes were used. The stoichiometric production of N₂ from NMU decomposition and the fact that NMU and DMN are believed to generate a common intermediate, monomethylnitrosamine, support a proposed pathway in which DMN is metabolized to methyldiazonium ion, which rapidly decomposes to methyl carbonium ion and N₂; the carbonium ion, being an S_N1 type alkylating agent, may react with water, producing methanol, or with cellular nucleophiles, leading to toxicity and genetic alterations. (17 refs)
- 79-3141 Effect of Vegetable Juices and Milk on Alkylating Activity of N-Methyl-N-nitrosourea. (Eng) Yano, K. (Dept. Chemistry, Saitama Medical Sch., 981 Kawakado, Moroyama, Iruma-gun, Saitama 350-04, Japan). *J Agric Food Chem* 27(2): 456-458; 1979.
- The effect of vegetable juices and milk, low-risk foods for gastric cancer, on the alkylation of 4-(p-nitrobenzyl)pyridine by N-methyl-N-nitrosourea (MNU) was studied to gain information about dietary factors for stomach cancer. A spectrophotometer was used to measure the absorbance of a mixture of MNU (7.2 micromoles) in 400 μ l of each test liquid before and after incubation at 37 C for 200 min. The juices and milk effectively decomposed MNU and consequently decreased its alkylating activity. The effectiveness (per dried materials) of the test liquids decreased in the order radish > cabbage > garden peas > lettuce > cucumber > celery > milk > tomato. The alkylating activity of N-methyl-N'-nitro-N-nitrosoguanidine was similarly reduced by the vegetable juices and milk. The results suggest that fresh vegetables and dairy products play an important role in stomach cancer prevention by reducing the alkylating properties of chemical carcinogens. (11 refs)
- 79-3142 Tissue Distribution of 1-Methyl-1-nitrosourea (MNU) and 1-Methyl-3-nitro-1-nitrosoguanidine (MNNG) in Guinea Pigs (Meeting Abstract). (Eng) Pinsky, S. D. (Div. Medical Oncology, V. T. Lombardi Cancer Res. Center, Georgetown Univ. Hosp., Washington, DC 20007); Woolley, P. V. *Proc Am Assoc Cancer Res* 20: 199; 1979 (no refs)

- 79-3143 The Effect of Alkylnitrosourea on a Coded Cell-free Synthesis System (Meeting Abstract). (Eng) Wei, S. C. (NCI, NIH, Bethesda, MD 20014); Chen, B. P. *Proc Am Assoc Cancer Res* 20: 32; 1979 (no refs)

- 79-3144 Comparison of Tumor Induction by Ethylnitrosourea and Methylnitrosourea in a Localized Lung Tumor Model (Meeting Abstract). (Eng) Grubbs, C. J. (IIT Res. Inst., Chicago, IL 60616); Moon, R. C. *Proc Am Assoc Cancer Res* 20: 97; 1979 (no refs)

- 79-3145 MNU Activates Endogenous DNA Synthesis by Isolated Hepatic Nuclei (Meeting Abstract). (Eng) Kaufmann, W. K. (Dept. Pathology, Univ. North Carolina Medical Sch., Chapel Hill, NC 27514); Grisham, J. W. *Proc Am Assoc Cancer Res* 20: 252; 1979 (no refs)

- 79-3146 4-Propionoxyphenyl Retinamide, A New Retinoid for Studies of Prevention of Cancer in Experimental Animals (Meeting Abstract). (Eng) Newton, D. L. (NCI, Bethesda, MD 20014); Smith, J. M.; Phillips, S. L.; Sporn, M. B. *Proc Am Assoc Cancer Res* 20: 120; 1979 (1 ref)

- 79-3147 Probes to Study the Effect of Methyl Nitrosourea (MNU) on ADP-Ribosylation and Chromatin Structure at the Subunit Level (Meeting Abstract). (Eng) Jump, D. B. (Biochemistry Dept., Lombardi Cancer Res. Center, Georgetown Univ. Medical Sch., Washington, DC 20007); Sudhakar, S.; Smulson, M. *Proc Am Assoc Cancer Res* 20: 191; 1979 (no refs)

- 79-3148 NMU-induced Polycarpellary Mutants in Redgram (Letter to Editor). (Eng) Chaturvedi, S. N. (Dept. Botany, R.B.S. Coll., Agra, India); Sharma, R. P. *Curr Sci (Bangalore)* 47(24): 960-961; 1978.

Four polycarpellary mutants were screened from the M_2 progeny of *Cajanus cajan*, whose seeds had been soaked in 0.03% nitrosomethylurea (NMU) for 14 hr prior to planting. All the mutants forming viable seeds bred true in the M_3 generation. At the 10- to 12-leaf stage, apical growth of the main shoot of the mutants was inhibited. All polycarpellary mutants had five types of flowers that exhibited an increase in the number of carpels. (6 refs)

- 79-3149 Bone Marrow and Thymus Depletion in RFM and C57BL Mice by N-Methyl-N-Nitrosourea

in Relation to Yield of Thymic Lymphomas and Methylation of DNA (Meeting Abstract). (Eng) Fry, P. M. (Pollards Wood Res. Station, Chalfont St. Giles, Bucks, England); Frei, J. V.; Lawley, P. D. *Proc Am Assoc Cancer Res* 20: 15; 1979 (no refs)

- 79-3150 Intercellular Junctions in Methylnitrosourea (MNU)-induced Carcinoma of the Rat Urinary Bladder (Meeting Abstract). (Eng) Severs, N. J. (Cell Pathology Unit, Sch. Pathology, Middlesex Hosp. Medical Sch., London, England); Hicks, R. M. *Br J Cancer* 39(4): 485; 1979 (3 refs)

- 79-3151 Distribution of O⁶-Methylguanine in Rat DNA Following Pretreatment In Vivo with Methylnitrosourea. (Eng) Chang, M. J. (Dept. Veterinary Pathobiology, Ohio State Univ., 1925 Coffey Road, Columbus, OH 43210); Webb, T. E.; Koestner, A. *Cancer Lett* 6(3): 123-127; 1979.

The possibility of a difference in the distribution of O⁶-methylguanine (O⁶-MG) within unique and repetitive DNA sequences of rat organs with different carcinogenic susceptibilities to N-methyl-N-nitrosourea (MNU) was investigated. Ten-day-old Sprague-Dawley rats were inoculated with MNU (80 µg/g ip), and brain, kidney, and liver were pooled 2 hr later. The unique and repetitive DNA fractions from each organ were separated, and the amount of O⁶-MG was determined chromatographically. There was little difference in the relative distribution of O⁶-MG between the unique and repetitive DNA sequences of all the organs examined. These results suggest that neoplastic transformation may involve not only mutations in the structural genes but also changes in the regulatory apparatus. Methylation of the O⁶-position of guanine in DNA occurred to similar extents in brain (the highest risk organ), kidney (of intermediate oncogenic susceptibility), and liver (the lowest risk organ). It is suggested that the significance of the formation of O⁶-MG to carcinogenesis will eventually be explained in terms of its persistence in specific DNA fractions. (16 refs)

- 79-3152 Steroid Chromatin Interactions Modify Nuclear Alkylation by Nitrosoureas (Meeting Abstract). (Eng) Tew, K. (Dept. Biochemistry, Div. Medical Oncology, Vincent T. Lombardi Cancer Res. Center, Georgetown Univ. Sch. Medicine, Washington, DC 20007); Schein, P.; Saslaw, L. D.; Smulson, M. *Proc Am Assoc Cancer Res* 20: 191; 1979 (no refs)

- 79-3153 Carcinogenic Action of BCNU Analogs after Repeated Intraperitoneal Application to

Sprague-Dawley (SD) Rats (Meeting Abstract). (Eng) Eisenbrand, G. (German Cancer Res. Center, 69 Heidelberg, W. Germany). *Proc Am Assoc Cancer Res* 20: 46; 1979 (no refs)

79-3154 Distribution of Nitrosoureas in Mice (Meeting Abstract). (Eng) Hill, D. L. (Kettering-Meyer Lab., Southern Res. Inst., Birmingham, AL 35205); Kari, P.; McConnell, W. R. *Proc Am Assoc Cancer Res* 20: 33; 1979 (no refs)

79-3155 Thyroid Tumors in Rats from Tetramethylthiourea. (Eng) Stula, E. F. (Haskell Lab. Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Wilmington, DE 19898); Sherman, H.; Barnes, J. R. *J Environ Pathol Toxicol* 2(3): 889-906; 1979.

The results of long-term toxicity testing of tetramethylthiourea (TMTU), an accelerator for neoprene vulcanization, in rats are presented. TMTU was added to the diet of male and female ChR-CD rats for up to 2 yr at levels of 0, 30, and 300 ppm. Fifty rats of each sex were used at each dietary level. High-level males and females and low-level males had a lower body wt and consumed less diet than did the controls. A slight anemia was found in the high-level males and females. Polyuria of a low osmolality was found in high level males and females. Serum thyroxine was lower in high-level males and females than in controls. TMTU-related gross and histologic changes were detected only in the thyroids of high-level males and females. After 3 mo on the test diet, the thyroids were enlarged and dark on gross examination. Histologically, there was an increase in follicle lumen size, loss of colloid staining, hypertrophy and hyperplasia of follicular cells, plus hyperemia. These early lesions were diffuse in distribution. After 18-24 mo on the test diet, 8/29 female high-level rats developed follicular cell carcinomas; no such tumors were observed in 26 comparable controls ($p < 0.0005$). Although no metastases were found, capsular and vascular invasion by tumor cells was seen. TMTU was considered to be carcinogenic for the female rat under the conditions of this test. (16 refs)

79-3156 Postulated Proximate Pancreatic Carcinogens in Syrian Hamsters (Meeting Abstract). (Eng) Pour, P. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105); Wallcave, L.; Gingell, R.; Lawson, T.; Grandjean, C. *Proc Am Assoc Cancer Res* 20: 119; 1979 (no refs)

79-3157 Single Exposure to a Carcinogen: Effect on Pancreatic Function (Meeting Abstract). (Eng)

Doria, J. C. (Dept. Surgery, Veterans Admin. Medical Center, Columbia, MO); Mosley, J. G.; Reber, H. A. *Gastroenterology* 76(5, part 2): 1124; 1979 (no refs)

79-3158 Mutagenic Activities of Three Pancreatic Carcinogens in the Liver Cell-mediated V79 Cell Mutagenesis System and the *Salmonella Typhimurium* Assay (Meeting Abstract). (Eng) Langenbach, R. (Eppley Inst. Res. Cancer, Omaha, NE 68105); Tompa, A.; Kuszynski, C.; Gingell, R.; Walker, B.; Pour, P. *Proc Am Assoc Cancer Res* 20: 229; 1979 (no refs)

79-3159 Comparative Carcinogenicity of Some Derivatives of Nitrosodi-n-propylamine in Rats. (Eng) Lijinsky, W. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, P.O. Box B, Frederick, MD 21701); Taylor, H. W. *Ecotoxicol Environ Saf* 2(3/4): 421-426; 1978.

The carcinogenicity of nitrosodi-n-propylamine (NDPA), two β -oxidized derivatives, nitrosobis(2-hydroxypropyl)amine (I) and nitrosobis(2-oxypropyl)amine (II), and halogenated derivative, nitrosobis(2-chloropropyl)amine (III), was compared in female Sprague-Dawley rats. NDPA and compounds I and II were given in the drinking water at equimolar doses at concentrations of 90, 108, and 106 mg/liter, 5 days/wk, for 30, 50, and 30 wk, respectively. Compound III was given by gavage: 0.2 ml of a 34-mg/ml soln in olive oil 2x/2w for 20 wk. NDPA was the most potent carcinogen. Compound I had the least effect on survival and was a relatively weak carcinogen. The effectiveness of the chlorinated derivative was difficult to assess because of its toxicity. NDPA was a broadly acting carcinogen and induced a high incidence of tumors in the liver, nasal turbinates, and esophagus. The three derivatives appeared to be less broadly acting, eg, compound II only induced liver tumors. These results indicate that the hypothetical formation of oxygenated derivatives of NDPA in vivo is not a likely mechanism of carcinogenesis. (8 refs)

79-3160 Inhibition of N-Butyl-N(4-hydroxybutyl)-nitrosamine (BHBN) Induced Bladder Cancer in Rats by Disulfiram (DSF) (Meeting Abstract). (Eng) Irving, C. C. (Veterans Admin. Medical Center, Memphis, TN); Tice, A. J.; Murphy, W. M. *Proc Am Assoc Cancer Res* 20: 92; 1979 (no refs)

79-3161 Inhibition of Urinary Bladder Cancer in the Rat by Retinoids (Meeting Abstract). (Eng) Thompson, H. J. (IIT Res. Inst., Chicago, IL 60616); Beci, P. J. *Proc Am Assoc Cancer Res* 20: 96; 1979 (no refs)

79-3162 Mapping Evaluation for Whole Urinary Bladder Epithelium of Dogs During BBN Carcinogenesis (Meeting Abstract). (Eng) Hirao, K. (Dept. Urology, Nara Medical Univ., Nara, 634, Japan); Hiramatsu, T.; Ijuin, M.; Okajima, E.; Ito, N. *Proc Am Assoc Cancer Res* 20: 47; 1979 (no refs)

79-3163 N-Nitrosamines in the Diet of Experimental Animals. (Eng) Walker, E. A. (International Agency Res. Cancer, Lyon, France); Castegnaro, M.; Gričute, L. *Cancer Lett* 6(3): 175-178; 1979.

Eighty-seven samples of experimental animal feed were analyzed in four independent laboratories for the presence of chemical carcinogens. Volatile N-nitrosamines were found regularly, the most common being N-nitrosodimethylamine (90% of samples at levels of 1-10 µg/kg). The highest level found was 300 µg/kg for N-nitrosopiperidine, which represented one isolated case. Other N-nitrosamines detected included N-nitrosodiethylamine, N-nitrosodipropylamine, N-nitrosodibutylamine, and N-nitrosopyrrolidine. Since all animal experiments include control groups for which pathology is performed, it is suggested that collective analysis of the food in these experiments could provide data on effects in animals of exposure to low levels of N-nitrosamines. (8 refs)

79-3164 Metabolism of Benzo[a]pyrene (BP), N-Nitrosodimethylamine (DMN) and N-Nitrosopyrrolidine (NPy) and Identification of the Major Carcinogen-DNA Adducts Formed in Cultured Human Esophagus (Meeting Abstract). (Eng) Harris, C. C. (Human Tissue Studies Section, LEXP, NCI, Bethesda, MD 20014); Autrup, H.; Trump, B. F.; Jeffrey, A. M. *Proc Am Assoc Cancer Res* 20: 65; 1979 (no refs)

79-3165 In Vitro Binding of Diethylnitrosamine (DEN) and N-Nitrosopiperidine (NPiP) to DNA (Meeting Abstract). (Eng) Lai, D. Y. (USPHS Res. Lab., Tulane Medical Center, 210 State St., New Orleans, LA 70118); Arcos, J. C.; Argus, M. F. *Proc Am Assoc Cancer Res* 20: 65; 1979 (no refs)

79-3166 Species Differences in Tumorigenic Response to Several Nitrosamines (Meeting Abstract). (Eng) Lijinsky, W. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701). *Proc Am Assoc Cancer Res* 20: 106; 1979 (no refs)

79-3167 Distribution of Nitrogen-13 from Labeled Nitrate ($^{13}\text{NO}_3^-$) in Humans and Rats. (Eng) Witter, J. P. (Dept. Medical Microbiology, Univ. Wisconsin Medical Sch., Madison, WI 53706); Gatley, S. J.; Balish, E. *Science* 204(27): 411-413; 1979.

The body distribution of po (by gavage) or iv administered nitrate labeled with nitrogen-13 was studied in two human volunteers and conventional-flora rats. The data suggested that the body can act as a temporary reservoir of ^{13}N (a significant amount of this label would probably be in the form of NO_3^-); the ^{13}N passes beyond the upper small intestine (duodenum and jejunum) when ingested; biliary, pancreatic, or intestinal secretions may contribute to the pool of ^{13}N present in the lower small and large intestines; and bowel alterations (resection or strangulation) may enhance the influx of NO_3^- into the bowel contents. These observations indicate that depletion of body stores, the passage of nitrate down the gut, or the secretion of nitrate into the intestinal lumen may be a better explanation of the urinary, ileal, and fecal concentrations of nitrate and nitrite recently measured in humans than a bacterial nitrification reaction in the intestines. (12 refs)

79-3168 Nitrate and Nitrite. (Eng) Jagerstad, M. (Dept. Clinical Res., Univ. Hosp., Lund, Sweden); Kolar, K.; Nilsson, R.; Norden, A. *Scand J Gastroenterol Suppl* 14(52): 214-216; 1979.

The mean daily intake of nitrate and nitrite was determined in pooled samples of lyophilized and fat-free food homogenate powder representing 7-day diets consumed by a group of pensioners in a predominantly agricultural area in Sweden. The mean daily intake of nitrate was 499 ± 271 micromoles (µmol) for the men and 307 ± 118 µmol for the women. Corresponding values for nitrite intake were 58 ± 21 and 36 ± 14 µmol for the men and women, respectively. No specific disease could be related to the highest intakes of nitrite. The only person known to have a malignant disease had a nitrate intake of 412 µmol/day. The nitrate intake among the men was <1.1 mg/kg/day and among the women was <0.8 mg/kg/day. The nitrite intake was <0.08 mg/kg/day for both men and women. These values are well within the acceptable levels recommended by the World Health Organization. However, there could be a salivary supply of 6-10 mg nitrite per day, which, together with the dietary intake, might exceed the accepted safe level for sodium nitrite of 0.2 mg/kg/day. (11 refs)

79-3169 An Improved Method for Nitrite Extraction from Plants. (Eng) Klepper, L. A. (Dept. Agronomy, Univ. Nebraska, Lincoln, NE 68583). *J Agric Food Chem* 27(2): 438-441; 1979.

A method for extracting nitrites from plant tissues is

presented that results in recoveries that are higher than those obtained by aqueous extraction. The soln to be extracted is treated with methylene chloride (MeCl_2) or insoluble polyvinylpyrrolidone (PVP) prior to extraction with sulfanilamide/ α -naphthylethylenediamine/HCl reagent. The mean recovery from a variety of plants, including tea, avocado, sweet potato, apple, wheat, sweet potato, radish, potato, and soy bean, was 89%-95%. These recoveries represented increases ranging up to 69.8% over those obtained with aqueous extraction. The lowest nitrite recovery (69%) was obtained from apple leaves; because of the high concentrations of tannins and polyphenols in this tissue, extraction might have been more effective with the addition of larger amounts of PVP to the extraction medium. In most extractions, the result after centrifugation was a clear, aqueous, nitrite-containing supernatant above the PVP/ MeCl_2 portion. Thus, additions of MeCl_2 and insoluble PVP can protect nitrite from reactive substances during aqueous extractions for nitrite analyses. (12 refs)

79-3170 Chronic Toxicity of Sodium Nitrite in Mice with Reference to its Tumorigenicity. (Eng) Inai, K. (Second Dept. Pathology, Hiroshima Univ. Sch. Medicine, Kasumi 1-2-3, Hiroshima 734, Japan); Aoki, Y.; Tokuoka, S. *Gann* 70(2): 203-208; 1979.

The toxicity and possible tumorigenicity of sodium nitrite (NaNO_2) were studied in 8-wk-old ICR mice. The 6-wk toxicity study demonstrated that the max tolerated dose of NaNO_2 was 0.5%. Three groups of 100 mice each received drinking water containing 0.5, 0.25, or 0.125% NaNO_2 for 109 wk; 40 controls received drinking water alone. With increasing dose, the mice drank less water, but the amount consumed by those receiving 0.5% NaNO_2 was approx 3x greater than that consumed by those receiving 0.125%. Although the tumor incidence in each experimental group appeared to exceed 50% of the effective mice (ie, those surviving more than 18 mo or those that died with malignant tumor during the experiment), there was no evidence of a relationship between the concentration or total amount of NaNO_2 consumed and tumor incidence. Thymus, lymph node, or bone marrow tumors occurred in 27% of the male controls and 33% of the female controls. In the experimental animals, the incidence of these tumors ranged between 11% and 44%, with the highest incidences found in mice that received 0.125% NaNO_2 . Total tumor incidence (including hematopoietic, soft tissue, lung, liver, uterus, mammary gland, and 10 miscellaneous tumors) in the experimental animals was not significantly different from that in the controls, and tumor latency periods were similar in both groups. These results, together with the fact that the daily NaNO_2 dose in animals receiving the highest level was approx 3x that consumed by persons in the US, suggest that dietary NaNO_2 levels are not tumorigenic. (15 refs)

79-3171 Carcinogenic Effect of N⁶-(Methylnitroso)-adenosine and Its Combined Precursors (Meeting Abstract). (Eng) Giner-Sorolla, A. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Anderson, L. M.; Greenbaum, J. H.; Barney, K.; Budinger, J. M. *Proc Am Assoc Cancer Res* 20: 89; 1971 (1 ref)

79-3172 Nitrosofluorene-Lipid Adducts. Observations on Their Mutagenicity and Electron Spin Resonance Spectra (Meeting Abstract). (Eng) Sridhar, R. (Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104); Hampton, M. J.; Floyd, R. A. *Proc Am Assoc Cancer Res* 20: 227; 1979 (1 ref)

79-3173 Comparative Carcinogenicity and Metabolism of the Tobacco Specific Carcinogens, NNN and NNK (Meeting Abstract). (Eng) Chen, C. B. (Naylor Dana Inst., American Health Foundation, Valhalla, NY 10595); Hecht, S. S.; Young, R.; Ohmori, T.; Hoffmann, D. *Proc Am Assoc Cancer Res* 20: 81; 1979 (no refs)

79-3174 Carcinogenicity of N-Nitroso Derivatives of N-Methylcarbamate Insecticides in Rats. (Eng) Lijinsky, W. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, P.O. Box B, Frederick, MD 21701); Schmahl, D. *Ecotoxicol Environ Saf* 2(3/4): 413-419; 1978.

N-Nitroso derivatives of N-methylcarbamate insecticides were synthesized and tested for carcinogenicity in rats. Sprague-Dawley rats (6-8 wk old) were given 1.0 ml/kg of a 60-mg/ml soln of nitrosocarbaryl (NC) in corn oil 1x/wk by gavage for a total dose of 600 mg/kg. Nitrosocarbaryl (NCF), nitrosobaygon, and nitrosomethylphenylcarbamate (NMPC) were so toxic at equimolar doses that the experiment was restarted at one-fourth the dose for a longer period of time. Other rats were given 0.2 ml of a soln of nitroso compound in olive oil at a concentration equivalent to 25 mg NC/ml 1x/wk by stomach intubation for 10 wk. Nitrosoaldicarb, NCF, and NMPC were toxic at this dose rate and at half this rate as well. All of the nitroso-N-methylcarbamates were carcinogenic, with the target organ being the nonglandular stomach. Nitrosobaygon, NCF, and nitrosolandrin were the most potent carcinogens, and there was little difference among them. NC, nitro-Bux-ten, and nitrosomethomyl were weak carcinogens. NMPC induced fewer tumors than the other compounds because its toxicity was higher and fewer rats survived to develop tumors. It is concluded that the formation of nitrosomethylcarbamates by reaction of the parent insecticide with nitrite in the environment or stomach represents an increased carcinogenic risk. (11 refs)

- 79-3175 Role of Liver Cell Necrosis in the Induction of Preneoplastic Lesions (Meeting Abstract). (Eng) Ying, T. S. (Dept. Pathology, Univ. Toronto, Toronto M5S 1A8, Canada); Sarma, D. S. *Proc Am Assoc Cancer Res* 20: 14; 1979 (1 ref)

- 79-3176 Induction of Sister Chromatid Exchange and Polyploidy by Carbaryl in V79 Cells (Meeting Abstract). (Eng) Sabharwal, P. S. (Univ. Kentucky, Lexington, KY 40506); Lockard, J. M. *In Vitro* 15(3): 172-173; 1979 (no refs)

- 79-3177 Effect of Implantation of Liver and Spleen Cells from Mature Parent Mice on Urethan Carcinogenesis in F₁ Hybrids. (Rus) Kraskovskii, G. V. (Inst. Genetics and Cytology, Minsk, USSR); Kagan, L. F. *Dokl Akad Nauk BSSR* 23(2): 176-179; 1979.

(Af x C57BL)F₁ female mice were inoculated with 10⁶ urethan (1.5 mg/mouse, ip) and, 1 day later, with 50 x 10⁶ lymphoid cells from C57BL mouse embryo liver. One month later, animals were inoculated ip with 50 x 10⁶ lymphoid cells from C57BL mouse embryo spleen. Implantation of liver cells alone did not change the incidence of urethan-induced lung adenomas (17.9%, vs 16.9% in mice exposed to urethan alone), but administration of spleen cells significantly decreased the incidence of lung adenomas (12.4%). (15 refs)

- 79-3178 Effect of Aluminium Chloride on Metabolism of 4-Nitroquinoline 1-Oxide. (Eng) Yamane, Y. (Faculty Pharmaceutical Sciences, Chiba Univ., 1-33, Yayoi-cho, Chiba 260, Japan); Ohtawa, M. *Gann* 70(2): 147-153; 1979.

Aluminum chloride (AlCl₃) was administered sc to male dd mice, and its effect on the activities of 4-nitroquinoline 1-oxide (4-NQO) 4-hydroxy-aminoquinoline 1-oxide (4-HAQO) reductases and the organ distribution of carcinogen(s) in mouse lung and liver were examined. AlCl₃ administration resulted in significant elevation of 4-NQO reductase and 4-HAQO reductase activities in both lung and liver, compared with the activities in control mice. Following the simultaneous administration of AlCl₃ and ¹⁴C-4-NQO, the distribution of radioactivity at 0.5 and 1 hr postadministration decreased in the lung but increased inversely in the liver. Radioactivity in the lung and liver 2 hr postadministration was not different from that in the controls. Simultaneous sc administration of ¹⁴C-4-NQO and AlCl₃ resulted in a decreased pulmonary distribution of 4-NQO and 4-HAQO but an increased distribution of the noncarcinogenic metabolites 4-aminoquinoline 1-oxide (4-AQO) and 4-hydroxyquinoline 1-oxide (4-OHQO). The

distribution of 4-aminoquinoline (4-AQ) and 4-hydroxyquinoline (4-OHQ) in the lung did not differ from that in the controls. The AlCl₃-induced decrease in the lung concentrations of 4-NQO and 4-HAQO may be a factor responsible for the suppression of lung carcinogenesis by AlCl₃. (13 refs)

- 79-3179 Mutagenic Effects of Sodium Azide in *Drosophila Melanogaster*. (Eng) Kambra, O. P. (Dept. Biology, Dalhousie Univ., Halifax N.S., Canada); Gollapudi, B. *Mutat Res* 66(4): 381-384; 1979.

The mutagenic effects of low-pH buffered solns of sodium azide (NaN₃, injected abdominally or given po) on male *Drosophila melanogaster* were studied by the sex-linked recessive-lethal test. Injection of NaN₃ at pH 4.6 or 3.8 had no significant effect on the frequency of recessive-lethal mutations in any of the male germ cell stages. A 24-hr feeding of 0.1 mM azide (pH 4.6) produced a nonsignificant increase in the frequency of sex-linked recessive-lethal mutations (0.98%) in brood B, which corresponded to spermatids. The overall mutation frequency in flies fed for 3 days on 0.1 mM azide (pH 4.6) was significantly higher than that among the controls and flies fed 5 mM azide (pH 7.0). Thus, even at low pH, NaN₃ is ineffective as a mutagen after injection into male *Drosophila*, but it is weakly mutagenic when fed for 3 days at a concentration of 0.1 mM at pH 4.6. (14 refs)

- 79-3180 Δ¹Tetrahydrocannabinol and 1α,2α-Epoxyhexahydrocannabinol: Mutagenicity Investigation in the Ames Test. (Eng) Glatt, H. (Section Biochemical Pharmacology, Inst. Pharmacology, Univ. Mainz, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany); Ohlsson, A.; Agurell, S.; Oesch, F. *Mutat Res* 66(4): 329-335; 1979.

Because many epoxides are mutagens, the mutagenicity of 1α,2α-epoxyhexahydrocannabinol (EHC) as well as that of Δ¹-tetrahydrocannabinol (THC) toward *Salmonella typhimurium* strains TA1535, TA1537, TA98, and TA100 was investigated in the presence and absence of an S9 mix from liver homogenates of Aroclor 1254-treated rats. Because EHC can be converted into a dihydrodiol by epoxide hydase, an epoxide hydratase inhibitor was added to the metabolizing system in some experiments. Although several other epoxides and additional positive controls, either not requiring activation or being activated under similar conditions, showed strong mutagenicity, there were no indications of a mutagenic hazard due to EHC or THC. (19 refs)

- 79-3181 Antioxidants Reduce the Mutagenic Effect of Malonaldehyde and β-Propiolactone. Part IX,

Antioxidants and Cancer. (Eng) Shamberger, R. J. (Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH 44106); Corlett, C. L.; Beaman, K. D.; Kasten, B. L. *Mutat Res* 66(4): 349-355; 1979.

The ability of antioxidants (vitamin C, vitamin E, selenium, and butylated hydroxytoluene) to reduce the mutagenic effects of malonaldehyde (MA) and β -propiolactone (PL) was studied using *Salmonella typhimurium* strains hisG46 and TA1975, which mutate by base-pair substitution, and variants hisC207, his3076, TA1977, hisD3052, and TA1978, which are frameshift mutants. MA increased mutagenesis in TA1975, hisC207, hisC3076, hisD3052, and TA1978 in a non-dose-dependent fashion, but it had no effect on hisG46. PL increased mutagenesis in all strains. The antioxidants decreased MA-induced mutagenesis in hisC207, hisC3076, TA1978, and, to some extent, in hisD3052, but not in hisG46 or TA1975. PL-induced mutagenesis was reduced by the antioxidants in all strains. (15 refs)

79-3182 Formation of 3-(2-Carboxyethyl)thymine Following In Vitro Reaction Between β -Propiolactone and Calf Thymus DNA (Meeting Abstract). (Eng) Segal, A. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, N.Y.U. Medical Center, New York, NY 10016); Mate, U.; Solomon, J. J. *Proc Am Assoc Cancer Res* 20: 3; 1979 (1 ref)

79-3183 Mutagenic Action of a Series of Epoxides. (Eng) Wade, M. J. (Life Sciences Res. Office, Federation American Societies Experimental Biology, 9650 Rockville Pike, Bethesda, MD 20014); Moyer, J. W.; Hine, C. H. *Mutat Res* 66(4): 367-371; 1979.

The mutagenicity of 13 epoxide compounds was determined in a bacterial plate assay system using the histidine-dependent *Salmonella typhimurium* tester strains TA98 (for frameshift mutagens) and TA100 (for base-pair substitution mutagens). Mutagenicity was evaluated both with and without the addition of a rat liver microsomal extract. Dieldrin, the diglycidyl ether of bisphenol A, and three of its homologs were not mutagenic. Allyl glycidyl ether, n-butyl glycidyl ether, vinyl cyclohexene diepoxide, glycidol, glycidaldehyde, diglycidyl ether, diepoxybutane, and the diglycidyl ether of substituted glycerine were mutagenic to TA100, causing reversion of the bacteria to histidine independence. Dose-response curves of the mutagenicity of the latter four compounds were obtained. On a molar basis, glycidaldehyde was about 20-50 times more potent in producing mutation than the other three epoxides. In general, the mutagenicity of the epoxides was not enhanced or diminished by the addition of the microsomal extract. (9 refs)

79-3184 Tumor Promoter Synergistically Enhances Cell Growth with a Variety of Serum Growth Factors (Meeting Abstract). (Eng) Frantz, C. N. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA 02115); Stiles, C. D.; Scher, C. D. *Proc Am Assoc Cancer Res* 20: 207; 1979 (no refs)

79-3185 Inhibition of Differentiation of Syrian Hamster Epidermal Cells in Culture by TPA (Meeting Abstract). (Eng) Sisskin, E. E. (NIEHS, Research Triangle Park, NC 27709); Barrett, J. C. *Proc Am Assoc Cancer Res* 20: 197; 1979 (no refs)

79-3186 Effect of 12-O-Tetradecanoylphorbol-13-acetate (TPA) on Mouse Epidermal Arginase and Ornithine Decarboxylase (ODC) Activities (Meeting Abstract). (Eng) Verma, A. K. (McArdle Lab., Univ. Wisconsin, Madison, WI 53706); Rice, H. M.; Shapas, B. G.; Boutwell, R. K. *Proc Am Assoc Cancer Res* 20: 193; 1979 (no refs)

79-3187 Effect of Polyamine (PA) Biosynthesis Inhibitors on Ornithine Decarboxylase (ODC) Activity, PA Levels, and Tumor Promotion (Meeting Abstract). (Eng) Weeks, C. E. (Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Bracken, W. M.; Slaga, T. J. *Proc Am Assoc Cancer Res* 20: 155; 1979 (no refs)

79-3188 Retinoic Acid Differentially Inhibits the Induction of Ornithine Decarboxylase (ODC) by 12-O-Tetradecanoylphorbol-13-acetate (TPA) and by Germicidal Ultraviolet Light (Meeting Abstract). (Eng) Lichti, U. (NCI, Bethesda, MD 20014); Patterson, E.; Yuspa, S. H. *Proc Am Assoc Cancer Res* 20: 105; 1979 (no refs)

79-3189 5,8,11,14-Eicosatetraynoic Acid (ETYA) Inhibits the Biological Action of 12-O-Tetradecanoylphorbol-13-acetate (TPA) in Bovine Lymphocytes (Meeting Abstract). (Eng) Wertz, P. W. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706); Mueller, G. C. *Proc Am Assoc Cancer Res* 20: 235; 1979 (no refs)

79-3190 Phorbol Esters and Melittin Alter Arachidonic Acid Metabolism and Share Other Phenotypic Effects on Cells in Culture (Meeting Abstract). (Eng) Mufson, R. A. (Div. Environmental Science, Columbia Univ.,

New York, NY 10032); Fisher, P. B.; Weinstein, I. B. *Proc Am Assoc Cancer Res* 20: 132; 1979 (no refs)

79-3191 Protein Fluorescence Polarization Studies on the Interaction of Phorbol Myristate Acetate with Mammalian Plasma Membranes (Meeting Abstract). (Eng) Witz, G. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016). *Proc Am Assoc Cancer Res* 20: 279; 1979 (no refs)

79-3192 The Effect of TPA on DNA Synthesis in Normal and RSV-transformed Rat Fibroblasts (Meeting Abstract). (Eng) Magun, B. E. (Univ. Arizona, Tucson, AZ 85724); Bowden, G. T. *Proc Am Assoc Cancer Res* 20: 88; 1979 (no refs)

79-3193 Decrease in Collagen Production in Normal and Rous Sarcoma Virus-transformed Chick Embryo Fibroblasts Induced by Phorbol Myristate Acetate. (Eng) Delclos, K. B. (Dept. Pharmacology, Harvard Medical Sch., Boston, MA 02115); Blumberg, P. M. *Cancer Res* 39(5): 1667-1672; 1979.

The ability of the potent tumor promoter phorbol-12-myristate 13-acetate (PMA) to inhibit collagen synthesis was investigated in chick embryo fibroblasts (CEF). Collagen synthesis, as measured by the rate of formation of [³H]hydroxyproline from [³H]proline, was decreased in cells treated with PMA (33 or 200 nanograms/ml) but not in cells treated with the parent alcohol phorbol (1 µg/ml). The effect was max at 33 nanograms/ml. The decrease in collagenase-sensitive proteins was confirmed by polyacrylamide gel electrophoresis of cell lysates, indicating that the decrease could not be ascribed simply to an effect on prolyl hydroxylase. Although a decrease in collagen synthesis was observed after 1 day, 5 days were required for a max reduction to 20% of that of dimethyl sulfoxide-treated controls. The effect of PMA on collagen synthesis was reversible. It was, therefore, not the result of a permanent transformation of the cells or of the selection of a cell population with a reduced capacity for collagen synthesis. Collagen synthesis was decreased in CEF transformed by Rous sarcoma virus. Treatment of these cells with PMA for 5 days brought about a further decrease to 50% of the level in dimethyl sulfoxide-treated transformed controls. (38 refs)

79-3194 Induction of Anchorage Independence by Tumor-promoting Phorbol Esters (Meeting Abstract). (Eng) Colburn, N. H. (NCI, NIH, Bethesda, MD 20014). *In Vitro* 15(3): 208; 1979 (no refs)

79-3195 The Relationship Between Morphology and Ornithine Decarboxylase Activity in Cultured Human and Hamster Cells (Meeting Abstract). (Eng) Warren, S. T. (Dept. Human Development and Biochemistry, Michigan State Univ., East Lansing, MI 48824); Moskal, J. R.; Mason, P. A.; Sweeley, C. C.; Trosko, J. E. *In Vitro* 15(3): 173; 1979 (no refs)

79-3196 Phorbol Myristate Acetate (PMA) Uncouples β -Adrenergic Receptors from Adenyl Cyclase in Mouse Epidermis: Antagonism by Butyric Acid (Meeting Abstract). (Eng) Garte, S. J. (New York Univ. Medical Center, New York, NY 10016); Belman, S. *Proc Am Assoc Cancer Res* 20: 52; 1979 (1 ref)

79-3197 The Action of a Tumor Promoter on the Mixed Lymphocyte Response (Meeting Abstract). (Eng) Mastro, A. M. (Dept. Biochemistry and Biophysics, Pennsylvania State Univ., University Park, PA 16802). *Proc Am Assoc Cancer Res* 20: 24; 1979 (2 refs)

79-3198 Promotion of Malignant Transformation in Carcinogen-exposed Tracheal Epithelium In Vitro (Meeting Abstract). (Eng) Steele, V. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Marchok, A. *Proc Am Assoc Cancer Res* 20: 136; 1979 (no refs)

79-3199 Promotion of Sister Chromatid Exchange by TPA in V79 Chinese Hamster Cells (Meeting Abstract). (Eng) Lockard, J. M. (Univ. Kentucky, Lexington, KY 40506); Chortyk, O.; Sabharwal, P. S. *In Vitro* 15(3): 172; 1979 (no refs)

79-3200 Tumor Promoter-induced Adhesion of the DS19 Clone of Murine Erythroleukemia Cells. (Eng) Yamasaki, H. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, 150 Cours Albert Thomas, Lyon 69372, France); Weinstein, I. B.; Fibach, E.; Rifkind, R. A.; Marks, P. A. *Cancer Res* 39(6, part 1): 1989-1994; 1979.

When suspension cultures of certain 12-O-tetradecanoylphorbol-13-acetate (TPA)-sensitive clones of murine erythroleukemia cells (MELC) are exposed to TPA, they become adherent to the tissue culture plate but TPA-resistant clones do not. The results of further studies of this phenomenon are presented. The TPA-induced adhesion of the DS19 clone of MELC to the surface of tissue culture flasks was apparent within 20 min and was max within 45

min. The effect required as little as 1-5 nanograms/ml TPA, and it was temperature-dependent. Adhesion was not inhibited by actinomycin D or cycloheximide, but partial inhibition was observed with colchicine and cytochalasin B. Among six phorbol esters and other plant diterpenes tested, there was good correlation between effectiveness in inducing MELC adhesion and mouse skin tumor-promoting activity. TPA-induced adhesion was not seen when MELC were grown in bacterial flasks, yet differentiation was inhibited under these conditions, indicating that adhesion is not a prerequisite for TPA inhibition of differentiation. Two subclones of DS19 that are resistant to TPA-induced inhibition of differentiation showed no adhesion to tissue culture flasks in the presence of TPA or other phorbol ester tumor promoters. When TPA-sensitive and -resistant cells were cocultured and TPA was added, almost all of the TPA-sensitive cells became adhesive to the plate, yet very few of the TPA-resistant cells became adhesive. These results suggest that an early site of action of TPA in MELC is the cell-surface membrane. The changes in MELC adhesion induced by TPA may provide clues to its mechanism of action and provide a rapid in vitro assay for certain types of tumor promoters. (35 refs)

79-3201 Increased Prostaglandin Levels in Mouse Epidermis Induced by Phorbol Esters (Meeting Abstract). (Eng) Ashendel, C. L. (McArdle Lab., Univ. Wisconsin, Madison, WI 53706). *Proc Am Assoc Cancer Res* 20: 268; 1979 (no refs)

79-3202 Induction of Terminal Differentiation in Human Myeloid Leukemia Cells by Tumor-promoting Agents (Meeting Abstract). (Eng) Huberman, E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830). *Proc Am Assoc Cancer Res* 20: 281; 1979 (no refs)

79-3203 In Vitro Studies on the Targets for Phorbol 12-Myristate-13-acetate and Related Diterpene Esters (Meeting Abstract). (Eng) Blumberg, P. M. (Dept. Pharmacology, Harvard Medical Sch., Boston, MA 02115); Driedger, P. E.; Nagle, D. S. *Proc Am Assoc Cancer Res* 20: 255; 1979 (no refs)

79-3204 The Tumor Promoter 12-O-Tetradecanoyl-phorbol-13-acetate (TPA) Elevates Serum Progesterone Levels in Chicks (Meeting Abstract). (Eng) Sharma, O. K. (AMC Cancer Res. Center and Hosp., Lakewood, CO 80214); Kerr, S. J. *Proc Am Assoc Cancer Res* 20: 194; 1979 (1 ref)

79-3205 Enzyme Responses to Tumor Promoters: Cathepsin B Induction in 10T1/2 Cells (Meeting Abstract). (Eng) Dolbeare, F. (Lawrence Livermore Lab., Livermore, CA 94550). *Proc Am Assoc Cancer Res* 20: 180; 1979 (2 refs)

79-3206 Increased Cyclic Adenosine 3':5'-Monophosphate Phosphodiesterase Activity in the Epidermis of Phorbol Ester-treated Mouse Skin and in Papillomas. (Eng) Mufson, R. A. (Inst. Cancer Res. Columbia Univ. Coll. Physicians and Surgeons, 701 W. 168 St., New York, NY 10032); Simsiman, R. C.; Boutwell, R. L. *Cancer Res* 39(6, part 1): 2036-2040; 1979.

The relationship of the phorbol ester-mediated induction of cyclic AMP phosphodiesterase activity to tumor promotion was examined. The potent tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) produced a two- to threefold increase in the activity of both the low- and high-affinity forms of cyclic AMP monophosphate phosphodiesterase activity 13 hr after application to female CD-1 mouse skin [17 nanomoles (nmol) in acetone]. At 13 hr after application, enzyme induction was dependent on the TPA dose applied (0.17-17 nmol). The magnitude of the enzyme induction correlated with the tumor-promoting activity of TPA and other phorbol esters: TPA > phorbol-12,13-didecanoate > phorbol-12,13-dibenzoate. The induction of the low-affinity phosphodiesterase could be blocked by prior injection of the microtubule poisons colchicine and vinblastine (250 nmol ip 3 hr before TPA). The low-affinity cyclic AMP phosphodiesterase activity of the epidermal component of mouse skin papillomas produced by two-stage tumorigenesis was three times that of the surrounding uninvolved epidermis. (21 refs)

79-3207 Differential Effect of Phorbol Esters on the Growth Rate of Normal Human Epithelial and Fibroblastic Cells (Meeting Abstract). (Eng) Lechner, J. F. (Pasadena Foundation for Medical Res., Pasadena, CA 91101); Kaighn, M. E. *In Vitro* 15(3): 225; 1979 (no refs)

79-3208 Modulation of Phagocytosis by Tumor Promoters and Epidermal Growth Factor in Normal and Transformed Macrophages (Meeting Abstract). (Eng) Laskin, D. L. (Medical Coll. Virginia, Richmond, VA 23298); Laskin, J. D.; Weinstein, I. B.; Munson, A. E.; Kessler, F. K.; Carchman, R. A. *Proc Am Assoc Cancer Res* 20: 16; 1979 (no refs)

79-3209 Selenium Effect on Initiation and Promotion of Tumors by Benzo(a)pyrene and 12-O-

Tetradecanoylphorbol (Meeting Abstract). (Eng) Wilt, S. (Dept. Pharmacology, Ohio State Univ. Coll. Medicine, Columbus, OH 43210); Pereira, M.; Couri, D. *Proc Am Assoc Cancer Res* 20: 21; 1979 (no refs)

79-3210 Studies on the Esophagus. II. Enhancement of [³H]Thymidine Incorporation in Rat Esophagus by *Bidens pilosa* (A Plant Eaten in South Africa) and by Croton Oil. (Eng) Mirvish, S. S. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NE 68105); Rose, E. F.; Sutherland, D. M. *Cancer Lett* 6 (3): 159-165; 1979.

The effects of *Bidens pilosa* (a plant eaten in South Africa) and croton oil in the diet on ³H-thymidine incorporation (TI) into DNA was studied in rat esophagus epithelium. The test materials were fed with a semisynthetic diet for 1 or 2 wk, and TI were measured 1 hr after ip injection of ³H-thymidine. TI was enhanced up to 2.3-fold in the esophagus of rats fed dried *B. pilosa* leaves (1:4 mixture of leaves:diet). A smaller enhancement of TI occurred with an ethanol extract of *B. pilosa* leaves, with a methanol-water fraction of the extract, and when *B. pilosa* was boiled under conditions resembling those used in cooking. The enhancement was not as great as that with unboiled material, indicating that some, but not all, of the active material survived the boiling. No enhancement occurred with raw spinach. TI was also enhanced significantly (1.08- to 1.21-fold) when croton oil was administered in the diet at levels of 2-6 g/kg. Since tumor promoters usually cause cell proliferation, these results suggest that *B. pilosa* consumption might be a promoting factor in the etiology of esophageal cancer in parts of South Africa that have a high incidence of this cancer. (17 refs)

79-3211 Effect of *Salmonella enteritidis* 11RX Infection on Two-Stage Skin Carcinogenesis in Mice. (Eng) Ashman, L. K. (Dept. Microbiology and Immunology, Univ. Adelaide, Adelaide 5000, South Australia, Australia); Kotlarski, I. *Aust J Exp Biol Med Sci* 56(6): 695-701; 1978.

The effects of *Salmonella enteritidis* 11RX infection on tumorigenesis in mice initiated with topical 7,12-dimethylbenz(a)anthracene (DMBA: 100 µg in 0.2 ml acetone) and promoted with repeated applications of croton oil (0.2 ml of 0.5% soln in acetone, 2x/wk 3-5 wk after initiation) were examined. DMBA/croton oil treatment produced small papillomas that gradually developed into lesions ranging from squamous papillomas to keratocanthomas and early invasive squamous cell carcinomas. Iv inoculation of LACA and (BALB/c x C57Bl/6J)F₁ mice with 10⁵ live 11RX 1 wk before DMBA application had no protective effect on tumorigenesis. However, a significant but transient protective effect was

achieved when mice were immunized with 11RX 4 wk before DMBA treatment and at intervals before and during promotion with croton oil. Relative to the LACA mice, (BALB/c x C57Bl/6J)F₁, BALB/c, C57Bl/6J, and CBA mice were more resistant to DMBA/croton oil skin carcinogenesis. (12 refs)

79-3212 Quantitative Cellular Studies on the Dynamics of Neoplastic Development in Tracheal Epithelium (Meeting Abstract). (Eng) Nettesheim, P. (NIEHS, Research Triangle Park, NC); Terzaghi, M. *Proc Am Assoc Cancer Res* 20: 44; 1979 (no refs)

79-3213 Contraceptive Enovid and Experimental Mammary Carcinogenesis in Hamster (Meeting Abstract). (Eng) Clayson, D. B. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105); Rustia, M.; Shubik, P.; Gingell, R. *Proc Am Assoc Cancer Res* 20: 32; 1979 (1 ref)

79-3214 Unscheduled DNA Synthesis in Rat Mammary Cells of Different Physiological States Exposed to Chemical Carcinogens In Vitro and In Vivo (Meeting Abstract). (Eng) Richards, J. E. (Cancer Res. Lab., Univ. California, Berkeley, CA 94720); Bowman, P. D. *In Vitro* 15(3): 173; 1979 (no refs)

79-3215 Isolation of a Transformed Keratinocyte Cell Line from a 7,12-Dimethylbenzanthracene (DMBA) Induced Rabbit Skin Tumor (Meeting Abstract). (Eng) Shoemaker, R. H. (Children's Hosp. Medical Center, Akron, OH 44308); Lake, R. S.; Ingel, H. J. *In Vitro* 15(3): 195; 1979 (no refs)

79-3216 Topographical Differences in Cell Cycle and Growth Fraction as Determinants of the Site of Origin of Mammary Carcinoma (Meeting Abstract). (Eng) Russo, I. H. (Michigan Cancer Foundation, Detroit, MI 48201). *Proc Am Assoc Cancer Res* 20: 230; 1979 (1 ref)

79-3217 Tumor Promotion by Damage Induced Epidermal Hyperplasia in the Skin of Mice (Meeting Abstract). (Eng) Argyris, T. S. (State Univ. New York, Upstate Medical Center, Syracuse, NY 13210). *Proc Am Assoc Cancer Res* 20: 4; 1979 (no refs)

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79-3218 Cell Cycle and Growth Fraction of Virgin and Parous Rat Mammary Gland as Determinants of Different Susceptibility to Carcinogenesis (Meeting Abstract). (Eng) Wilgus, G. (Experimental Pathology Lab., Michigan Cancer Foundation, Detroit, MI 48201); Russo, J. *Proc Am Assoc Cancer Res* 20: 238; 1979 (1 ref)

79-3219 Ultrastructural Features of Glioma Formation in Rats. (Rus) Abdullakhodzhaeva, M. S. (Dept. Pathoanatomy, Medical Inst., Tashent, USSR); Ibragimov, T. K. *Biull Eksp Biol Med* 87(4): 371-375; 1979.

9,10-Dimethyl-1,2-benz(a)anthracene (DMBA) pellets were implanted intracerebrally in rats, and changes that occurred in the astrocytes during glioma formation were studied ultrastructurally. In one group of rats, the DMBA pellet was implanted 45 days after the animals underwent pinealectomy. Brain specimens were obtained on days 1, 5, 15, 30, 45, 60, 90, 120 and 150 postimplantation (PI). On PI days 15-30, the pinealectomized rats showed a predominance of astrocytes with an oval nucleus, an enlarged perinuclear space, and a granular endoplasmic reticulum (collectively termed intracellular hyperplasia). By PI day 45, however, the number of these astrocytes was reduced, and between days 60 and 90, astrocytes with an osmiophobic cytoplasm predominated. Rats subjected to intracerebral implantation of DMBA alone showed only intracellular hyperplasia on PI days 15-30. These findings indicate that pinealectomy potentiates the malignant transformation of glial cells. (15 refs)

79-3220 The Effect of Pinealectomy on Induction of Mammary Tumors (MT) by 7,12-Dimethylbenzanthracene (DMBA) in Female Rats Maintained in Short Photoperiod (Meeting Abstract). (Eng) Cutty, G. B. (Roswell Park Memorial Inst., Buffalo, NY 14263); Sinha, D. K. *Proc Am Assoc Cancer Res* 20: 210; 1979 (no refs)

79-3221 Metabolites of 7,12-Dimethylbenz[a]anthracene and the Integrity of the Metabolite-generating System (Meeting Abstract). (Eng) Bigger, C. A. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701); Tomaszewski, J. E.; Dipple, A. *Proc Am Assoc Cancer Res* 20: 232; 1979 (1 ref)

79-3222 Chemical Carcinogen-induced Malignant Transformation of Epithelial Cells in Organ

Culture of the Whole Mammary Gland (Meeting Abstract). (Eng) Telang, N. T. (Tumor Biology Lab., Sch. Life Sciences, Univ. Nebraska, Lincoln, NE 68588); Kundu, A. B.; Iyer, A. P.; Banerjee, M. R. *In Vitro* 15(3): 174; 1979 (no refs)

79-3223 Effect of Thymosin and Adrenalectomy on Development of Chemically Induced Tumors. (Ukr) Chebotarev, V. F. (Res. Inst. Endocrinology and Metabolism, Kiev, USSR); Grinevich, Iu. A. *Dopov Akad Nauk Ukr RSR [B]* (12): 1121-1123; 1978.

An attempt was made to determine whether thymosin (TM) can enhance the immunocompetence of adrenalectomized animals against the development of chemically induced tumors. Random-bred rats were injected with 7,12-dimethylbenz(a)anthracene (DMBA: 3 doses of 2 mg, iv, at 3-day intervals). Ten days after the termination of DMBA administration, the rats were adrenalectomized and/or given injections of TM (10 mg, iv, at 10-day intervals for 3 mo). Adrenalectomy + TM significantly decreased the number of animals with mammary gland tumors. The max difference was recorded 22 wk after the termination of DMBA administration, when tumors were detected in 78.8% of the animals exposed to DMBA alone (controls), 58.3% of the animals DMBA + adrenalectomy, 51.7% of the rats treated with DMBA + TM, and 25.0% of the rats treated with DMBA + adrenalectomy + TM. (5 refs)

79-3224 Enhancement of Mammary Tumorigenesis in Rats by Vitamin E Deficiency (Meeting Abstract). (Eng) Lee, C. (Northwestern Univ. Medical Sch., Chicago, IL 60611); Chen, C. *Proc Am Assoc Cancer Res* 20: 132; 1979 (no refs)

79-3225 Null Effect of BHA and α -Tocopherol on 7,12-Dimethylbenz (α) anthracene- induced Mammary Tumors in Rats Fed Different Levels and Types of Dietary Fat (Meeting Abstract). (Eng) King, M. M. (Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104); Otto, P. *Proc Am Assoc Cancer Res* 20: 227; 1979 (no refs)

79-3226 The Inhibition of Rat Mammary Tumors by Dietary Retinyl Acetate at Various Times During and After Treatment with a Carcinogen (Meeting Abstract). (Eng) McCormick, D. L. (New York Univ. Inst. Environmental Medicine, New York, NY 10016); Burns, F. J.; Albert, R. E. *Proc Am Assoc Cancer Res* 20: 99; 1979 (no refs)

- 79-3227 Effect of Antiprolactin Drugs on Growth and Regression of DMBA-induced Tumors in Rats (Meeting Abstract). (Eng) Formelli, F. (Natl. Tumor Inst., Milan, Italy); Di Marco, A.; Di Salle, E.; Zaccheo, T.; Pacciarini, M. A. *Proc Am Assoc Cancer Res* 20: 30; 1979 (no refs)
- 79-3228 Further Observations of Anti-Mammary Carcinogenic Activity of Estriol (Meeting Abstract). (Eng) Lemon, H. M. (Univ. Nebraska Medical Center, Omaha, NE 68105); Thompson, J.; Haven, G. *Proc Am Assoc Cancer Res* 20: 136; 1979 (4 refs)
- 79-3229 Modification of 7,12-Dimethylbenz[a]-anthracene (DMBA) Tumorigenesis In Vivo by Low-Dose X-Radiation: Temporal Considerations (Meeting Abstract). (Eng) Lurie, A. G. (Univ. Connecticut Health Center, Farmington, CT 06032). *Proc Am Assoc Cancer Res* 20: 12; 1979 (no refs)
- 79-3230 Prevention of Transformation of Mammary Gland by Retinoid in Whole Organ Culture (Meeting Abstract). (Eng) Dickens, M. S. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111); Sorof, S. *Proc Am Assoc Cancer Res* 20: 71; 1979 (no refs)
- 79-3231 7,12-Dimethylbenz[a]anthracene (DMBA)-induced Malignant Transformation in Organ Culture of the Whole Mammary Gland (Meeting Abstract). (Eng) Telang, N. T. (Tumor Biology Lab., Sch. Life Sciences, Univ. Nebraska, Lincoln, NB 68588); Kundu, A. B.; Iyer, A. P.; Banerjee, M. R. *Proc Am Assoc Cancer Res* 20: 153; 1979 (no refs)
- 79-3232 7,12-Dimethylbenz(a)anthracene-3,4-dihydrodiol (DMBA 3,4-Diol) Is an Intermediate in the Binding of DMBA To DNA (Meeting Abstract). (Eng) Jeffrey, A. M. (Inst. Cancer Res., Columbia Univ., New York, NY 10032); Weinstein, I. B.; Harvey, R. G. *Proc Am Assoc Cancer Res* 20: 131; 1979 (no refs)
- 79-3233 Identification of Four *trans*-3,4-Dihydrodiols as Metabolites of 7,12-dimethylbenz[a]anthracene and Their DNA Binding and Mutagenic Activities (Meeting Abstract). (Eng) Chou, M. W. (Uniformed Services Univ. Health Sciences, Bethesda, MD 20014); Yang, S. K. *Proc Am Assoc Cancer Res* 20: 33; 1979 (no refs)
- 79-3234 ATP-mediated Binding of Hydroxymethyl Aromatic Hydrocarbons to DNA (Meeting Abstract). (Eng) Rogan, E. G. (Eppley Inst., Univ. Nebraska Medical Center, Omaha, NE 68105); Roth, R. W.; Cavalieri, E. L. *Proc Am Assoc Cancer Res* 20: 54; 1979 (no refs)
- 79-3235 Biological Activities of Dihydrodiols Derived from Two Polycyclic Hydrocarbons in Rodent Test Systems. (Eng) Chouroulinkov, I. (Institut de Recherches Scientifiques sur le Cancer, Boite Postale No. 8, 94800 Villejuif, France); Gentil, A.; Tierney, B.; Grover, P. L.; Sims, P. *Br J Cancer* 39(4): 376-382; 1979.
- The carcinogenic and hyperplastic effects of dihydrodiols derived from 7-methylbenz(a)anthracene (MBA) or benzo(a)pyrene (BP) were tested by topical application to female CDI mice and female Wistar rats. The 3,4-dihydrodiol derived from MBA was more effective in producing skin tumors in mice than MBA, and MBA was more effective in this respect than the related 1,2-, 5,6-, and 8,9-dihydrodiols. The 7,8-dihydrodiol of BP was less effective than BP in the induction of hyperplasia and the suppression of sebaceous glands in mouse skin. The 7,8-dihydrodiol was also very much less active than BP in the induction of sarcomas in rats. The data indicate that the 7,8-dihydrodiol of BP may be more effective as an initiating agent than as a complete carcinogen. Of the systems tested, only the mouse skin initiation-promotion model gave results that are in broad agreement with data that have identified the 7,8-dihydrodiol of BP and the 3,4-dihydrodiol of MBA as important in the metabolic activation of the carcinogenic parent hydrocarbons. (27 refs)
- 79-3236 Potent Tumor-initiating Activity of the 3,4-Dihydrodiol of 7,12-Dimethylbenz(a)anthracene in Mouse Skin. (Eng) Slaga, T. J. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN 37830); Gleason, G. L.; DiGiovanni, J.; Sukumaran, K. B.; Harvey, R. G. *Cancer Res* 39(6, part 1): 1934-1936; 1979.
- The abilities of the racemic *trans*-3,4-, 5,6-, and 8,9-dihydrodiols of 7,12-dimethylbenz(a)anthracene (DMBA) to initiate skin tumors in mice were determined with the use of a two-stage system of tumorigenesis. Groups of 30 female Sencar mice received a single topical application of the test compound, followed 1 wk later by twice-weekly applications of 2 μ g 12-O-tetradecanoylphorbol-13-acetate.

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The incidence of papillomas was recorded weekly, and they were removed at random for histological verification. DMBA and DMBA dihydrodiols were applied topically at a dose of either 10 or 100 nanomoles in acetone. The 7,12-dimethylbenz(a)anthracene trans-3,4-dihydrodiol was much more active as a tumor initiator than the parent hydrocarbon, producing 15.20 and 22.84 tumors/mouse at 15 wk at the two respective doses, vs 4.80 and 10.20 tumors/mouse produced by DMBA. The trans-5,6- and 8,9-dihydrodiols were essentially inactive as skin tumor initiators. The results suggest that the 3,4-dihydrodiol of DMBA is a proximal carcinogen and that the bay region diol-epoxide may be the ultimate carcinogenic form of DMBA. (42 refs)

- 79-3237 Effects of Diet and MER on DMBA-induced Mammary Carcinoma (Meeting Abstract). (Eng) Kollmorgen, G. M. (Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104); Sansing, W. A.; Lehman, A. A.; Longley, R.; Fischer, G.; McCay, P. B. *Proc Am Assoc Cancer Res* 20: 41; 1979 (no refs)

- 79-3238 Induction of Aryl Hydrocarbon Hydroxylase Activity in Lung Cells and Tissues of Syrian Hamsters (Meeting Abstract). (Eng) Mitchell, C. E. (Inhalation Toxicology Res. Inst., Albuquerque, NM 87115); Pflieger, R. C.; Hobbs, C. H. *Proc Am Assoc Cancer Res* 20: 25; 1979 (no refs)

- 79-3239 DNA Damage In Vivo/Alkaline Elution Assay: Strategies for Minimizing False Negative Results (Meeting Abstract). (Eng) Parodi, S. (Dept. Oncology, Univ. Genoa, Genoa, Italy); Taningher, M.; Pala, M.; Cavanna, M.; Brambilla, G. *Proc Am Assoc Cancer Res* 20: 31; 1979 (2 refs)

- 79-3240 Metabolism of Polycyclic Hydrocarbons by Rat Embryo Cell Cultures (Meeting Abstract). (Eng) Baird, W. M. (Wistar Inst., Philadelphia, PA 19104); Diamond, L. *Proc Am Assoc Cancer Res* 20: 281; 1979 (2 refs)

- 79-3241 The Effect of Dietary Lipids on the Activity of Benzo(a)pyrene Hydroxylase in Lung and Liver (Meeting Abstract). (Eng) Kellermann, G. H. (Wisconsin Clinical Cancer Center, Madison, WI); Elson, C. E. *Proc Am Assoc Cancer Res* 20: 83; 1979 (no refs)

- 79-3242 Long Term Organ Culture of Human Endometrium (Meeting Abstract). (Eng) Adamec, T. A. (Dept. Pathology, Univ. North Carolina, Chapel Hill, NC 27514); Kaufman, D. G.; Mass, M. J.; Genta, V. M.; Dorman, B. H.; Melin, S. A.; Powell, J. *Proc Am Assoc Cancer Res* 20: 252; 1979 (no refs)

- 79-3243 Metabolism and Conjugation of Benzo(a)pyrene By Human Pulmonary Alveolar Macrophages (Meeting Abstract). (Eng) Marshall, M. V. (Univ. Texas System Cancer Center, Houston, TX 77030); McLemore, T. L.; Arnott, M. S.; Martin, R. R.; Griffin, A. C. *Proc Am Assoc Cancer Res* 20: 183; 1979 (no refs)

- 79-3244 Planimetric Determination of Benzo(a)pyrene. (Rus) Alekseev, I. B. (Design Inst. Aluminum, Magnesium and Electrode Industry, Leningrad, USSR); Khomenko, V. N. *Gig Sanit* (2): 56-58; 1979.

Advantages of the planimetric method of determining benzo(a)pyrene (BP) levels in wastes generated by the aluminum industry are discussed. The technique was found to be more accurate than chromatography. (no refs)

- 79-3245 Correlation of In Vitro Growth Properties and Tumorigenicity of Syrian Hamster Cell Lines. (Eng) Barrett, J. C. (Natl. Inst. Environmental Health Sciences, NIH, PO Box 12233, Research Triangle Park, NC 27709); Crawford, B. D.; Mixter, L. O.; Schechtman, L. M.; Ts'o, P. O.; Pollack, R. *Cancer Res* 39(5): 1504-1510; 1979.

Several in vitro phenotypic characteristics frequently associated with neoplastic cells were examined in a series of spontaneously transformed and benzo(a)pyrene (BP)-transformed Syrian hamster clonal cell lines that differed in their degree of tumorigenicity. Some chemically transformed cell lines were highly tumorigenic in nonimmunosuppressed newborn hamsters, requiring ≤ 10 cells to produce tumors, others required 10^4 - 10^5 cells. Of five spontaneously established cell lines studied, only one became tumorigenic. After 34 in vitro passages, it was capable of producing tumors following inoculation of newborn hamsters with 2×10^6 cells. Anchorage-independent cloning efficiency, elevated fibrinolytic activity, reduced serum requirement for growth, increased cloning efficiency, and loss of intracellular actin organization were correlated with tumorigenicity, both qualitatively and quantitatively, by nonparametric statistical analyses. These results suggest that the factors controlling tumorigenicity in this test system are the growth properties of the cells rather than their antigenicity in the host. (44 refs)

79-3246 Tracheal Ring Cultures Used for the Evaluation of Oncogenic Activity of Polycyclic Aromatic Hydrocarbons (Meeting Abstract). (Eng) Beales, N. N. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439); Rahman, Y. E.; Peraino, C. *In Vitro* 15(3): 219; 1979 (no refs)

79-3247 Preferential Binding of Benzo(a)pyrene into Nuclear Matrix Fraction. (Eng) Hemminki, K. (Dept. Industrial Hygiene and Toxicology, Inst. Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland); Vainio, H. *Cancer Lett* 6(3): 167-173; 1979.

The distribution of covalently bound ^3H -benzo(a)pyrene (^3H -BP) was investigated in subfractions of rat lung and liver nuclei in vivo and in vitro. For the in vivo experiments, 75 μCi ^3H -BP was injected sc into 2- to 3-wk-old rats, and the animals were killed 2.5 hr later. For in vitro labeling, rat lung tissue was incubated with medium containing 75 μCi ^3H -BP for 90 min. Lung and liver nuclei were fractionated into a matrix and a chromatin fraction. Both in vivo and in vitro, the DNA, RNA, and protein of the matrix fraction were labeled more extensively (3- to 10-fold) than the DNA, RNA, and protein of the chromatin fraction. The specific radioactivity of protein was highest and that of DNA lowest. In vivo, the rates of ^3H -BP incorporation were higher into liver RNA and DNA than into lung RNA and DNA. It is suggested that BP molecules, which are activated at the nuclear envelope by the benzo(a)pyrene hydroxylase system, may react preferentially with nearby targets, such as the nuclear matrix. (21 refs)

79-3248 Binding of ^3H -Labeled Benzo(a)pyrene (^3H -BP) and/or Its Metabolites to Cellular Macromolecules in the Rat Pancreas (Meeting Abstract). (Eng) Iqbal, Z. M. (Sch. Public Health, Univ. Illinois at the Medical Center, Chicago, IL 60680); Whalley, C. E.; Nguyen, L.; Epstein, S. S. *Proc Am Assoc Cancer Res* 20: 173; 1979 (2 refs)

79-3249 Comparative Metabolism of Benzo(a)pyrene in Organ and Cell Cultures Derived from Rat Tracheas. (Eng) Cohen, G. M. (Dept. Biochemistry, Univ. Surrey, Guildford GU25XH, Surrey, England); Marchok, A. C.; Nettesheim, P.; Steele, V. E.; Nelson, F.; Huang, S.; Selkirk, J. K. *Cancer Res* 39(6, part 1): 1980-1984; 1979.

The metabolism of benzo(a)pyrene (BP) to oxidative (primarily ethyl acetate-soluble) and conjugated (primarily water-soluble) metabolites in cells from Fischer 344 rat tracheas at different levels of tissue and cell organization was studied. Primary cultures from tracheal explants and a

nontumorigenic tracheal epithelial cell line (2Cl), derived from 12-O-tetradecanoylphorbol-13-acetate-exposed tracheal explants, metabolized BP to qualitatively similar organic solvent-soluble and water-soluble metabolites. Similar metabolites were formed by short-term organ cultures of tracheas. In contrast, a tumorigenic tracheal epithelial cell line (1000 W) did not metabolize BP to any significant extent. The major metabolites formed by these different tracheal systems were 9,10-dihydro-9,10-dihydroxybenzo(a)pyrene with smaller amounts, mainly as their glucuronide conjugates, of 7,8-dihydro-7,8-dihydroxybenzo(a)pyrene, 3-hydroxybenzo(a)pyrene, and 9-hydroxybenzo(a)pyrene. The higher formation of dihydrodiols, precursors of highly reactive diol-epoxides, relative to monohydroxybenzo(a)pyrenes, together with low rates of detoxification of the dihydrodiols by conjugation, may in part explain the high susceptibility of the trachea to carcinogenesis. This study indicates that nontumorigenic respiratory epithelium at different levels of organization possesses similar activating and detoxifying enzymes. Since it is the balance of these oxidative and conjugating enzymes that determines how much of a metabolite is available for reaction with critical cellular macromolecules, these enzymes appear to be suitable for metabolic studies of factors affecting initiation and transformation of respiratory tract epithelium. (33 refs)

79-3250 Isolation of Stable Mutagenic Photodecomposition Products of Benzo(a)pyrene by Thin-Layer Chromatography. (Eng) Issaq, H. J. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701); Andrews, A. W.; Janini, G. M.; Barr, E. W. *J Liquid Chromatography* 2(3): 319-325; 1979.

The effects of UV radiation on the stability of benzo(a)pyrene (BP) in soln and on thin-layer chromatography (TLC) plates were examined, and stable photodecomposition products were isolated and tested for mutagenic activity. When BP solns were spotted on silica gel, alumina, cellulose, and reversed-phase TLC plates and dried under nitrogen, followed by development and elution, no loss to decomposition was observed. When the solns were spotted on the plates and allowed to dry in a stream of air, several developed spots were observed. Similar results were obtained when plates spotted with BP were exposed to sunlight. Two of these products were eluted off the plate and were characterized as the 1,6- and 3,6-BP quinones. Solns of BP (334 $\mu\text{g}/\text{ml}$) were streaked on silica gel plates, allowed to dry, exposed to sunlight or UV radiation for 1 hr, and then developed. Regions from the developed plates were eluted and tested for mutagenicity. BP was mutagenic for the base-pair-substitution *Salmonella typhimurium* strain TA100 and the frameshift TA1538, with and without metabolic activation. These data suggest that the photodecomposition products may be more highly mutagenic than the parent compound. The two identified

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photodecomposition products do not account for the high mutagenicity observed. Thus, characterization of the stable photodecomposition products of polycyclic aromatic hydrocarbons on coated TLC plates may lead to the identification of previously unappreciated environmental carcinogens. (16 refs)

79-3251 Differences in Benzo(a)pyrene-4,5-epoxide Hydrase Activities of Intact Hamster Hepatocytes, Embryo Cells, and Homogenates of These Cells (Meeting Abstract). (Eng) Raineri, R. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Poiley, J. A.; Pienta, R. J. *Proc Am Assoc Cancer Res* 20: 232; 1979 (no refs)

79-3252 Metabolism and Excretion of Benzo(a)pyrene 4,5-Oxide by the Isolated Perfused Rat Liver. (Eng) Smith, B. R. (Lab. Pharmacology, Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Triangle Research Park, NC 27709); Bend, J. R. *Cancer Res* 39(6, part 1): 2051-2056; 1979.

Benzo(a)pyrene 4,5-oxide was metabolized in isolated perfused Sprague-Dawley rat liver by epoxide hydrase and glutathione S-transferases to the corresponding dihydrodiol and to thioether conjugates (derivates of glutathione), respectively. Epoxide hydrase was more important relative to the glutathione S-transferases in the biotransformation of this oxide by the intact organ than was indicated by the results of earlier studies with subcellular fractions. The dihydrodiol was rapidly released into the circulation or conjugated with glucuronic acid; sulfuric acid esters were not found. All conjugated metabolites were excreted rapidly in the bile, but they were also released into the circulation. The enzymatic systems responsible for the metabolism and excretion of benzo(a)pyrene 4,5-oxide remained viable in the perfused liver for at least 60 min. The toxicological significance of the release of polycyclic aromatic hydrocarbon metabolites from the liver into the vascular circulation and the possible significance of uridine diphosphate:glucuronyltransferase activity in preventing chemically induced carcinogenesis are discussed. (46 refs)

79-3253 Metabolism of Benzo(a)pyrene (BP) by Human Placental Microsomes (Meeting Abstract). (Eng) Gurtoo, H. L. (Dept. Experimental Therapeutics, Roswell Park Memorial Inst., Buffalo, NY 14263); LeBoeuf, R.; Doctor, G. *Proc Am Assoc Cancer Res* 20: 86; 1979 (no refs)

79-3254 Malignant Transformation of Cultured Rat Hepatocytes by (+) and (-) *trans* 7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrene (Meeting Abstract). (Eng) Schaeffer, W. I. (Dept. Microbiology, Univ. Vermont, Coll. Medicine, Burlington, VT 05405); Heintz, N.; Bresnick, E. *Proc Am Assoc Cancer Res* 20:38; 1979 (no refs)

79-3255 Metabolism of Benzo(a)pyrene and (-)*trans*-7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrene by Human Blood Monocytes and Lymphocytes (Meeting Abstract). (Eng) Okano, P. (NCI, NIH, Bethesda, MD 20014); Robinson, R. C.; Gelboin, H. V. *Proc Am Assoc Cancer Res* 20: 181; 1979 (no refs)

79-3256 Prostaglandin Synthetase Dependent Metabolism of 7,8-Dihydroxy-7,8-dihydrobenzo[a]pyrene (Meeting Abstract). (Eng) Marnett, L. J. (Wayne State Univ., Detroit, MI 48202); Johnson, J. T.; Reed, G. A. *Proc Am Assoc Cancer Res* 20: 60; 1979 (no refs)

79-3257 Induction of Mutations and Transformation of C3H/10T1/2 Mouse Fibroblasts by Chemical Carcinogens (Meeting Abstract). (Eng) Landolph, J. R. (Univ. Southern California Cancer Center, Los Angeles, CA 90033); Fuchs, R. P. *Proc Am Assoc Cancer Res* 20: 61; 1979 (1 ref)

79-3258 DNA-Benzo[a]pyrene Adducts Formed in a *Salmonella typhimurium* Mutagenesis Assay and a Mouse 10 T1/2 Cell Line (Meeting Abstract). (Eng) Santella, R. M. (Columbia Univ. Coll. Physicians and Surgeons, New York, NY 10032); Brown, H. S.; Jeffrey, A. M.; Grunberger, D.; Weinstein, I. B. *Proc Am Assoc Cancer Res* 20: 79; 1979 (no refs)

79-3259 Antibodies Specific for DNA Modified with a Diol Oxide of Benzo(a)Pyrene (BP) (Meeting Abstract). (Eng) Poirier, M. C. (NCI, Bethesda, MD 20014); Grunberger, D.; Weinstein, I. B.; Yuspa, S. H. *Proc Am Assoc Cancer Res* 20: 102; 1979 (1 ref)

79-3260 Human Alveolar Macrophages Mediate Mutations and Sister Chromatid Exchanges in Chinese Hamster Cells Exposed to Chemical Carcinogens (Meeting Abstract). (Eng) Hsu, I. C. (Human Tissue

Studies Section, Lab. Experimental Pathology, NCI, Bethesda, MD 20014); Yeager, H.; Yamaguchi, M.; Myers, G. *Proc Am Assoc Cancer Res* 20: 92; 1979 (no refs)

79-3261 Single-Strand Specific Endonuclease Sensitive Sites in Newly Synthesized DNA as Evidence for Gapped DNA Synthesis in Cells Treated with Dihydrodiol Epoxide Derivatives of Benzo[a]pyrene (Meeting Abstract). (Eng) Bowden, G. T. (Lab. Experimental Pathology, NCI, Bethesda, MD 20014); Yuspa, S. H. *Proc Am Assoc Cancer Res* 20: 88; 1979 (no refs)

79-3262 Inhibition of DNA Synthesis In Vitro by the Binding to DNA of Benzo(a)Pyrene Metabolite Diol-Epoxide 1 (Meeting Abstract). (Eng) Mizusawa, H. (NCI-NIH, Bethesda, MD 20014); Kakefuda, T. *Proc Am Assoc Cancer Res* 20: 80; 1979 (no refs)

79-3263 Differences in Detoxification Pathways for Benzo(a)pyrene and Benzo(e)pyrene in Rodent Fibroblasts and Epithelial Cells (Meeting Abstract). (Eng) MacLeod, M. C. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Cohen, G. M.; Selkirk, J. K. *Proc Am Assoc Cancer Res* 20: 268; 1979 (no refs)

79-3264 Carcinogenicity of Benzo[e]Pyrene (B[e]P) Compared to Benzo[a]Pyrene (B[a]P) (Meeting Abstract). (Eng) Levin, W. (Dept. Biochemistry and Drug Metabolism, Hoffmann-La Roche Inc., Nutley, NJ 07110); Wood, A. W.; Buening, M. K.; Kumar, S.; Lehr, R. E.; Jerina, D. M.; Conney, A. H. *Proc Am Assoc Cancer Res* 20: 222; 1979 (no refs)

79-3265 Mutagenicity of 43 Structurally Related Heterocyclic Compounds and Its Relationship to Their Carcinogenicity. (Eng) Glatt, H. R. (Section Biochemical Pharmacology, Inst. Pharmacology, Univ. Mainz, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany); Schwind, H.; Zajdela, F.; Croisy, A.; Jacquignon, P. C.; Oesch, F. *Mutat Res* 66(4): 307-328; 1979.

The mutagenicity and carcinogenicity of 43 structurally related heteropolycyclic compounds were evaluated, and the results were compared. Mutagenicity was determined in the Ames assay with *Salmonella typhimurium* strains TA1535, TA1537, TA98 and TA100 in the presence and absence of a liver 10,000-g supernatant from rats treated with Aroclor 1254. Carcinogenicity was tested by sc injection

of the compounds into XVIIInc/Z mice. Eighteen test compounds showed carcinogenic activity, some strongly, others only weakly. Of these, 17 were also mutagenic: 1 weak carcinogen did not revert the *Salmonella* strains. There was no quantitative correlation between degree of mutagenicity and carcinogenicity. Of the 25 substances that did not produce tumors, 13 showed mutagenicity (12 in the presence, 2 in the absence of the liver homogenate). The mutagenic effects of these compounds were quantitatively similar to those of the compounds that produced tumors. The most sensitive strain of *S. typhimurium* was TA100, detecting all 30 mutagens. TA98 was mutated by 25 compounds, TA1537 by 16 compounds. No mutagenic effects were seen with TA1535. Possible reasons for the high percentage of apparently false-positives in the Ames assay and the lack of a quantitative correlation between mutagenic and carcinogenic potency are given. The complexity of the metabolism of these heterocyclic compounds may lead to critical differences in metabolism in mouse sc tissue in vivo and in liver homogenates from Aroclor-treated rats. (16 refs)

79-3266 The Metabolism of Benzo(a)pyrene (BP) by Cytochrome P450 in Transformable and Non-transformable C3H Mouse Fibroblasts (Meeting Abstract). (Eng) Gehly, E. B. (Los Angeles County-Univ. Southern California Comprehensive Cancer Center, Los Angeles, CA 90033); Fahl, W. E.; Jefcoate, C. R. *Proc Am Assoc Cancer Res* 20: 70; 1979 (no refs)

79-3267 Effects of Bile Acids on Microsomal Metabolism of, and Mutagenesis by Benzo(a)pyrene (Meeting Abstract). (Eng) Unger, J. (Dept. Biochemistry, New York Univ. Dental Center, New York, NY 10010); Guttentplan, J. B. *Proc Am Assoc Cancer Res* 20: 132; 1979 (no refs)

79-3268 Lung Cancer Model Systems in Inbred Strains of Mice (Meeting Abstract). (Eng) Henry, C. J. (Dept. Experimental and Biochemical Oncology, Microbiological Assoc., Bethesda, MD 20016); Billups, L. H.; Avery, M. D.; Dansie, D. R.; Lopez, A.; Breth, L. A.; Rude, T. R.; Kouri, R. E. *Proc Am Assoc Cancer Res* 20: 242; 1979 (no refs)

79-3269 Enhancement of Viral Transformation by Carcinogenic Polycyclic Hydrocarbons: Modulation by Benz(a)anthracene or 7,8 Benzoflavone (Meeting Abstract). (Eng) Casto, B. C. (Northrup Services, Inc., P.O. Box 12313, Research Triangle Park, NC 27709); DiPaolo, J. A. *Proc Am Assoc Cancer Res* 20: 283; 1979 (no refs)

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- 79-3270** Induction of Neoplasms after Implantation of Hamster Trachea Exposed In Vitro to 3-Methylcholanthrene on Ferric Oxide (Meeting Abstract). (Eng) Mossman, B. T. (Univ. Vermont Coll. Medicine, Burlington, VT 05405); Craighead, J. E. *Proc Am Assoc Cancer Res* 20: 228; 1979 (no refs)
- 79-3271** 3-Methylcholanthrene Binding to Lung and Liver DNA of Various Strains of Mice (Meeting Abstract). (Eng) Eastman, A. (Dept. Biochemistry, Univ. Vermont Coll. Medicine, Burlington, VT 05405). *Proc Am Assoc Cancer Res* 20: 40; 1979 (no refs)
- 79-3272** The Characterization of "Oat Cell Like" Carcinoma in the Lung of Rodents (Meeting Abstract). (Eng) Blair, W. H. (Mercy Hosp. and Medical Center, Chicago, IL 60616). *Proc Am Assoc Cancer Res* 20: 166; 1979 (no refs)
- 79-3273** Effects of Phenobarbital and Methylcholanthrene on Hepatic Mixed-Function-Oxidase Activities in Hamsters. (Eng) Tucker, A. N. (Dept. Pharmacology, Medical Coll. Virginia, Health Sciences Div. Virginia Commonwealth Univ., Richmond, VA 23298); Tang, T. *J Environ Pathol Toxicol* 2(3): 613-623; 1979.
- The relationship between the effects of the inducers phenobarbital (PB) and 3-methylcholanthrene (MC) on hamster liver microsomal mixed-function-oxidase activities and the abilities of the microsomes to activate known carcinogens was studied. Both inducers increased the amount of cytochrome P-450 in the microsomes, aminopyrine demethylase activity, and biphenyl 4-hydroxylase activity when given for 8 days. The ability of liver homogenates from treated animals to activate compounds to mutagens was tested using the Salmonella/microsome test. Neither inducer appreciably altered the mutagenicity of 2-acetylaminofluorene, ben-zidine, benzo(a)pyrene, aflatoxin B₁, or sterigmatocystin. The mutagenicity of MC was increased when homogenates from MC-treated hamsters were used as a source of activating enzymes, and this mutagenicity could be correlated with increased biphenyl 2-hydroxylase activity. (25 refs)
- 79-3274** Measurement of the Transformation Mutation Ratio in BALB/3T3 Cells (Meeting Abstract). (Eng) Lang, D. R. (Univ. Cincinnati Coll. Medicine, Cincinnati, OH 45267). *In Vitro* 15(3): 223-224; 1979 (1 ref)
- 79-3275** Tumor-initiating Activities on Mouse Skin of Diol Metabolites of 7,12-Dimethylbenz[a]-anthracene (DMBA) and 3-Methylcholanthrene (3-MC) (Meeting Abstract). (Eng) Grover, P. L. (Chester Beatty Res. Inst., Fulham Road, London SW3 6JB, England); Chouroulinkov, I.; Tierney, B.; Sims, P. *Proc Am Assoc Cancer Res* 20: 66; 1979 (no refs)
- 79-3276** Pretransformation Membrane Changes Associated with Chemical Carcinogens and Aqueous Extracts of Fossil Fuel-generated Respirable Particulates (Meeting Abstract). (Eng) Facklam, T. J. (Battelle Columbus Lab., Columbus, OH 43201); Crowley, J. P.; Dennis, A. J. *Proc Am Assoc Cancer Res* 20: 205; 1979 (no refs)
- 79-3277** Sustained Release of Aromatic Hydrocarbon Carcinogens at Specific Endobronchial Sites (Meeting Abstract). (Eng) Benfield, J. R. (City of Hope Medical Center, Duarte, CA 91010); Shors, E. C.; Fu, P.; Cohen, A. *Proc Am Assoc Cancer Res* 20: 235; 1979 (no refs)
- 79-3278** Comparative Epidermal Metabolism in Strains of Mice with Differing Sensitivity to Skin Tumorigenesis by DMBA (Meeting Abstract). (Eng) DiGiovanni, J. (Dept. Pharmacology, Univ. Washington, Seattle, WA 98195); Slaga, T. J.; Juchau, M. R. *Proc Am Assoc Cancer Res* 20: 134; 1979 (no refs)
- 79-3279** Lymphocyte Aryl Hydrocarbon Hydroxylase (AHH) Inducibility in Acute Leukemia of Childhood (AL) (Meeting Abstract). (Eng) Blumer, J. L. (Dept. Pediatrics, Case Western Reserve Univ., Cleveland, OH 44106); Dunn, R.; Gross, S. *Proc Am Assoc Cancer Res* 20: 310; 1979 (no refs)
- 79-3280** Metabolic Formation of the Highly Tumorigenic 3,4-Dihydrodiol From Dibenzo[a,h]anthracene (Meeting Abstract). (Eng) Nordqvist, M. (NIH, Bethesda, MD 20014); Ryan, D. E.; Thomas, P. E.; Levin, W.; Conney, A. H.; Jerina, D. M. *Proc Am Assoc Cancer Res* 20: 94; 1979 (no refs)
- 79-3281** Mutagenicity of Phenanthrene and Phenanthrene K-Region Derivatives. (Eng) Bucker, M. (Section Biochemical Pharmacology, Inst. Pharmacology, Univ. Mainz, Obere Zahlbacher Strasse 67, D-

6500 Mainz, W. Germany); Glatt, H. R.; Platt, K. L.; Avnir, D.; Ittah, Y.; Blum, J.; Oesch, F. *Mutat Res* 66(4): 337-348; 1979.

Phenanthrene (PAT) and nine of its K-region derivatives were tested for mutagenicity in the *Salmonella typhimurium* reversion assay using the histidine-dependent strains TA1535, TA1537, TA1538, TA98, and TA100 and in the rec assay using *Bacillus subtilis* strains H17 (rec+) and M45 (rec-). In the reversion assay, the strongest mutagenic effects were observed with phenanthrene 9,10-oxide (I), 9-hydroxyphenanthrene (II), and N-benzylphenanthrene 9,10-imine (III). Interestingly, the mutagenic potency of the arene imine was similar to that of the corresponding arene oxide. The mutagenic effects of these derivatives were much weaker than that of the positive control, benzo(a)pyrene 4,5-oxide (IV). Even weaker mutagenicity was found with cis- (V) and trans-9,10-dihydroxy-9,10-dihydrophenanthrene (VI). The other derivatives were inactive in this test. However, 9,10-dihydroxyphenanthrene and 9,10-phenanthrenequinone were more toxic to *B. subtilis* strain M45 strain than to H17. This was also true for compounds I and II, but not with the other test compounds that reverted (compounds III-VI) or did not revert (PAT, 9,10-bis-(p-chlorophenyl)phenanthrene 9,10-oxide, 9,10-diacetoxyphenanthrene) the *Salmonella* tester strains. Although the K region is a main site of metabolism and although all potential K-region metabolites were mutagenic, PAT did not show a mutagenic effect in the presence of mouse liver microsomes and an NADPH-generating system under standard conditions. However, when epoxide hydratase was inhibited, PAT was activated to a mutagen that reverted *his*- *S. typhimurium*. Thus, demonstration of the mutagenicity of metabolites plus the knowledge that a major metabolic route proceeds via these metabolites do not automatically imply a mutagenic hazard of the parent compound, because the metabolites in question may not accumulate in sufficient quantities and, therefore, the presence and relative activities of enzymes that control the mutagenically active metabolites are crucial. (31 refs)

79-3282 Comparative Reactivity of Diolepoxide Metabolites of Carcinogenic Hydrocarbons with ϕ X 174 Viral DNA (Meeting Abstract). (Eng) Harvey, R. G. (Ben May Lab. Cancer Res., Univ. Chicago, Chicago, IL 60637); Hsu, W. T.; Weiss, S. B. *Proc Am Assoc Cancer Res* 20: 131; 1979 (1 ref)

79-3283 Peroxidase-catalyzed Binding of Hydrocarbons to DNA: A Model for Activation by One-Electron Oxidation (Meeting Abstract). (Eng) Cavalieri, E. (Eppley Inst., Univ. Nebraska Medical Center, Omaha, NE 68105); Rogan, E.; Katomski, P.; Sinha, D. *Proc Am Assoc Cancer Res* 20: 54; 1979 (no refs)

79-3284 On the Metabolic Activation of the Environmental Carcinogens Benzo(j)fluoranthene and Benzo(b)fluoranthene (Meeting Abstract). (Eng) LaVoie, E. J. (Naylor Dana Inst., American Health Foundation, Valhalla, NY 10595); Hecht, S. S.; Bedenko, V.; Amin, S.; Mazzaresse, R.; Hoffmann, D. *Proc Am Assoc Cancer Res* 20: 81; 1979 (no refs)

79-3285 Effect of 2-Aminoanthraquinone Feeding on Phospholipid Levels of Rat Liver and Kidney (Meeting Abstract). (Eng) Reddy, T. V. (NCI, NIH, Bethesda, MD 20014). *Proc Am Assoc Cancer Res* 20: 130; 1979 (no refs)

79-3286 Biological Activity of the Bay Region Diol Epoxides of Chrysene and Phenanthrene (Meeting Abstract). (Eng) Wood, A. W. (Dept. Biochemistry and Drug Metabolism, Hoffmann-La Roche Inc., Nutley, NJ 07110); Chang, R. L.; Levin, W.; Yagi, H.; Jerina, D. M.; Conney, A. H. *Proc Am Assoc Cancer Res* 20: 222; 1979 (2 refs)

79-3287 Electrophilic Superdelocalizability and Carcinogenesis by Polycyclic Aromatic Hydrocarbons--Pullman Theory. (Eng) Memory, J. D. (Sch. Physical and Mathematical Sciences, North Carolina State Univ., Raleigh, NC 27606). *Int J Quantum Chem* 15(4): 363-368; 1979.

Correlations between electrophilic superdelocalizabilities in the K region and carcinogenic potency were studied for benzantracene, 5,6- and 7,8-benzacridine, chrysene, and benzphenanthrene and their monomethyl and dimethyl derivatives. The results support the Pullman hypothesis that carcinogenicity is correlated with a reaction K region and an inactive L region. If the basic hypothesis is correct, the first step of the chain of reactions leading to carcinogenesis would be electrophilic attack of a carcinogenic hydrocarbon in the K region. The fact that several non-carcinogens have active K region as well as active L regions supports the L-region portion of the Pullman hypothesis. (10 refs)

79-3288 Ozonation of Mutagenic and Carcinogenic Polyaromatic Amines and Polyaromatic Hydrocarbons in Water. (Eng) Burleson, G. R. (Lobund Lab., Dept. Microbiology, Univ. Notre Dame, Notre Dame, IN 46556); Caulfield, M. J.; Pollard, M. *Cancer Res* 39(6, part 1): 2149-2154; 1979.

The *Salmonella*-microsome assay was used to monitor the

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effects of ozone on the mutagenicity of selected carcinogens and mutagens in water. Short periods of ozonation (1-4 min) completely inactivated the mutagenicity of several polyaromatic amine mutagens, including acriflavine, proflavine, and β -naphthylamine. Selected polyaromatic hydrocarbons were also sensitive to ozonation. Kinetic studies revealed that the mutagenicity of benzo(a)pyrene, 3-methylcholanthrene (3-MC), and 7-12-dimethylbenz(a)anthracene (DMBA) was destroyed after short periods of ozonation. To correlate loss of mutagenicity with loss of carcinogenicity, two polyaromatic hydrocarbons were treated with ozone, extracted from water with hexane, and tested for carcinogenicity in mice. When DMBA and 3-MC were treated with ozone, there was a substantial reduction in carcinogenicity compared with that in control groups exposed to hexane extracts of oxygenated DMBA and 3-MC. However, a small number of tumors developed in the group of animals receiving a hexane extract of ozonated DMBA. This activity may be due to breakdown products of DMBA that are not mutagenic to the Salmonella tester strains used. (32 refs)

79-3289 Correlation Between Benzo(a)pyrene (BP)-induced Transformation of Hamster Embryo Cells, and Inducible Aryl Hydrocarbon Hydroxylase (AHH) and Epoxide Hydrase (EH) (Meeting Abstract). (Eng) Poiley, J. A. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Raineri, R.; Pienta, R. J. *In Vitro* 15(3): 219; 1979 (no refs)

79-3290 Studies on Cocarcinogenesis Using C3H10T1/2CL8 Mouse Embryo Fibroblasts (Meeting Abstract). (Eng) Nesnow, S. (Metabolic Effects Section, US Environmental Protection Agency, Research Triangle Park, NC 27711); Garland, H.; Leavitt, S.; Vaughan, O. *Proc Am Assoc Cancer Res* 20: 27; 1979 (no refs)

79-3291 In Vitro Carcinogenesis with Cells in Early Passage. (Eng) DiPaolo, J. A. (Biology Branch, NCI, NIH, Public Health Service, U.S. Dept. Health, Education and Welfare, Bethesda, MD 20014); Casto, B. C. *Natl Cancer Inst Monogr* (48): 245-257; 1978.

Syrian golden hamster fetal cells mixed with irradiated hamster fetal cells were used in several studies of the phenomenon of transformation. Agents that are potent carcinogens in vivo proved to be potent transforming agents in vitro, but they reduced the cloning efficiency. Dose-response experiments showed that transformation is independent of toxicity and that it appears to be a one-hit

phenomenon that results from induction rather than selection. Agents (ie, α -naphthoflavone) that protect cells from the lethality induced by benzo(a)pyrene (BP) can either inhibit or promote BP transformation. The transforming ability of various agents may be increased by metabolic activation in vivo, the transformation in such cases being unassociated with viral activation. Transformed cells are able to produce tumors in vivo, and transformation and the subsequent neoplasia usually are not associated with an observable specific karyotypic abnormality. Pretreatment of cells with x-irradiation or methyl methanesulfonate or posttreatment with caffeine increases the frequency of chemical transformation. Most, if not all, chemical carcinogens will increase the sensitivity of hamster fetal cells to transformation by the carcinogenic simian adenovirus SA7. The enhancement of virus transformation is related to the length of chemical treatment and the interval between chemical and viral addition. Some of the transforming agents may also show mutagenic activity and produce chromosome aberrations in some systems. However, although DNA is the critical site for a mutagen, the critical target(s) of chemical carcinogens is still unknown. (49 refs)

79-3292 Decreased Half-Life of Benzo(a)pyrene Hydroxylase in the Presence of Aminophylline and 7,8-Benzoflavone (BF) (Meeting Abstract). (Eng) Avdalovic, N. (Wistar Inst., 36th St. at Spruce, Philadelphia, PA 19104). *Proc Am Assoc Cancer Res* 20: 266; 1979 (no refs)

79-3293 Membrane Transformation in Malignancy: The Normal and Neoplastic Transitional Epithelium of the Rat Urinary Bladder. A Thin Section and Freeze-Fracture Study. (Eng) Thiele, J. (Inst. Pathology, Medical Sch. Hannover, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany); Adolphs, H. D.; Reale, E. *J Submicrosc Cytol* 11(2): 249-261; 1979.

Thin sections and freeze-fracture replicas of normal Wistar rat transitional epithelium and of an N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT)-induced infiltrating papillary carcinoma of the urinary bladder in these rats were examined electron microscopically to study possible alterations of the plasma membranes following malignant transformation. Thin sections of the carcinoma showed that most of the cells exhibited numerous microvilli and a broad glycocalyx at their luminal surface; the high degree of asymmetry of the luminal aspect of the plasma membrane had disappeared. In addition, the characteristic discoid vesicles of the normal urothelium were replaced by flattened vesicles containing electron-dense material. Replicas of freeze-fractured normal transitional cells demonstrated plaques of closely arranged particles within the luminal plasma membrane and the limiting membrane of the discoid vesicles. Similar par-

ticulated plaques also marked the membrane of some sacculi of the Golgi apparatus. Replicas of freeze-fractured papillary carcinoma cells revealed the disappearance of the particulated plaques from the membranes in all these sites, an alteration that coincided with the loss of membrane asymmetry in thin sections. (31 refs)

79-3294 Growth Stimulating Effect of Normal Urine on the Rat Bladder Epithelium (Meeting Abstract). (Eng) Oyasu, R. (Northwestern Univ. Medical Sch., Chicago, IL 60611); Rowland, R. G.; Hirao, Y.; Izumi, K. *Proc Am Assoc Cancer Res* 20: 100; 1979 (1 ref)

79-3295 Induction of Papillary Tumors and Flat Carcinoma-In-Situ (CIS) of the Urinary Bladder by Cyclophosphamide in Male Fischer Rats (Meeting Abstract). (Eng) Cohen, S. M. (St. Vincent Hosp., Worcester, MA 01604); Arai, M.; Jacobs, J. B.; Friedell, G. H. *Proc Am Assoc Cancer Res* 20: 231; 1979 (no refs)

79-3296 Early Induction of Urinary Bladder (UB) Ornithine Decarboxylase (ODC) by Rodent Vesical Carcinogens (Meeting Abstract). (Eng) Matsushima, M. (Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53792); Bryan, G. T.; Pamukcu, A. M. *Proc Am Assoc Cancer Res* 20: 158; 1979 (no refs)

79-3297 Effects of Carcinogenic Nitrofurans on Murine Immunity (Meeting Abstract). (Eng) Erturk, E. (Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53792); Headley, D. B.; Bryan, G. T. *Proc Am Assoc Cancer Res* 20: 160; 1979 (no refs)

79-3298 A Sensitive Assay for Carcinogen-induced Early Changes in tRNA Methyltransferase Activity (Meeting Abstract). (Eng) Wainfan, E. (New York Blood Center, New York, NY 10021); Brody, H.; Balis, M. E. *Proc Am Assoc Cancer Res* 20: 65; 1979 (no refs)

79-3299 Evidence that Alteration of the Endoplasmic Reticulum Precedes Formation of Hyperplastic Nodules (Meeting Abstract). (Eng) Kizer, D. E. (Samuel Roberts Noble Foundation, Inc., Ardmore, OK 73401); Ringer, D. P.; Clouse, J. A.; Troop, K.; Levin,

W.; Griffin, M. J. *Proc Am Assoc Cancer Res* 20: 7; 1979 (no refs)

79-3300 2-Acetylaminofluorene (AAF) Ingestion and DNA Damage (Meeting Abstract). (Eng) Schwartz, E. L. (Dept. Pharmacology and Toxicology, Michigan State Univ., East Lansing, MI 48824); Goodman, J. I. *Proc Am Assoc Cancer Res* 20: 125; 1979 (no refs)

79-3301 Alteration of S-Adenosylmethionine Synthetases During Chemical Hepatocarcinogenesis and in Resulting Carcinomas. (Eng) Liau, M. C. (Dept. Biochemistry, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Chang, C. F.; Becker, F. F. *Cancer Res* 39(6, part 1): 2113-2119; 1979.

S-Adenosylmethionine synthetase (SAMS) isozymes were studied in hepatic nodules, primary hepatocellular carcinomas (HCC's), and various transplantable HCC's induced in rats by dietary acetylaminofluorene. With the exception of hepatoma 7800, the intermediate-Km isozyme was the only enzyme species detectable in transplantable hepatomas, including 7777, 311c, 253, 252, 56, and Novikoff hepatoma. Although hepatoma 7800 retained all of the hepatic isozymes, the levels of various isozymes were different from those of normal liver. During acetylaminofluorene hepatocarcinogenesis, there was an alteration in enzyme composition that commenced in neoplastic nodules and eventually attained the pattern commonly observed for many primary and transplantable HCC's. Evidence that the intermediate-Km isozyme of the Novikoff hepatoma was derived from the low-Km isozyme and that protein factors were partly responsible for this higher Km is presented. This alteration of the kinetic properties of tumor isozyme was unique to neoplastic nodules and tumors and, therefore, may be a significant characteristic of malignant evolution. The existence of an SAMS-transfer RNA (tRNA) methyltransferase complex was supported by the demonstration of cochromatography of these two enzymes on Sepharose 6B, coprecipitation at pH 5, and a higher efficiency of utilization of SAMS-synthesized S-adenosylmethionine by tRNA methyltransferases in the enzyme complex than that provided exogenously. After pH 5 treatment, most of the tumor enzyme complex retained the complex form, whereas liver enzyme complex was almost totally dissociated, indicating that tumor enzyme complex was more stable than liver enzyme complex against pH variations. The difference in the stability of this enzyme complex is probably due to factors characteristically associated only with SAMS from neoplastic tissues. (42 refs)

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- 79-3302 Alteration of S-Adenosylmethionine Synthetases During Chemical Hepatocarcinogenesis and in Resulting Carcinomas (Meeting Abstract). (Eng) Chang, C. F. (Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Liau, M. C.; Becker, F. F. *Proc Am Assoc Cancer Res* 20: 13; 1979 (no refs)
- 79-3303 Induction of both Epoxide Hydratase and a Putative Hemoprotein in Rat Liver Microsomes by Hepatocarcinogens (Meeting Abstract). (Eng) Sharma, R. N. (Univ. Toronto, Toronto, Ontario M5S 1A8, Canada); Murray, R. K. *Proc Am Assoc Cancer Res* 20: 37; 1979 (3 refs)
- 79-3304 The Principal Carcinogen-Protein Complex of the Hepatocarcinogen N-2-Fluorenylacetylamide in Rat Liver Cytosol (Meeting Abstract). (Eng) Blackburn, G. (Inst. Cancer Res., Fox Chase Cancer Center, 7701 Burholme Ave., Philadelphia, PA 19111). *Proc Am Assoc Cancer Res* 20:237; 1979 (no refs)
- 79-3305 A Quantitative Liver Colony Assay for a Carcinogen-resistant Proliferating Hepatocyte Phenotype (Meeting Abstract). (Eng) Laishes, B. A. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Center, Madison, WI 53706). *Proc Am Assoc Cancer Res* 20: 200; 1979 (1 ref)
- 79-3306 Administered AAF Is Bound to Rat Liver Microsomal Epoxide Hydrase (Meeting Abstract). (Eng) Griffin, M. J. (Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104). *Proc Am Assoc Cancer Res* 20: 203; 1979 (no refs)
- 79-3307 DNA Single-Strand Breaks in Various Tissues of Mice Treated with N-Diazoacetyl glycine Amide (DGA) (Meeting Abstract). (Eng) Brambilla, G. (Dept. Pharmacology, Univ. Genoa, I-16132 Genoa, Italy); Pino, A.; Robbiano, L.; Sciaba, L.; Parodi, S. *Proc Am Assoc Cancer Res* 20: 31; 1979 (no refs)
- 79-3308 Increased Nuclear Envelope ATPase Activity is Associated with Thioacetamide Treatment in Rats (Meeting Abstract). (Eng) Clawson, G. A. (Univ. California, San Francisco, CA 94143); Smuckler, E. A. *Proc Am Assoc Cancer Res* 20: 282; 1979 (no refs)
- 79-3309 Binding of the Carcinogen 2-Acetamidophenanthrene (AAP) to Rat Liver Nucleic Acids: Failure of the Hydroxamic Acid Ester Model for In Vivo Activation (Meeting Abstract). (Eng) Scribner, J. D. (Pacific Northwest Res. Foundation, 1102 Columbia St., Seattle, WA 98104); Koponen, G. *Proc Am Assoc Cancer Res* 20: 10; 1979 (4 refs)
- 79-3310 Repair of DNA Damage Induced in Human Cells by Aromatic Amine Carcinogens (Meeting Abstract). (Eng) Heflich, R. H. (Michigan State Univ., East Lansing, MI 48824); Hazard, R. M.; Lommel, L.; Maher, V. M.; McCormick, J. J. *Proc Am Assoc Cancer Res* 20: 90; 1979 (no refs)
- 79-3311 Small Scale Synthesis of N-Acetoxy-2-acetylaminofluorene (N-AcO-AAF) and Its Adducts (Meeting Abstract). (Eng) Van Roy, F. P. (Dept. Pathology, UCLA, Los Angeles, CA 90024); Moyer, G. H.; Austin, G. E. *Proc Am Assoc Cancer Res* 20: 219; 1979 (2 refs)
- 79-3312 Sulfotransferase in Rat Liver Nuclei (Meeting Abstract). (Eng) Stohrer, G. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Harmonay, L. A.; Brown, G. B. *Proc Am Assoc Cancer Res* 20: 285; 1979 (no refs)
- 79-3313 Rat Mammary Epithelium and Carcinogen-induced Mammary Adenocarcinoma in Long-Term Culture (Meeting Abstract). (Eng) Potter, A. H. (Veterans Admin. Medical Center, Minneapolis, MN 55417); Malejka-Giganti, D.; Rydell, R. E. *In Vitro* 15(3): 226; 1979 (no refs)
- 79-3314 Studies on Inhibition by Disulfiram (DSF) of Mammary Carcinogenesis in the Rat (Meeting Abstract). (Eng) Malejka-Giganti, D. (Veterans Admin. Medical Center, Minneapolis, MN 55417); McIver, R. C.; Rydell, R. E. *Proc Am Assoc Cancer Res* 20: 36; 1979 (no refs)
- 79-3315 Ascorbic Acid Inhibits Covalent Binding of Enzymatically Generated 2-Acetylaminofluorene-N-sulfate to DNA under Conditions in Which It Increased Mutagenesis in Salmonella TA-1538. (Eng) Andrews, L. S. (Pharmacology-Toxicology Pro-

gram, Natl. Inst. General Medical Sciences, NIH, Bethesda, MD 20014); Fysh, J. M.; Hinson, J. A.; Gillette, J. R. *Life Sci* 24(1): 59-64; 1979.

The effects of ascorbic acid on the covalent binding of ¹⁴C-labeled N-hydroxy-2-acetylaminofluorene (NOH-2AAF) to DNA were studied to investigate the apparent inverse relationship between mutagenicity and protein covalent binding in this system. NOH-2AAF became covalently bound to DNA during generation of the reactive N-sulfate ester. Elution of DNA digests from Sephadex LH-20 columns yielded two peaks of radioactivity (fractions A and B). The presence of these fractions was dependent on the sulfation reaction, since nonincubated zero-time samples had very little radioactivity associated with them. The radioactivity in fraction B appeared only when DNA was present during sulfation and during generation of the arylating species. This fraction may represent a 2AAF guanosine monophosphate adduct. The amount of radioactivity in fraction A was almost the same whether or not DNA was present during the reaction. Fraction A probably represents NOH-2AAF covalently bound to protein that associates with DNA and is not lost during DNA purification. Ascorbic acid inhibited total binding by 78% and DNA binding by 88%, as measured in fraction B. Since previous reports demonstrated that ascorbic acid increases mutagenesis 12-fold under these conditions, it is suggested that free radicals may play a role in the non-covalent ascorbate-dependent increase in mutagenesis. (25 refs)

79-3316 Role of Persistent DNA-Bound Residues of N-Hydroxy-2-Acetylaminofluorene (NOH-AAF) in Tumor Induction (Meeting Abstract). (Eng) Beland, F. A. (Natl. Center for Toxicological Res., Jefferson, AR 72079); Dooley, K. L.; Evans, F. E.; Jackson, C. D. *Proc Am Assoc Cancer Res* 20: 128; 1979 (no refs)

79-3317 Metabolism of Arylamines: Evidence for the Identity of Arylhydroxamic Acid Acyltransferase (AHAT) and Genetically Polymorphic N-Acetyltransferase (NAT) of Rabbit Liver (Meeting Abstract). (Eng) Glowinski, I. B. (Univ. Michigan, Ann Arbor, MI 48109); Weber, W. W.; Fysh, J. M.; Vaught, J. B.; King, C. M. *Proc Am Assoc Cancer Res* 20: 117; 1979 (no refs)

79-3318 Photoaffinity Label Derivatives of Fluorene as Probes in In Vitro Carcinogenesis (Meeting Abstract). (Eng) White, W. E. (Univ. Alabama, Birmingham, AL 35294); Sarraf, A. M.; Hixon, S. C. *In Vitro* 15(3): 220; 1979 (no refs)

79-3319 Direct Mutagenic Activity of Some Fluorene Derivatives in the TK+/- to TK-/- Mouse Lymphoma Assay (Meeting Abstract). (Eng) Amacher, D. (Dept. Safety Evaluation, Pfizer Central Res., Groton, CT 06340); Paillet, S.; Ellis, J. *Proc Am Assoc Cancer Res* 20: 185; 1979 (1 ref)

79-3320 Acyltransferase-mediated Binding of N-Hydroxyarylamides to Nucleic Acids (Meeting Abstract). (Eng) Allaben, W. T. (Natl. Center Toxicological Res., Jefferson, AR 72079); Beland, F. A. *Proc Am Assoc Cancer Res* 20: 67; 1979 (2 refs)

79-3321 In Vitro N-Oxidation of the Carcinogenic Arylamines by Dog Liver Microsomes (Meeting Abstract). (Eng) Brill, E. (Univ. Miami Sch. Medicine, Miami, FL 33101). *Proc Am Assoc Cancer Res* 20: 109; 1979 (no refs)

79-3322 Rearrangement of 1-Naphthylhydroxylamine (1-NOH) and Its N-Glucuronide Under Mildly Acidic Conditions as It Relates to Bladder Cancer (Meeting Abstract). (Eng) Hearn, W. L. (Dept. Pharmacology, Univ. Miami, Miami, FL 33101); Radomski, J. L. *Proc Am Assoc Cancer Res* 20: 117; 1979 (no refs)

79-3323 Effect of Urinary pH on the Excretion, Resorption, and Macromolecular Binding of Carcinogenic N-Hydroxy Arylamines in the Urinary Bladder (Meeting Abstract). (Eng) Kadlubar, F. F. (Natl. Center Toxicological Res., Jefferson, AR 72079); Oglesby, L. A.; Flammang, T. J. *Proc Am Assoc Cancer Res* 20: 129; 1979 (no refs)

79-3324 Carcinogenesis by Edible Mushroom Hydrazines (Meeting Abstract). (Eng) Toth, B. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105); Nagel, D. *Proc Am Assoc Cancer Res* 20: 43; 1979 (1 ref)

79-3325 Evidence Against the Polyp-Cancer Sequence in Dimethylhydrazine Induced Carcinoma of the Large Intestine (Meeting Abstract). (Eng) Maskens, A. P. (Cancer Res. Unit, Clinique Saint-Michel, B-1040 Brussels, Belgium); Dujardin-Loits, R. M. *Proc Am Assoc Cancer Res* 20: 51; 1979 (no refs)

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79-3326 Evidence for a Multi-Step Mechanism in Mouse Dimethylhydrazine-induced Colon Tumorigenesis (Meeting Abstract). (Eng) Deschner, E. E. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Long, F. C.; Maskens, A. P. *Proc Am Assoc Cancer Res* 20: 146; 1979 (no refs)

79-3327 Sex Difference in the Response to Colon Carcinogens (Meeting Abstract). (Eng) Yamamoto, R. S. (NCI, NIH, Bethesda, MD 20014); Nawata, H.; Rector, S. *Proc Am Assoc Cancer Res* 20: 270; 1979 (no refs)

79-3328 Dietary Fibre and Experimental Colon Cancer in the Rat. (Eng) Asp, N. G. (Dept. Nutrition, Chemical Center, Univ. Lund, Box 740, S-220 07, Lund 7, Sweden); Bauer, H.; Dahlqvist, A.; Fredlund, P.; Oste, R. *Nutr Cancer* 1(2): 70-73; 1979.

The possible effect of three different types of dietary fiber, wheat bran, carrot fiber, and citrus pectin, on tumor development was investigated in male Sprague-Dawley rats. The rats were fed a basic diet with or without wheat bran (4:1), carrot fiber (4:1), or citrus pectin (13.8:1) from 3 days before to 14 days after the administration of 1,2-dimethylhydrazine (DMH: 15 mg/kg sc, 1x/wk for 12 wk). The animals were killed 12-14 wk after the last DMH injection. The number of rats with colorectal tumors was as follows: 35/40 rats on the basic diet, 36/40 given wheat bran, 36/40 given carrot fiber, and 39/40 given citrus pectin. In addition, each group had about 20 infiltrating adenocarcinomas in the small intestine and several ear duct tumors. No tumors were detected in any other organs. The rats fed the wheat bran diet had more than twice the amount of fecal dry substance than the rats fed the basic diet, but no protective effect of wheat bran against the development of colorectal tumors was evident. The reason for the enhancement of carcinogenesis seen with the citrus pectin is not known. (17 refs)

79-3329 Effect of 2-Nitrofluorene, 1,2-Dimethylhydrazine, and Azoxymethane on *Salmonella typhimurium* Mutants in the Gastrointestinal Tract of Gnotobiotic Rats. (Eng) Carter, J. H. (Dept. Pharmacology, Harvard Medical Sch., Beth Israel Hosp., Boston, MA 02215); McLafferty, M. A.; Goldman, P. *Cancer Res* 39(6, part 1): 2026-2030; 1979.

The effect of *Lactobacillus plantarum* and *Bacteroides vulgatus* on the response of *Salmonella typhimurium* strain TA1538-associated gnotobiotic Sprague-Dawley rats to 2-nitrofluorene (NF) was investigated. Studies were also made of the use of other *Salmonella* tester strains in this

host-mediated assay to detect mutagenicity with the colon carcinogens 1,2-dimethylhydrazine (DMH) and azoxymethane (AM). After the ingestion of 3.4 mg NF in the diet, the fecal concentration of *his*⁺ revertants was greatly elevated in all groups of rats except those associated with TA1538 and *B. vulgatus*. The concentrations of the various bacteria were measured at eight sites in the gastrointestinal tract. *B. vulgatus* achieved high concentrations in the stomach when it was associated with TA1538, but its concentration was not as high when it was associated with *L. plantarum* plus TA1538. The concentration of *B. vulgatus* in individual rats correlated negatively with their response to NF. *B. vulgatus* readily reduced NF to 2-aminofluorene, a reaction that was negligible in cultures of *L. plantarum* and TA1538. Since 2-aminofluorene is less mutagenic than NF, *B. vulgatus* appears to diminish the revertant response by removing the more potent mutagen from within the gastrointestinal tract. Other Ames *Salmonella* tester strains (TA1535, TA100, and TA98) could also be maintained in association with otherwise germ-free rats. The feces of animals associated with TA1535 and TA100, however, showed a variable increase in the concentration of *his*⁺ revertants in response to the ingestion of DMH (21 mg/kg) and AM (19 mg/kg). (8 refs)

79-3330 The Effect of Dioctyl Sodium Sulfosuccinate (DSS) on Rat Colorectal Carcinogenesis (Meeting Abstract). (Eng) Karlin, D. A. (Dept. Medicine, M. D. Anderson Hosp. and Tumor Inst., Houston, TX); O'Donnell, R. T.; Jensen, W. E. *Gastroenterology* 76(5, part 2): 1164; 1979 (no refs)

79-3331 Discontinuities in Dose Response Curves from Toxicological Tests. (Eng) Anderson, R. L. (Procter and Gamble Co., Miami Valley Labs., Cincinnati, OH). *Household Pers Prod Ind* 16(3): 68-86; 1979.

Studies concerning dose-response curves for nitrilotriacetic acid (NTA) and demonstrating a clear threshold dose for the induction of carcinogenesis by this chemical are surveyed. A National Cancer Institute study demonstrated a relationship between high levels of NTA ingestion and an increased incidence of urinary tract cancer in rats. Other studies demonstrated that carcinogenic doses of both H₃NTA (1% in drinking water) and Na₃NTA (2%) resulted in the appearance of crystalluria in rats. The only crystalline material present in the urine was CaNaNTA. In males, changes in urinary Ca levels occurred only when the urinary NTA concentration was at least 100 micromoles/g which resulted from 0.75% NTA in the diet; once this threshold dose was exceeded, urinary Ca increased linearly with NTA dose, up to approx 200 micromoles/g. In males, but not females, the level of Na₃NTA that produced saturation of urinary Ca output was associated with increased urine volume, hematuria, and increased kidney/body wt

ratios. At all NTA doses, including zero, females excreted more urinary Ca than males eating the same diet. The ability of the females to excrete more Ca did not saturate, as was shown for males. In both sexes, H_3NTA ingestion was associated with more urinary Ca than Na_3NTA ingestion at levels that produced the same urinary load of NTA. These results suggest that urinary tract tumors are associated only with NTA doses that produce crystalluria. Thus, secondary physical damage, and not an effect produced by a tissue accumulation process, appears to cause these tumors. In addition, it seems unreasonable to conclude that any extrapolation of carcinogenesis incidence to possible exposure levels of NTA would lead to a rational assessment of risk to humans. (no refs)

- 79-3332 Effect of Dietary Polyunsaturated Fatty Acids on Growth of Mammary Adenocarcinomas in Mice and Rats (Meeting Abstract). (Eng) Hillyard, L. A. (Bruce Lyon Memorial Res. Lab., Children's Hosp. Medical Center, Oakland, CA 94609); Abraham, S. *Proc Am Assoc Cancer Res* 20: 17; 1979 (no refs)

- 79-3333 Transformation of Hamster Embryo Cells by Cholesterol- α -Epoxide and Lithocholic Acid. (Eng) Kelsey, M. I. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701); Pienta, R. J. *Cancer Lett* 6(3): 143-149; 1979.

Cholesterol and three of its derivatives, cholesterol- α -epoxide, sulfolithocholic acid, and lithocholic acid, were tested for their ability to transform hamster embryo cells. Of the two cholesterol, only cholesterol- α -epoxide was active in this system, with transformation occurring at total concentrations of 54-2,100 nanomoles (nmol) [the positive control, 3-methylcholanthrene, produced max transformation (0.35%-1.0%) at a total concentration of 360 nmol]. In tests with sulfolithocholic and lithocholic acids, only lithocholic acid was active. Max transformation (0.11%-0.13%) occurred at the lowest doses tested (1.2-1.8 nmol). These results are the first examples of in vitro cell transformation activity by cholesterol derivatives and metabolites that may be important in colon carcinogenesis. The results suggest that the hamster embryo cell transformation system may be useful for screening endogenously formed steroid metabolites for their potential carcinogenicity. (29 refs)

- 79-3334 Vaginal Adenocarcinoma in a Gravid with Prenatal DES Exposure. (Eng) Del Castillo, H. (Dept. Gynecology, Medical Center del Oro Hosp., Houston, TX); Rubio, P. A.; Farrell, E. M. *Int J Gynaecol Obstet* 16(4): 271-273; 1979.

A case report is presented of a 22-yr-old pregnant woman with vaginal carcinoma who had been exposed to diethylstilbestrol (DES) in utero. The appearance of the tumor (in the 4th month of pregnancy) was consistent with descriptions of vaginal adenocarcinoma, but not metastatic adenocarcinoma. Surgical excision of the lesion failed, but interstitial irradiation was successful. Six mo after treatment, the patient gave birth to a normal term infant by cesarean section. Two subsequent children were delivered by cesarean section after normal pregnancies. There has been no evidence of metastatic disease or recurrence in 11.5 yr of follow-up. Since adenocarcinoma of the cervix and vagina is asymptomatic during its early growth and since these carcinomas may develop soon after menarche, girls exposed to DES prenatally should begin screening examinations either after first menstruation or at 14 yr of age, if menstruation has not yet begun. The recent identification of elevated serum (cathepsin B1) activity in tumor patients suggests the potential development of a serum enzyme test to detect recurrent disease. (4 refs)

- 79-3335 Covalent Binding of Diethylstilbestrol to DNA Catalyzed by Hepatic and Uterine Microsomes (Meeting Abstract). (Eng) Okey, A. B. (Univ. Windsor, Windsor, Ontario, Canada); Nebert, D. W. *Proc Am Assoc Cancer Res* 20: 205; 1979 (no refs)

- 79-3336 Ovarian Abnormalities in Mice Following Prenatal Exposure to Diethylstilbestrol (Meeting Abstract). (Eng) Newbold, R. R. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); McLachlan, J. A. *Proc Am Assoc Cancer Res* 20: 103; 1979 (no refs)

- 79-3337 The Testicular Estrogen Receptor System in Two Strains of Mice Differing in Susceptibility to Estrogen-induced Leydig Cell Tumors. (Eng) Sato, B. (Third Dept. Internal Medicine, Osaka Univ. Hosp., Fukushima-ku, Osaka, Japan); Spomer, W.; Huseby, R. A.; Samuels, L. T. *Endocrinology* 104(3): 822-831; 1979.

Studies were conducted to establish the presence of an intracellular estrogen transport system in the Leydig cells of mouse testes and to compare the system in tumor-susceptible (BALB/c) and tumor-resistant [C_3H Bi (Z)] mouse strains. Cytosols obtained from cryptorchid testes of both mouse strains bound 17β -estradiol (E_2) and diethylstilbestrol (DES) specifically. The dissociation constant of this binding component for E_2 was 5×10^{-9} M. Gel filtration of the cytosol before incubation markedly increased the affinity (3×10^{-10} M). In vitro translocation of the cytosol binding component to the nucleus was demonstrated in both mouse strains using either estrogen.

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Quantitation of the *in vivo* translocation, using the exchange method after a single injection of E_2 (2.5 μ g/mouse), revealed a rapid decrease in cytoplasmic receptor content accompanied by a concomitant increase in nuclear receptor content. Greater nuclear receptor content was identified in nuclei from BALB/c mice than in those from Z mice 45 min after E_2 injection. The binding behavior of the E_2 -receptor complexes to nuclei was studied by KCl extraction. The percent extracted from the nuclei of Z strain mice was significantly greater than that from the BALB/c strain at all KCl concentrations tested. Cross-over experiments in a cell-free system suggest that the difference in binding is due to differences in chromatin rather than in nuclear estrogen-receptor complexes. The greater nuclear receptor content and its stronger binding to chromatin might explain the higher susceptibility of BALB/c mice to estrogen-induced phenomena, including neoplastic transformation. (32 refs)

- 79-3338 **Endometrial Pathology and Estrogens.** (Eng) Rosenwaks, Z. (Dept. Obstetrics and Gynecology, Div. Reproductive Endocrinology, State Univ. New York at Stony Brook, Health Sciences Center, Stony Brook, NY 11794); Wentz, A. C.; Seegar Jones, G.; Urban, M. D.; Lee, P. A.; Migeon, C. J.; Parmley, T. H.; Woodruff, J. D. *Obstet Gynecol* 53(4): 403-410; 1979.

Between 1954 and 1977, endometrial biopsies were obtained from 46 hypogonadal patients (44 with gonadal dysgenesis; 2 panhypopituitary) who were receiving estrogen-progestogen therapy. The age of the patients ranged from 16 to 46 yr (mean 24.3 yr), and the mean duration of therapy ranged from 5 mo to 22 yr (mean, 86 mo). The estrogen therapy consisted of Premarin or diethylstilbestrol; most patients received medroxyprogesterone acetate as the progestational agent. Four patients received estrogen treatment alone. Endometrial abnormalities occurred only in patients receiving a total lifetime conjugated estrogen dose of $\geq 2,500$ mg and who had received estrogen treatment for a period >4.2 yr. The biopsy outcome was significantly related ($p < 0.01$) to estrogen dose at the time of biopsy and to the total lifetime dose ($p < 0.05$). The progestational drugs administered did not protect against the development of endometrial abnormalities. None of the abnormal endometrial patterns were associated with abnormal vaginal bleeding. (29 refs)

- 79-3339 **Oral Estro-progestative Contraception and Cervical and Vaginal Cytology.** (Fre) Favre, J. (Laboratoire de Cytologie, Centre Hospitalier Regional de Grenoble, BP 217 X, 38043 Grenoble Cedex, France); Siebert, S.; Drevet, N. *Sem Hop Paris* 55(7/8): 384-388; 1979.

A cytological study of 1,685 cervical and vaginal smears

from 1,500 women using oral contraceptives (OC) and of 1,000 smears from 1,000 control women not using such contraceptives is described. The frequency of benign abnormal smears (ie, abnormalities of the malpighian layer, basal layer, and columnar epithelium) was 1,028/1,685 vs 561/1,000. The incidence of metaplasia in the two groups was 494/1,685 and 319/1,000, respectively. Mild dysplasia was found in 54 women using OC and in 8 controls; the respective incidences of moderate dysplasia were 6 and 3. Severe dysplasia (malignant cells) was found in 1 OC user and in 3 controls; this difference could not be evaluated statistically due to the number of patients involved. Spontaneous regression of the abnormal cytological changes was seen in most cases after cessation of OC use. (37 refs)

- 79-3340 **A Direct Effect of Hydrocortisone on Metastasis of Premalignant and Malignant Mouse Mammary Cells (Meeting Abstract).** (Eng) Anderson, L. W. (Dept. Zoology, Washington State Univ., Pullman, WA 99164); Danielson, K. G.; Hosick, H. L. *Proc Am Assoc Cancer Res* 20: 25; 1979 (no refs)

- 79-3341 **Pituitary Secretion Stimulates Growth of Malignant Lymphomas in Nb Rats - Evidence In Vivo and In Vitro (Meeting Abstract).** (Eng) Gout, P. W. (Dept. Biochemistry, Univ. British Columbia, Vancouver, B.C., V6T 1W5, Canada); Beer, C. T.; Noble, R. L. *Proc Am Assoc Cancer Res* 20: 27; 1979 (no refs)

- 79-3342 **Demonstration of a Direct Carcinogenic Effect of Estradiol (E_2) on Leydig Cells of the Mouse (Meeting Abstract).** (Eng) Huseby, R. A. (Henry Ford Hosp., Detroit, MI 48202). *Proc Am Assoc Cancer Res* 20: 55; 1979 (no refs)

- 79-3343 **Influence of Sex on the Rate of Growth of Human Malignant Melanoma (Meeting Abstract).** (Eng) Beattie, C. W. (Div. Surgical Oncology, Univ. Illinois, Chicago, IL 60680); Chaudhuri, P. K.; Walker, M. J.; Das Gupta, T. K. *Proc Am Assoc Cancer Res* 20: 130; 1979 (no refs)

- 79-3344 **Hormone Responsiveness of a Rat Chondrosarcoma (Meeting Abstract).** (Eng) Salomon, D. S. (LDBA, NIDR, NIH, Bethesda, MD 20014); Paglia, L. M.; Verbruggen, L. *Proc Am Assoc Cancer Res* 20: 20; 1979 (no refs)

79-3345 Effect of Estrogen and Progesterone on Growth Fraction of Human Breast Tumors (Meeting Abstract). (Eng) Dao, T. L. (Roswell Park Memorial Inst., Buffalo, NY 14263); Sinha, D. K.; Nemoto, T. *Proc Am Assoc Cancer Res* 20: 105; 1979 (1 ref)

79-3346 Effect of Androgens on Steroid Hormone Receptors in the MTW-9B Transplantable Rat Mammary Tumor (Meeting Abstract). (Eng) Ip, M. M. (Roswell Park Memorial Inst., Buffalo, NY 14263); Milholland, R. J.; Kim, U.; Rosen, F. *Proc Am Assoc Cancer Res* 20: 123; 1979 (1 ref)

79-3347 Inhibition of Spontaneous Breast Cancer Formation in C3H (Avy/a) Mice by Long-Term Treatment with Dehydroepiandrosterone (Meeting Abstract). (Eng) Schwartz, A. G. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA 19140). *Proc Am Assoc Cancer Res* 20: 2; 1979 (2 refs)

79-3348 Carcinoma of the Prostate May be Caused by the Sequential Action of Antagonistic Sex Hormones - Transplants in Nb Rats Show Unique Hormone Responses (Meeting Abstract). (Eng) Noble, R. L. (Animal Care Centre, Univ. British Columbia, Vancouver, B.C., V6T 1W5, Canada). *Proc Am Assoc Cancer Res* 20: 26; 1979 (2 refs)

79-3349 Spontaneous Rupture of a Liver Cell Adenoma after Long Term Methyltestosterone: Report of a Case Successfully Treated by Emergency Right Hepatic Lobectomy. (Eng) Bird, D. (Carey Coombes Res. Fellow, Dept. Surgery, Bristol Royal Infirmary, Bristol, England); Vowles, K.; Anthony, P. P. *Br J Surg* 66(3): 212-213; 1979.

A case of spontaneous rupture of a liver cell adenoma in a 39-yr-old female-to-male transsexual who had received 150 mg/day methyltestosterone (MT) for 7 yr is reported. An emergency right hepatic lobectomy was performed successfully. Histologic examination also revealed peliosis hepatis. 17- α -Alkylated androgenic-anabolic steroids such as MT may cause abnormalities of standard liver function tests and cholestatic jaundice, and they have been implicated in the development of hepatic adenoma or hepatocellular carcinoma. It has been suggested that a single pathological process (hyperplasia, related perhaps to the anabolic effects of MT) could be responsible for both peliosis and the formation of nodules and tumors. Since treatment in transsexuals must be life-long, further cases

such as the one reported here can be expected. It is hoped that virilizing drugs other than 17- α -alkylated steroids can be used. (19 refs)

79-3350 Acute Non-lymphocytic Leukemia (ANLL) in Patients with Ovarian Carcinoma Following Long Term Treatment with Treosulfan (=Dihydroxybusulfan) (Meeting Abstract). (Eng) Pedersen-Bjergaard, J. (Finsen Inst., 49, Strandboulevarden, Copenhagen DK-2100, Denmark); Sorensen, H. M.; Hou-Jensen, K.; Ersbol, J. *Proc Am Assoc Cancer Res* 20: 287; 1979 (no refs)

79-3351 Inhibitory Effects of Trimethoprim-Sulfamethoxazole (TMP-SMZ) on L1210 Leukemic Cells (Meeting Abstract). (Eng) Rivard, G. E. (Centre de Recherche Pediatrique, Hopital Ste-Justine, Montreal, Quebec H3T 1C5, Canada); Momparler, L. *Proc Am Assoc Cancer Res* 20: 290; 1979 (1 ref)

79-3352 Oncogenicity of 4-Demethoxydaunomycin (DMD): Lack of Correlation among In Vitro Short-Term Tests and Between In Vitro and In Vivo Findings (Meeting Abstract). (Eng) Marquardt, H. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Philips, F. S.; Marquardt, H.; Sternberg, S. S. *Proc Am Assoc Cancer Res* 20: 45; 1979 (no refs)

79-3353 Scanning Electron Microscopy (SEM) of Regenerating Hyperplastic Urinary Bladder Epithelium (Meeting Abstract). (Eng) Fukushima, S. (St. Vincent Hosp., Worcester, MA 01604); Cohen, S. M.; Jacobs, J. B.; Friedell, G. H. *Proc Am Assoc Cancer Res* 20: 233; 1979 (no refs)

79-3354 Time Dependence of Cyclophosphamide Enhancement-Inhibition of Experimental Metastasis (Meeting Abstract). (Eng) Tseng, M. H. (Roswell Park Memorial Inst., Buffalo, NY 14263); Tan, M. H. *Proc Am Assoc Cancer Res* 20: 212; 1979 (no refs)

79-3355 The Oncogenic Effect of Immunosuppressive (Cytotoxic) Agents in (NZB x NZW) Mice. (Eng) Mitrou, P. S. (Zentrum der Inneren Medizin, Johann Wolfgang Goethe-Universitat, Theodor-Stern-Kai 7, D-6000 Frankfurt/M. 70, W. Germany); Fischer, M.;

Mitrou, G.; Rottger, P.; Holtz, G. *Arzneim Forsch* 29(3): 483-488; 1979.

The effects of daily and weekly sc treatment with azathioprine (ATP) or isophosphamide (IPA) over periods of 14 or 16 mo on survival, glomerulonephritis, antinuclear antibodies (ANA), blood cell counts, and tumorigenesis in female (NZB x NZW) mice were studied. ANA titers were significantly reduced by daily ATP and daily or weekly IPA; weekly ATP had no significant effect on ANA titers. IPA significantly increased survival time in a dose-dependent fashion at doses of 0.2 or 0.4 mg daily or weekly. At 0.2 mg/day, ATP also increased survival time, but 0.4 mg/day produced significant mortality due to anemia; weekly ATP did not prolong survival. At low daily doses, ATP and IPA produced glomerular lesions in all mice within 20 mo. Glomerulonephritis was also induced in a high percentage of mice treated intermittently or at high daily doses with ATP or IPA. Both ATP and IPA caused a significant, dose-dependent increase in malignant lymphomas. Most tumors appeared after long-term treatment (at least 10 mo). Intermittent IPA treatment was as oncogenic as daily high-dose treatment. In contrast, intermittent ATP produced results statistically comparable to those of the control group. It is not known whether the cytotoxic agents themselves or the immunosuppression they induce initiated tumor formation. (32 refs)

79-3356 Mutagenic Intercalating Agents and their Induction of Immunoreactivity to Antinucleoside Antibodies (Meeting Abstract). (Eng) Bases, R. (Albert Einstein Coll. Medicine, Bronx, NY 10461); Liebeskind, D.; Mendez, F.; Mendez, L.; Neubort, S. *Proc Am Assoc Cancer Res* 20: 66; 1979 (no refs)

79-3357 New Leukemia in the Course of Therapy of Acute Lymphoblastic Leukemia. (Eng) Ravindranath, Y. (Children's Hosp. of Michigan, 3901 Beaubien Blvd., Detroit, MI 48201); Inoue, S.; Considine, B.; Lusher, J.; Zuelzer, W. W. *Am J Hematol* 5(3): 211-223; 1978.

The development of a second, morphologically different leukemia was observed recently in three acute lymphoblastic leukemia (ALL) patients while on therapy. The second leukemias occurred 23, 27, 30 mo after initial remission induced with vincristine, 6-mercaptopurine, and prednisone. All three were receiving systemic chemotherapy, including methotrexate, and prophylactic fractional irradiation (100 rads) to the CNS. The second leukemias involved one case of juvenile chronic myelogenous leukemia (JCML) and two cases of acute myeloblastic leukemia, as diagnosed by the usual morphologic criteria including the presence of Auer rods in one. In two patients, a cytogenetically new clone was detected in the remission

marrow 10 and 12 mo preceding the overt change in clinical status. These three cases demonstrate that second leukemias occur in ALL patients and that some late relapses fall into this category. The possible etiologic role of modern intensive treatment methods in the development of second leukemia is discussed. (35 refs)

79-3358 Relationship Between Levodopa Therapy and Malignant Tumors. (Swe) Botteri, A. (Langvardskliniken, Sundsvalls sjukhus, S-851 86 Sundsvall, Sweden); Nordstrom, P. O. *Lakartidningen* 76(5): 316-317; 1979.

The literature on the possible relationship between malignant melanoma and L-dopa [3-(3,4-dihydroxyphenyl)-L-alanine] treatment for Parkinson's disease is reviewed, and a new case is reported. Activation of malignant melanoma or unusually high activity of melanoma was observed in many patients during or shortly after L-dopa treatment, which suggests a possible causal relationship between malignant melanoma and L-dopa, but the relationship cannot be definitely proved in view of the unpredictable clinical course of melanoma. L-Dopa, which is a physiological intermediate in melanin synthesis, may facilitate the growth of melanoma. Melanocytes are able to transform L-dopa into melanin via tyrosinase. Increased secretion of somatotropin, which can stimulate the activity of melanocytes, was observed during L-dopa treatment. A 73-yr-old woman with Parkinsonism was treated with L-dopa (max daily dose 0.8 g) for about 2 yr, during which time she developed uterine melanoma. A recurrent melanoma was found in the vulva after excision of the primary tumor. (15 refs)

79-3359 Correlation of L-Azaserine Toxicity and Gamma-Glutamyl Transpeptidase (GGT) Activity in Established Cell Lines (Meeting Abstract). (Eng) Perantoni, A. (NCI, NIH, Bethesda, MD 20014); Berman, J. J.; Rice, J. M. *Proc Am Assoc Cancer Res* 20: 13; 1979 (1 ref)

79-3360 Mutagenicity and Carcinogenicity of Polyhydric Phenols (Meeting Abstract). (Eng) Wang, C. Y. (Michigan Cancer Foundation, Detroit, MI 48201); Klemencic, J. M. *Proc Am Assoc Cancer Res* 20: 117; 1979 (no refs)

79-3361 Diffuse Metanephric Adenoma after In Utero Aspirin Intoxication. A Unique Case of Progressive Renal Failure. (Eng) Bove, K. E. (Children's Hosp. Res. Foundation, Children's Hosp. Medical Center,

Cincinnati, OH); Bhatena, D.; Wyatt, R. J.; Lucas, B. A.; Holland, N. H. *Arch Pathol Lab Med* 103(4): 187-190; 1979.

Diffuse persistent glomerular immaturity and focal proximal tubular ectasia were seen in bilateral open renal biopsy specimens from a male infant with fluid and salt depletion and slowly progressive renal failure. The pregnancy was complicated by a maternal suicide attempt at 2 mo gestation by ingestion of 19 g aspirin. The baby's nursery course was uncomplicated, but he was hospitalized at ages 6 and 10 wk for failure to thrive. During the first year, severe renal rickets developed despite treatment. Subsequently, diffuse tubulopapillary renal adenoma subtotally replaced each kidney, thereby necessitating renal transplantation at age 7. Origin of diffuse metanephric adenoma from persistent primitive epithelium of the proximal nephron is postulated and partly substantiated. It is proposed that this case of persistent proximal nephronic epithelial immaturity and diffuse metanephric adenoma is a variant of nephroblastomatosis and that the first trimester suicide attempt with aspirin may have initiated the maturation defect that preceded neoplastic transformation. (13 refs)

79-3362 Effect of Hepatocarcinogens on the Adenine Purine Nucleotide Cycle During the Initiation Phase of Carcinogenesis. (Eng) Smith, L. D. (H. L. Snyder Memorial Res. Foundation, Winfield, KS 67156); Emerson, R.; Nixon, L. K. *Cancer Res* 39(6, part 1): 2132-2138; 1979.

The activities of the purine nucleotide cycle enzymes, ie, adenylosuccinate (SAMP) synthetase, SAMP lyase, and adenosine 5'-monophosphate (AMP) deaminase, were determined in hepatic tissues of female Sprague-Dawley rats fed and/or given ip injections of 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB), 4'-methyl-4-dimethylaminoazobenzene (4'-Me-DAB), thioacetamide, ethionine, or 2-acetylaminofluorene (2-AAF). SAMP lyase activity showed an early increase in rats fed diets containing 0.06% of the hepatocarcinogens. AMP deaminase activity increased with 3'-Me-DAB and thioacetamide but not with ethionine or 2-AAF. SAMP synthetase activity was either unaffected or was inhibited by the carcinogens. Increases in SAMP lyase activity were noted as early as 48-72 hr following ip injections of thioacetamide and ethionine (each 50 mg/kg/day). The response of this enzyme was not duplicated by carcinogenic analogs such as 4'-Me-DAB or methionine. These data imply the interaction of active carcinogens with SAMP lyase and to some extent with AMP deaminase or with some mechanism responsible for their synthesis and/or release. This interaction may be a significant component of the initiation phase of carcinogenesis. (20 refs)

79-3363 γ -Glutamyl Transferase (γ GT) and Disulfiram (DSF) as Tools in the Elucidation of the

Preneoplastic to Neoplastic Change in Azodye-induced Carcinogenesis (Meeting Abstract). (Eng) Fiala, S. (Veterans Admin. Medical Center, Martinsburg, WV 25401); Ostrander, H.; Trout, E. C. *Proc Am Assoc Cancer Res* 20: 82; 1979 (no refs)

79-3364 Role of Nutritional Factors During a Complex Social-Hygienic Examination of Female Aniline Dye Workers. (Rus) Podluzhnyi, P. A. (Medical Inst., Perm', USSR). *Gig Sanit* (1): 44-47; 1979.

The role of various vocational-hygienic and social-hygienic factors, including nutritional regimen and dietary habits, in the development of genital organ pathology was studied in a group of 1,264 female aniline dye workers. The women were 25-39 yr old, all were married, and all had a duration of exposure >5 yr. A matching group of female workers who had not been exposed to aniline dyes served as controls. Compared with the control group, the aniline dye workers had a greatly increased incidence of menstrual-ovarian function disorders (26.41% vs 3.34%), inflammatory diseases of the genital organs (22.01% vs 11.66%), and precancerous conditions of the genital organs (10.69% vs 6.67%). Analysis of dietary habits showed that gynecological morbidity was significantly greater in workers who did not adhere to a regular nutritional regimen (ie, they had only 1-2 meals per day and irregular intervals between meals). (2 refs)

79-3365 Recent Data on the Examination of the Mutagenic Effect of a Dinitro-o-cresol-containing Pesticide by Different Test Methods. (Eng) Nehez, M. (Inst. Hygiene and Epidemiology, Szeged Univ. Medicine, Szeged, Hungary); Selyes, A.; Paldy, A.; Berencsi, G. *Ecotoxicol Environ Saf* 2(3/4): 243-248; 1978.

The mutagenic effect of the 50% dinitro-o-cresol (DNOC)-containing pesticide Krezonit E was assayed by several methods. Human WBC cultures were incubated for 72 hr with 0.02, 0.2, or 2.0 μ g/ml DNOC. The percentage of aberrations increased with increasing concentration (56.4% at 0.02 to 74.5% at 2.0 μ g/ml). Chromatid-type aberrations were dose-dependent, but chromosome-type aberrations were not. CFLP white male mice were given DNOC in two doses regimens. Ten mice were given a single 20-mg/kg dose ip. Other groups of mice were given DNOC ip at doses of 1.0, 5, or 10 mg/kg on days 1, 3, 5, and 9 of the experiment. Bone marrow preparations were prepared 24 hr after the last administration. The subchronic dose had a more harmful effect on bone marrow cells than the single dose. The rate of aberrant cells increased from 75% for the single dose or the chronic 1.0-mg/kg dose to 95% for the chronic 10-mg/kg dose. Gametic mutations were studied in 12-wk-old male mice treated with a single 10-mg/kg dose of

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DNOC ip. Only gametic aberrations occurred in the control group, but gametic and autosomal aberrations were observed in the treated group. Significantly higher results were obtained on postinjection day 20, which indicates that the pesticide has a harmful effect on spermatogonia. The dominant lethal values were significantly higher in the sixth week. The rate of aberrations in F_1 embryonic chromosomes was significantly higher in the first, second, and third weeks after treatment of the male parent than in the control group. It is concluded that Krezonit E is mutagenic in human WBC cultures in vitro, in mouse bone marrow chromosomes in vivo, and in mouse germ cells. (18 refs)

- 79-3366 Study of Potential Carcinogenicity of m-Toluenediamine. (Rus) Pylev, L. N. (Cancer Res. Center, Moscow, USSR); Koval'skaia, G. D.; Genin, V. A. *Gig Sanit* (2): 37-41; 1979.

The potential carcinogenicity of m-toluenediamine (MTD) was evaluated in random-bred albino rats and CC57 mice. MTD was given either sc (rats: 1-8 mg in 0.5 ml saline 1x/wk; mice: 2 mg in 0.2 ml saline 1x/wk) or intragastrically (rats: 0.5-4 mg in 0.5 ml saline 5x/wk for 16 mo and then 3x/wk; mice: 2 mg in 0.2 ml saline 5x/wk for 16 mo and then 3x/wk). The sc administration of MTD induced tumors in 9/25 rats (3 had reticulosis, 5 had fibroadenoma of the mammary gland, and 1 had an adrenal tumor) and in 14/25 mice (12 had leukemia, and 2 had a lung tumor). The av latent period of tumor development was 16 and 5.5 mo and the av duration of survival was 19 and 14 mo in rats and mice, respectively. The intragastric administration of MTD induced tumors in 8/36 rats (2 had leukemia, 5 had fibroadenoma of the mammary gland, and 1 had an abdominal tumor) and in 10/55 mice (all 10 had leukemia). The av latent period of tumor development was 12.5 and 3.5 mo and the av duration of survival was 20 and 6.5 mo in rats and mice, respectively. (6 refs)

- 79-3367 Covalent Modification of Polynucleotides with the Carcinogen 1-Methyl-3-acetoxanthine (Meeting Abstract). (Eng) Mathews, R. A. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Stohrer, G. *Proc Am Assoc Cancer Res* 20: 224; 1979 (no refs)

- 79-3368 Development of Kidney and Liver Tumors in Offspring of Mice Exposed to Safrole During Gestation and Lactation (Meeting Abstract). (Eng) Vesselinovich, S. D. (Univ. Chicago, Pritzker Sch. Medicine, Chicago, IL 60637); Mihailovich, N.; Rao, K. V. *Proc Am Assoc Cancer Res* 20: 164; 1979 (no refs)

- 79-3369 The Mutagenicities of Safrole, Estragole, Eugenol, trans-Anethole, and Some of Their Known or Possible Metabolites for *Salmonella typhimurium* Mutants. (Eng) Swanson, A. B. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Sch., Madison, WI 53706); Chambliss, D. D.; Blomquist, J. C.; Miller, E. C.; Miller, J. A. *Mutat Res* 60(2): 143-153; 1979.

The mutagenicity of safrole, estragole, anethole, and eugenol and some of their known or possible metabolites toward *Salmonella typhimurium* strains TA1535, TA100, and TA98 was determined. Highly purified 1'-hydroxyestragole (HEG) and 1'-hydroxysafrole (HS) were mutagenic (approx 15 and 10 revertants/micromole (μ mol), respectively) for TA100 in the absence of NADPH-fortified liver microsomes; trans-anethole and estragole appeared to have very weak activity. 3'-Hydroxyanethole was too toxic to be evaluated. Supplementation with fortified rat liver microsomes and cytosol converted 3'-hydroxyanethole to a mutagen(s) and increased the mutagenicity of HEG, HS, estragole, and anethole for TA100. No mutagenicity was detected for safrole or eugenol with or without the fortified liver preparations. The electrophilic 2',3'-oxides of safrole, HS, 1'-acetoxysafrole, 1'-oxosafrole, estragole, HEG, and eugenol showed dose-dependent mutagenic activities for TA1535 in the absence of fortified liver microsomes. These mutagenicities ranged from about 330 revertants/ μ mol for 1'-oxosafrole-2',3'-oxide to about 7,000 revertants/ μ mol for safrole-2',3'-oxide. The arylalkenes, their hydroxylated derivatives, or their epoxides were not mutagenic toward TA98, except for 1'-oxosafrole-2',3'-oxide, which had weak activity. Since the arylalkenes are hydroxylated and/or epoxidized by hepatic microsomes, hydroxy and epoxide derivatives appear to be proximate and ultimate mutagenic metabolites, respectively, of the arylalkenes. (30 refs)

- 79-3370 Effect of Benzene and Its Metabolites on SCE in Human Lymphocyte Cultures (Meeting Abstract). (Eng) Diaz, M. (Fundacion de Genetica Humana, Salta 661, 1074, Buenos Aires, Argentina); Fijtman, N.; Carricarte, V.; Braier, L.; Diez, J. *In Vitro* 15(3): 172; 1979 (no refs)

- 79-3371 Proposed Feed and Food Additive Tolerances for 4-Amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one. (Eng) Taylor, R. (Office Pesticide Programs, Registration Div., TS-767, Environmental Protection Agency, E. Tower, 401 M St., SW, Washington, DC 20460). *Fed Regist* 44(89): 26750-26751; 1979.

It is proposed that tolerances for residues of the herbicide

4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one in the milled fractions of barley and wheat (excluding flour of both grains) be established at 3 ppm. (no refs)

- 79-3372 Tolerances and Exemptions from Tolerances for Pesticide Chemicals in or on Raw Agricultural Commodities; 4-Amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one. (Eng) Taylor, R. (Registration Div. (TS-767), Office Pesticide Programs, Environmental Protection Agency, 401 M St., SW, Washington, DC 20460). *Fed Regist* 44(89): 26743-26744; 1979.

Tolerances for residues of the herbicide 4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one were established at 0.75 ppm on barley grain and wheat grain, at 1 ppm on barley straw and wheat straw, and at 2 ppm on wheat forage. Toxicological data supporting the proposed tolerances included rat acute po lethal dose studies, rabbit and rat teratogenicity studies, 2-yr dog and rat feeding studies, an 18-mo mouse carcinogenicity study (negative at 2,500 ppm, the highest level fed), a three-generation rat reproduction study, and a mouse mutagenicity study. (no refs)

- 79-3373 Ames Test of 1-(X-Phenyl)-3,3-dialkyltriazenes. A Quantitative Structure-Activity Study. (Eng) Venger, B. H. (Dept. Chemistry, Pomona Coll., Claremont, CA 91711); Hansch, C.; Hatheway, G. J.; Amrein, Y. U. *J Med Chem* 22(5): 473-476; 1979.

The mutagenicity of 1-(X-phenyl)-3,3-dialkyltriazenes was tested in the Ames test with *Salmonella typhimurium* strain TA92, and a quantitative structure-activity relationship (QSAR) was formulated. The equation is based on 17 congeners and has a correlation coefficient of 0.974. The QSAR for mutagenicity was compared with the QSAR for antileukemia action and toxicity (LD₅₀) in mice. In addition, the mutagenicity of aflatoxin B₁ and the antitumor drug 5-(3,3-dimethyl-1-triazeno)imidazole-4-carboxamide (DTIC) was determined. Increasing lipophilicity and increased electron release via through resonance were found to increase mutagenicity. There was a 16,000-fold range in the mutagenicity of the triazenes, and an equation is given that can predict the potency within a factor of ± 2 . These results provide preliminary evidence that quantitative comparisons of mutagenicity can be made via the Ames assay. Mutagenicity was much more sensitive than antitumor activity to the electronic effect of substituents, but the reason for this is not clear. The differentiated electronic effects may be related to the bacterial DNA that is presumably being attacked. In mice, both gross toxicity and antitumor activity showed the same dependence on the electronic effects

of substituents. Among the triazenes, the species attacking DNA in mutagenesis or antileukemic activity is shown to be a carbonium ion. By manipulating the electronic and lipophilic character of the substituents, much could be done to decrease mutagenicity without destroying the pharmaceutical properties of a drug. Aflatoxin B₁ was about 10⁷ times (on the scale used) more mutagenic than DTIC. (20 refs)

- 79-3374 Multiple Tumor Types Induced with the Trichomonacide Drug Flagyl in Rats (Meeting Abstract). (Eng) Rustia, M. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105); Shubik, P.; Patil, K.; Clayson, D. *Proc Am Assoc Cancer Res* 20: 54; 1979 (no refs)

- 79-3375 Carcinogen-macromolecular Adducts in Liver Subcellular Fractions During Aflatoxin (AFB₁) Feeding (Meeting Abstract). (Eng) Mainigi, K. D. (Div. Nutritional Sciences, Cornell Univ., Ithaca, NY 14853); Campbell, T. C. *Proc Am Assoc Cancer Res* 20: 50; 1979 (no refs)

- 79-3376 Distribution and Time Course of Aflatoxin B₁ Binding in Rat Liver Nuclei and Identification of Histone 1 as a Major Site of Aflatoxin B₁ Adduction to Rat Liver Chromosomal Proteins In Vivo (Meeting Abstract). (Eng) Groopman, J. (Massachusetts Inst. Technology, Cambridge, MA 02139); Busby, W. *Proc Am Assoc Cancer Res* 20: 182; 1979 (no refs)

- 79-3377 Long-Term Studies on the Carcinogenicity of Aflatoxin B₁ (AFB₁) in Non-Human Primates (Meeting Abstract). (Eng) Sieber, S. M. (NIH, Bethesda, MD 20014); Correa, P.; Dalgard, D. W.; Adamson, R. H. *Proc Am Assoc Cancer Res* 20: 82; 1979 (no refs)

- 79-3378 Further Studies on the Mechanism of Aflatoxin B₁ Inhibition of Rat Liver Nuclear RNA Synthesis (Meeting Abstract). (Eng) Yu, F. L. (Thomas Jefferson Univ., Philadelphia, PA 19107). *Proc Am Assoc Cancer Res* 20: 123; 1979 (1 ref)

- 79-3379 Activation of Hepatic Carcinogens by Purified Rat Liver Mitochondria (Meeting Abstract). (Eng) Niranjan, B. G. (Dept. Animal Biology, Sch. Veterinary Medicine, Univ. Pennsylvania, Philadelphia,

PA 19104); Avadhani, N. G. *Proc Am Assoc Cancer Res* 20: 282; 1979 (no refs)

79-3380 Cell Mediated Activation of Aflatoxin B₁ to Transform C3H/10T1/2 Cells (Meeting Abstract). (Eng) Mondal, S. (Univ. Southern California Comprehensive Cancer Center, Los Angeles, CA 90033); Lillehaug, J. R.; Heidelberger, C. *Proc Am Assoc Cancer Res* 20: 62; 1979 (3 refs)

79-3381 Effect of Dietary Dieldrin on Aflatoxin B₁ Carcinogenesis in Rainbow Trout (*Salmo gairdneri*). (Eng) Hendricks, J. D. (Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR 97331); Putnam, T. P.; Sinnhuber, R. O. *J Environ Pathol Toxicol* 2(3): 719-728; 1979.

The effects of the cyclodiene pesticide dieldrin on the mixed-function-oxidase system and on aflatoxin B₁ (AFB₁) carcinogenesis were determined in rainbow trout. Six-week-old trout were fed four semipurified diets for 12 mo. The four diets were formulated as follows: control diet (CD), CD + 6 ppb AFB₁, CD + 5 ppm dieldrin, and CD + 6 ppb AFB₁ + 5 ppm dieldrin. Samples were taken at 1, 2, 4, 6, 9, and 12 mo to determine growth, dieldrin accumulation, tumor incidence, and histopathology. Growth rate was depressed by 5 ppm dieldrin and to a greater extent by 6 ppb AFB₁. The diet containing both compounds produced growth similar to the 5-ppm dieldrin diet. Dieldrin accumulated rapidly during the first 2 mo of feeding to approx 0.9 ppm (whole-fish basis). The rate of accumulation was slower over the following 10 mo, resulting in tissue levels of approx 1.6 ppm at 12 mo. Dieldrin exerted a slight, perhaps questionable, cocarcinogenic effect with AFB₁. The 12-mo incidence of hepatocellular carcinomas was 8% higher in the AFB₁ + dieldrin diet group than in the AFB₁-positive controls; however, the significance of this increase was low ($p < 0.1$). The number of tumors/liver and tumor diameters were both slightly greater in fish fed the AFB₁ + dieldrin diet, but neither parameter was significant due to high variability. No tumors were seen in fish fed either the CD or the diet containing dieldrin alone. No histopathology was produced by this level of dieldrin. (23 refs)

79-3382 Metabolism of Aflatoxin B₁ by Cultured Human Tissues (Meeting Abstract). (Eng) Autrup, H. (Human Tissue Studies Section, Lab. Experimental Pathology, NCI, Bethesda, MD 20014); Essigmann, J. M.; Trump, B. F.; Wogan, G. N.; Harris, C. C. *Proc Am Assoc Cancer Res* 20: 3; 1979 (no refs)

79-3383 Identification of an Aflatoxin P₁-DNA Adduct Formed In Vivo in Rat Liver (Meeting Abstract). (Eng) Croy, R. (Massachusetts Inst. Technology, Cambridge, MA 02139); Wogan, G. *Proc Am Assoc Cancer Res* 20: 182; 1979 (no refs)

79-3384 Aflatoxin B₁ and Aflatoxicol Metabolism in Rainbow Trout (*Salmo gairdneri*) and the Effects of Dietary Cyclopropene. (Eng) Loveland, P. M. (Dept. Food Science and Technology, Oregon State Univ., Corvallis, OR 97331); Nixon, J. E.; Pawlowski, N. E.; Eisele, T. A.; Libbey, L. M.; Sinnhuber, R. O. *J Environ Pathol Toxicol* 2(3): 707-718; 1979.

The results of continuing work on the metabolism of aflatoxin B₁ in rainbow trout (*Salmo gairdneri*) are reported. Aflatoxin M₁ (AFM₁) was identified as a minor metabolite formed by the in vitro incubation of AFB₁ with the postmitochondrial fraction or microsomes from rainbow trout liver. Fresh, whole trout liver converted perfused AFB₁ to aflatoxicol (AFL) and AFM₁, and converted perfused AFL to AFB₁ and AFM₁. AFB₁ reductive activity was found in the 105,000 x g supernatant, but activity converting AFL to AFB₁ was distributed about equally in the 105,000 x g supernatant and in the microsomal pellet. Transformation of AFL to AFB₁ was not inhibited by CO, but formation of AFM₁ was completely blocked. Fish fed cyclopropene fatty acids (CPFA) produced only one-half to one-third as much AFL from AFB₁ as controls, and they produced no detectable AFM₁. Most of the unreacted AFB₁ was recovered by extraction of the incubation medium by organic solvent, whereas in controls, much of it remained bound to the protein. There was no difference in conversion of AFL to AFB₁ when results were expressed in terms of postmitochondrial protein levels. CPFA-fed fish had lower microsomal protein and cytochrome P-450 levels and lower NADPH-cytochrome c reductase and aldrin epoxidation activities than did controls. (22 refs)

79-3385 Effects of the Aflatoxins on ATPase Activities in Mouse and Rat Liver. (Eng) Desai, D. (Dept. Pharmacology and Toxicology, Univ. Mississippi Medical Center, Jackson, MS 39216); Phillips, T. D.; Hayes, A. W.; Ho, I. K. *J Environ Sci Health [B]* 14(3): 265-278; 1979.

The effects of the aflatoxins on ATPase activities in Male ICR mouse and male Sprague-Dawley rat tissues were investigated in vitro. The hepatic oligomycin-sensitive (OS) Mg⁺⁺ ATPase was inhibited significantly. The order of inhibition was aflatoxins G₁ > B₁ > G₂ > B₂. Mouse OS Mg⁺⁺ ATPase was more sensitive than the corresponding rat enzyme. The oligomycin-insensitive (OI) Mg⁺⁺ ATPase activities in rat and mouse liver were not altered. Although aflatoxin G₁ (AFG₁) and aflatoxin B₁ (AFB₁) were the more

potent inhibitors of hepatic OS Mg^{++} ATPase, no concentration-response was observed. In contrast, AFG₁ and AFB₂ inhibited enzyme activity in a concentration-dependent manner. Spectral analysis of AFG₁ solns suggested that solubility was not related to the observed effects. In addition, the effects of AFB₁ and AFG₁ on mouse brain microsomal Na^+-K^+ ATPase were examined. Although AFB₁ was more potent than AFG₁, both mycotoxins significantly inhibited enzyme activity in a concentration-dependent fashion. (26 refs)

- 79-3386 Antibacterial Activity of Zearalenone.** (Fre) Boutibonnes, P. (Laboratoire de Physiologie bacterienne II, Universite de Caen, Caen, France). *Can J Microbiol* 25(3): 421-423; 1979.

The antibacterial activity of zearalenone was limited to some gram-positive aerobic sporulating bacteria, especially *Bacillus thuringiensis* (Berliner). In this species, zearalenone decreased cell division and induced atypical cells. These effects resemble those seen with aflatoxin B₁ and patulin. (12 refs)

- 79-3387 Foreign DNA Sequences in Crown Gall Teratoma and Their Fate During the Loss of the Tumorous Traits (Meeting Abstract).** (Eng) Nester, E. W. (Univ. Washington, Seattle, WA); Yang, F.; Chilton, M. D.; Gordon, M. P. *In Vitro* 15(3): 166-167; 1979 (no refs)

- 79-3388 Attempts at High Frequency Transformation of Plant Cells (Meeting Abstract).** (Eng) Dellaporta, S. L. (Iowa State Univ., Ames, IA 50010); Giles, K. L. *In Vitro* 15(3): 190; 1979 (no refs)

- 79-3389 Bioassay of Emetine for Possible Carcinogenicity.** (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (43): 1-108; 1978.

The possible carcinogenicity of emetine, an amebicide and anticancer drug, was investigated in rats and mice. Groups of 35 Sprague-Dawley rats of each sex were inoculated ip with 0.5 or 1 mg/kg emetine 3x/wk for 52 wk. Groups of 35 B6C3F1 mice of each sex were treated with 1.6, 3.2, or 6.4 mg/kg emetine 3x/wk for 52, 52, and 28 wk (males) and for 52, 40, and 33 wk (females), respectively, with increasing dose. The high-dose male rats were observed for 31 wk and all other rats for 32 wk after treatment, with only 9 high-dose males surviving to the end of the study com-

pared with 32 low-dose males, 29 low-dose females, and 24 high-dose females. Increased mortality was even more marked in mice: only 14 low-dose males and 8 low-dose females survived to the end of the 26-wk posttreatment observation period; only 2 mid-dose males survived >52 wk; and all high-dose males, mid-dose females, and high-dose females died during treatment. There was a variety of tumors in the treated rats, but none occurred at a statistically significant incidence in comparison with the controls. In male rats, tumor incidences were 13/27, 5/35, and 3/10, respectively, in the low-dose, high-dose, and control rats examined histopathologically; in females, these incidences were 26/35, 22/35, and 7/10. The mid-dose and high-dose male mice and mid-dose female mice died before the appearance of tumors. There were 7/35 and 6/48 high-dose and control tumor-bearing male mice, respectively, and 2/34, 1/27, and 3/50 low-dose, high-dose and control tumor-bearing female mice, respectively; there was no statistical significance to the differences among these groups. Because the treatment and observation periods were shorter than those usually employed in bioassays of this type and because of the poor survival among the treated mice, it is concluded that the results do not allow evaluation of the possible carcinogenicity of emetine. (23 refs)

- 79-3390 Bioassay of Ethionamide for Possible Carcinogenicity.** (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (46): 1-86; 1978.

To evaluate the carcinogenicity of ethionamide (EA), a synthetic antitubercular drug, chronic feeding studies were carried out in 41-day-old Fischer 344 rats and 70-day-old B6C3F1 mice following subchronic feeding studies in male Sprague-Dawley rats and Swiss mice to establish max tolerated doses. Groups of 34 or 35 rats of each sex were fed 1,500 or 3,000 ppm EA 5 days/wk for 78 wk, and groups of 34 or 35 mice of each sex were fed 1,000 or 2,000 ppm EA 5 days/wk for 78 wk. All animals were observed for an additional 25-26 wk and were killed and necropsied when moribund or at the end of the experiment. Only animals surviving >100 days were evaluated. The only toxic sign in the animals was a lower mean body wt compared with that of controls. A variety of neoplasms was observed in both the treated and control rats of both sexes, but they were types commonly found in Fischer 344 rats. None of the tumor incidences in treated rats was statistically significant compared with control levels. The incidences of malignant lymphomas in mice were 8/34 and 4/34 in low- and high-dose males, respectively, and 4/31 and 10/34 in low- and high-dose females, respectively. They were slightly but not significantly higher than control levels (2/15 and 2/15 for males and females, respectively). There was a variety of other neoplasms in both experimental and control mice with no significant differences between the groups. It is

concluded that, under the conditions of this bioassay, EA was not carcinogenic in either Fischer 344 rats or B6C3F1 mice. (13 refs)

79-3391 Bioassay of Thio-TEPA for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (58): 1-168; 1978.

The carcinogenicity of thio-TEPA (triethylene thiophosphoramide) was studied in Sprague-Dawley rats and B6C3F1 mice. The animals were inoculated ip with 0.7, 1.4, or 2.8 mg/kg (rats) or 1.15 or 2.3 mg/kg (mice) 3x/wk for a max of 52 wk. The experiments were terminated at 81-87 wk (rats) or at 86-87 wk (mice). Thio-TEPA was toxic to both species, causing reduced wt gain and early deaths in the middle-dose (MD) and high-dose (HD) rats and HD mice. Since all HD rats died by 21 wk, microscopic examination of the tissues was performed only in the low-dose (LD) and MD groups. The tumor incidence rates were time-adjusted. Under these conditions, thio-TEPA was carcinogenic in both species at all evaluable doses. Rats had a significantly increased frequency of squamous cell carcinomas (SCC) of the skin or ear canal (both sexes) and hematopoietic neoplasms (males). Mice showed a significantly increased frequency of SCC of the skin and associated glands (males) and lymphocytic leukemia or lymphomas (both sexes). Uterine adenocarcinomas were also increased in female rats (7/21 MD and 2/33 LD rats; none in controls). Treated rats also showed a non-significantly increased frequency of neuroepitheliomas (neuroblastomas) or nasal carcinomas (3/34 LD males, 2/33 LD females, and 2/21 MD females). Since no neuroepitheliomas or nasal carcinomas were seen in 380 control Sprague-Dawley rats of both sexes in other bioassays, they may be associated with administration of thio-TEPA. It is concluded that, under the conditions of this bioassay, thio-TEPA was carcinogenic in both Sprague-Dawley rats and B6C3F1 mice. (20 refs)

79-3392 Bioassay of Estradiol Mustard for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (59): 1-116; 1978.

The carcinogenicity of the experimental anticancer agent estradiol mustard (EM) was investigated in groups of 35 Sprague-Dawley rats and 34-36 B6C3F1 mice of each sex. The animals received 0.62 or 1.25 mg/kg (rats) or 15 or 30 mg/kg (mice) by gavage 3x/wk for 52 wk. They were observed for an additional 30-34 wk, except that the high-dose (HD) female mice were all dead or sacrificed by 24 wk after the end of treatment. Survival was adequate in all

groups for animals to be at risk for late-developing tumors; however, the increased mortality after 40 wk in the HD male and female mice may have limited late tumor development. Survival in the low-dose (LD) and HD animals, respectively, at the end of the study was 32 and 26 male rats, 25 and 21 female rats, 12 and 9 male mice, and 12 and 0 female mice. In mice but not in rats, there were some statistically significant tumor incidences in each test group compared with controls: lymphoma and lymphocytic leukemia (6/32 vs 0/28), lung adenoma and carcinoma (12/30 vs 2/28), and myocardial sarcoma (6/30 vs 0/28) in LD males; lymphoma and lymphocytic leukemia (17/29 vs 0/28) in HD males; lymphoma (9/30 vs 1/30), lung adenoma and carcinoma (7/27 vs 1/28), and myocardial sarcoma (8/27 vs 0/28) in LD females; and lymphoma (1/23 vs 1/30) in HD females. Also, squamous cell carcinoma of the stomach occurred in 2/29 HD male mice and 2/26 LD and 2/14 HD female mice but it was absent in all controls and in >500 male and >500 female historical-control mice, indicating that these gastric tumors were related to EM. The overall tumor incidences in LD, HD, and control animals, respectively, were 10/35, 10/33 and 9/19 male rats, 27/34, 26/33, and 12/18 female rats, 24/32, 23/29, and 10/29 male mice, and 25/30, 20/24, and 2/30 female mice. It is concluded that, under the conditions of this bioassay, EM was not carcinogenic in Sprague-Dawley rats but was carcinogenic in both male and female B6C3F1 mice. (20 refs)

79-3393 Bioassay of Phenoxybenzamine Hydrochloride for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (72): 1-90; 1978.

The carcinogenicity of the antihypertensive agent phenoxybenzamine hydrochloride (PBH) was investigated in groups of 35 Sprague-Dawley rats and B6C3F1 mice of each sex. The animals were inoculated ip with 5 or 10 mg/kg (rats or 12.5 or 25 mg/kg (mice) 3x/wk for 52 wk. All the high-dose (HD) male mice were dead by 50 wk, with 39 wk being the median survival time (MST). All the HD female mice died by 54 wk (MST 43 wk). The low-dose (LD) mice were observed until 83-84 wk, with 19 males and 25 females being alive at the end of the study. All HD male rats died by 58 wk (MST 43 wk); LD males had a MST of 69 wk, with 13 being alive at the end of the study (83 wk). HD and LD female rats were observed until 83-84 wk, with 23 and 12, respectively, being alive at the end of the study. In rats, the incidence of peritoneal sarcoma (PS) was significant in all but the LD females, being 11/31, 16/20, and 16/30 in LD and HD males and HD females, respectively; PS did not occur in controls. The incidence of PS was also significant in HD mice: 17/21 males and 16/33 females had this lesion, but it did not occur in controls. Other tumors occurred in both treated and control animals,

but none were found at significant incidences in dosed animals in comparison with controls. Overall tumor incidences for LD, HD, and control animals, respectively, were 15/31, 16/20, and 5/20 male rats, 20/35, 20/30, and 12/19 female rats, 7/30, 18/21, and 10/29 male mice, and 7/33, 16/20 (all PS's) and 9/28 female mice. It is concluded that, under the conditions of this bioassay, PBA was carcinogenic for both sexes of Sprague-Dawley rats and B6C3F1 mice, causing PS's. (18 refs)

79-3394 Report on the Bioassay of Chlorobenzilate for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (75): 1-45; 1978.

The carcinogenicity of the pesticide chlorobenzilate (CB) was investigated in chronic feeding studies in groups of 50 Osborne-Mendel rats and B6C3F1 mice of each sex. The rodents were fed CB either continuously for 78 wk or continuously for a certain period and then in a pattern of 1 wk off followed by 4 wk on for varying lengths of time (discontinuous dosing); the total treatment time was 72-78 wk. Male rats received 1,600 ppm continuously or a time-weighted av dose (TWAD) of 2,995 ppm discontinuously for 73 wk; female rats received 1,175 ppm continuously or a TWAD of 2,229 ppm discontinuously for 74 wk; survivors were sacrificed at 110-111 wk. Male mice received TWAD's of 4,231 ppm for 78 wk or 7,846 ppm discontinuously for 72 wk; female mice received 3,200 ppm continuously or 5,908 ppm discontinuously for 72 wk; survivors were sacrificed at 90-91 wk. Survival was adequate in all low-dose (LD) and high-dose (HD) groups, with 29 and 34 male rats, 38 and 36 female rats, 34 and 41 male mice, and 43 and 44 female mice being alive, respectively, at the termination of each study. Tumor incidences in the LD, HD, and control groups were 31/50, 23/50, and 28/49 male rats, 38/48, 34/48, and 36/50 female rats, 34/47, 25/45, and 5/17 male mice, and 18/49, 15/50, and 5/20 female mice. The only statistically significant finding, compared with controls, was the higher incidence of adrenal cortical adenomas in LD males (7/50 vs 0/49) and HD females (5/48 vs 0/50). In mice, only the incidence of hepatocellular carcinoma was statistically significant, compared with controls (4/17 males and 0/20 females), in the LD and HD (32/47 males, 11/49 females and 22/45 males, 13/50 females, respectively). It is concluded that, under the conditions of this bioassay, CB was carcinogenic in male and female B6C3F1 mice but that insufficient evidence was obtained to demonstrate the carcinogenicity of CB in Osborne-Mendel rats. (17 refs)

79-3395 Bioassay of ICRF-159 for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer

Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (78): 1-100; 1978.

The experimental anticancer drug ICRF-159[(±)bis-4,4'-(1-methyl-1,2-ethanediyl)-2,6-piperazinedione] was tested for carcinogenicity in Sprague-Dawley rats and B6C3F1 mice. The animals were inoculated ip with 48 or 96 mg/kg (rats) or 40 or 80 mg/kg (mice) 3x/wk for 52 wk. The experiment was terminated at 81-86 wk in rats and 86 wk in mice. Mortality was dose-related in male mice and in rats of both sexes. The high mortality rate in male rats may have been associated with inflammatory diseases of the pleural and peritoneal cavities, lungs, and liver. The tumor incidence rate was time-adjusted for male rats. Under these bioassay conditions, ICRF-159 caused a dose-related increase in the occurrence of tumors in female rats (uterine adenocarcinomas) and female mice (lymphomas), but it was not significantly carcinogenic in males of either species. (17 refs)

79-3396 Bioassay of Trimethylphosphate for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (81): 1-98; 1978.

The carcinogenicity of trimethyl phosphate (TMP), an alkylating agent used as a gasoline additive, was investigated in groups of 50 Fischer 344 rats and groups of 49-50 B6C3F1 mice of each sex. TMP was administered 3x/wk by gavage in distilled water at doses of 50 or 100 mg/kg (rats) and 250 or 500 mg/kg (mice) for 103-104 wk. Rats were observed 1 wk beyond the end of dosing, mice only to the end of dosing. Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. All rats survived at least 52 wk, and at 105 wk there were 28 and 17 males and 36 and 27 females alive in the low-dose (LD) and high-dose (HD) groups, respectively. At 103 wk, the respective figures for mice were 44 and 39 males and 31 and 29 females. The only statistically significant tumor incidences in dosed animals in comparison with controls were those of sc fibromas in HD male rats (9/49 vs 0/20) and of uterine/endometrial adenocarcinomas in female mice (13/37 vs 0/16). The overall tumor incidences in LD, HD, and control animals, respectively, were 48/50, 43/49, and 19/20 male rats, 40/50, 42/49, and 15/20 female rats, 26/50, 26/49, and 11/20 male mice, and 28/50, 30/49, and 11/20 female mice. It is concluded that, under the conditions of this bioassay, TMP was carcinogenic in male Fischer rats and female B6C3F1 mice, but not carcinogenic in female Fischer rats or male B6C3F1 mice. (24 refs)

79-3397 Bioassay of Daminozide for Possible Carcinogenicity. (Eng) National Cancer In-

stitute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (83): 1-114; 1978.

The carcinogenicity of the plant growth regulator daminozide (DOZ) was investigated in 104-wk feeding studies using groups of 50 Fischer 344 rats and B6C3F1 mice of each sex given doses of 5,000 or 10,000 ppm. The experiment was terminated at 105 wk by sacrifice of the survivors in the low-dose (LD) and high-dose (HD) groups, which were, respectively, 37 and 44 male rats, 35 and 39 female rats, 38 and 32 male mice, and 32 and 30 female mice. Survival in all groups was adequate for the animals to be at risk for late-developing tumors. In the male rats, only the incidence of testicular interstitial cell tumors was significantly higher than that in the controls, but because these lesions occur at a high spontaneous rate in this strain, the result is probably not attributable to DOZ. In female rats, the incidences of endometrial adenocarcinomas and uterine leiomyosarcoma in the LD and HD groups (5/50 and 3/50; 1/50 and 3/50, respectively) were below statistical significance in comparison with controls, but because these lesions were absent in the controls and occurred with low incidence in historical controls, they are probably associated with DOZ. Although the incidence of hepatocellular carcinoma in HD male mice (13/46) was statistically significant compared with controls, there was a high incidence of this lesion in historical-control rats, so that its appearance in the test rats is not clearly associated with DOZ. There was a large variety of tumors in all test animals, but none of the other results was statistically significant compared with controls. Tumor incidences in LD, HD, and control animals examined histopathologically were 50/50, 49/50, and 16/20 male rats, 32/50, 28/50, and 11/20 female rats, 28/50, 37/46, and 8/14 male mice, and 27/41, 25/48, and 13/20 female mice. It is concluded that, under the conditions of this bioassay, DOZ was not carcinogenic in male Fischer 344 rats or female B6C3F1 mice, that it induced endometrial adenocarcinomas and uterine leiomyosarcomas in female Fischer 344 rats, and that it may have been associated with the hepatocellular carcinomas in male B6C3F1 mice. (18 refs)

79-3398 Bioassay of 2,4-Diaminoanisole Sulfate for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (84): 1-69; 1978.

The carcinogenicity of 2,4-diaminoanisole (DAA), a hair-dye component, was investigated by chronic feeding studies in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex. DAA was mixed into the diet at a concentration of 0.12% or 0.5% (rats) or 0.12% or 0.24% (mice) for 78 wk, followed by observation periods of 29 wk (rats) and 18-19 wk (mice). Five animals from each test group except the low-dose (LD) male and female mice were sacrificed in

week 78; survival in all dosed groups was adequate for animals to be at risk for late-developing tumors. At 106-107 wk, survival in the LD and high-dose (HD) groups, respectively, was 30 and 27 female rats, 29 and 22 female rats, 46 and 41 male mice, and 38 and 39 female mice. None of the tumor incidences in any LD group was statistically significant compared with controls, except that of malignant lymphoma in LD female mice (14/45 vs 5/48). In the HD male rats there were several significant results in comparison with controls: thyroid C-cell adenoma or carcinoma (10/49 vs 1/48), thyroid carcinoma (14/49 vs 0/48), thyroid papillary adenocarcinoma, follicular cell carcinoma, or papillary cystadenoma (17/49 vs 0/48), preputial gland neoplasms (8/49 vs 0/48), Zymbal's gland neoplasms (8/49 vs 0/48), and skin squamous cell or basal cell carcinoma or sebaceous adenocarcinoma (7/49 vs 0/48). Among HD female rats, the statistically significant tumor incidences were those of thyroid adenocarcinoma, follicular cell carcinoma, or papillary cystadenocarcinoma (10/49 vs 1/45), and Zymbal's gland sebaceous adenocarcinoma (7/49 vs 0/50). In HD male and female mice, only the incidence of thyroid follicular cell adenoma was statistically significant when compared with controls (11/45 vs 0/40 in males, 8/45 vs 0/41 in females). In the HD female mice, the incidence of malignant lymphoma (9/50) was marginally significant compared with controls (2/45). The overall tumor incidences in the LD, HD, LD control, and HD control animals, respectively, were 48/48, 47/49, 34/46, and 44/48 male rats, 44/49, 37/49, 32/49, and 38/50 female rats, 24/49, 33/49, 17/48, and 22/49 male mice, and 25/44, 30/50, 20/47, and 10/50 female mice. It is concluded that, under the conditions of this bioassay, DAA was carcinogenic to both sexes of Fischer 344 rats and B6C3F1 mice. (29 refs)

79-3399 Bioassay of 4-Chloro-m-phenylenediamine for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (85): 1-46; 1978.

The carcinogenicity of 4-chloro-m-phenylenediamine (CMPD), a dye intermediate, was investigated by chronic feeding studies in groups of 49-50 Fischer 344 rats and B6C3F1 mice of each sex. The animals received 0.2% or 0.4% (rats) or 0.7% or 1.4% (mice) CMPD in their diets for 78 wk and were observed for an additional 26-27 wk (rats) or 17 wk (mice). Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. Five animals in each high-dose (HD) group were sacrificed in week 78. At the end of the study, survival in the low-dose (LD) and HD groups, respectively, was 43 and 36 for male rats, 39 and 33 for female rats, 41 and 40 for male mice, and 37 and 38 for female mice. The only statistically significant tumor incidence in rats was that of adrenal pheochromocytoma in HD male rats (14/48) compared with controls (4/46). In LD female rats, the in-

cidence of uterine endometrial stromal polyps (12/48) was statistically significant compared with controls (2/48), but it was lower than that in HD females (5/46) or historical controls (31/249), so that this result was not attributed to CMPD. None of the tumor incidences in male mice was statistically significant compared with controls, but in LD and HD female mice, the combined incidence of hepatocellular adenomas and carcinomas (11/44 and 8/45, respectively) was significant compared with controls (0/46). The overall tumor incidences in the LD, HD, and control groups, respectively, were 46/49, 46/49, and 42/48 male rats, 33/50, 30/47, and 27/50 female rats, 19/49, 25/48, and 22/50 male mice, and 20/44, 16/46, and 14/47 female mice. It is concluded that, under the conditions of this bioassay, CMPD was carcinogenic to male Fischer 344 rats and female B6C3F1 mice, but not to female Fischer 344 rats or male B6C3F1 mice. (21 refs)

79-3400 Bioassay of 1H-Benzotriazole for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (88): 1-116; 1978.

The carcinogenicity of 1H-benzotriazole (HBT), an anticorrosive chemical, was investigated by chronic feeding studies in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex. Time-weighted av concentrations of HBT were administered in the diets by the following dose schedules: 6,700 or 12,100 ppm for 78 wk (rats), and 11,700 or 23,500 ppm for 104 wk (mice). Rats were observed for an additional 26-27 wk, mice for an additional 2 wk. Survival was adequate in all groups for the animals to be at risk for late-developing tumors. At the end of the study, survival in the low-dose (LD) and high-dose (HD) groups, respectively, was 34 and 36 for male rats, 40 and 43 for female rats, 36 and 42 for male mice, and 39 and 47 for female mice; 5 rats in each HD group were sacrificed in week 78. None of the tumor incidences in rats or mice was statistically significant compared with test and/or historical controls. In HD male rats, the incidence of neoplastic liver nodules (5/45) was significantly higher than that in the controls (0/48), but 2/13 historical controls had this tumor. In LD female mice, the incidence of alveolar/bronchiolar carcinomas and the combined incidence of alveolar/bronchiolar adenomas and carcinomas (9/49 and 10/49) were significant compared with controls (0/49), but the incidence in the HD group was not significant and the incidence in historical controls varied from 0 to 7% (av: 4%), so that these tumors could not be clearly related to HBT. The overall tumor incidences in LD, HD, and control groups, respectively, were 43/46, 40/46, and 42/48 male rats, 27/48, 30/50, and 27/50 female rats, 23/44, 18/48, and 24/39 male mice, and 25/49, 14/50, and 21/49 female mice. It is concluded that, under the conditions of this bioassay, there was no convincing evidence that HBT was carcinogenic in Fischer 344 rats or B6C3F1 mice of either sex. (25 refs)

79-3401 Bioassay of o-Anisidine Hydrochloride for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (89): 1-130; 1978.

The carcinogenicity of o-anisidine (OA), which is used in dye manufacture, was investigated by chronic feeding studies in groups of 55 Fischer 344 rats and B6C3F1 mice. OA was administered in the diets at doses of 5,000 or 10,000 ppm for 83-103 wk (rats) or 2,500 or 5,000 ppm for 103 wk (mice); mice and low-dose (LD) rats were observed or an additional 1-2 wk. Considerable dose-related mortality occurred in rats: all high-dose (HD) males and females were dead by 83 and 88 wk, respectively, only 7 LD males were alive at 104 wk, and the last LD female died at 103 wk. Survival in mice was adequate for the animals to be at risk for late-developing tumors. In rats, the incidence of transitional cell carcinoma of the bladder and the combined incidence of transitional cell carcinoma and papilloma of the bladder were statistically significant in all groups compared with controls; the respective figures were 50/54, 52/54, and 0/51 in LD males, 51/52, 52/52, and 0/51 in HD males, 41/49, 46/49, and 0/49 in LD females, and 50/51, 50/51, and 0/49 in HD females. In LD and HD male rats, the incidences of thyroid follicular cell tumors (7/40 and 6/40, respectively) and of thyroid adenomas, cystadenomas, or papillary adenomas (4/40 and 4/40, respectively) were statistically significant compared with controls (0/53 and 0/53, respectively). In HD male rats, the incidence of kidney or kidney pelvis transitional cell carcinomas (7/53) was statistically significant compared with controls (0/53). None of the tumor incidences in LD male or female mice was statistically significant in comparison with controls, but in the HD males and females, the combined incidence of transitional cell carcinomas or papillomas of the bladder (22/53 and 22/50, respectively) were statistically significant compared with controls (0/48 and 0/50). The overall tumor incidences in LD, HD, and control groups, respectively, were 53/55, 53/53, and 54/54 male rats, 50/53, 51/54, and 52/54 female rats, 27/55, 30/53, and 43/55 male mice, and 20/52, 33/54, and 34/55 female mice. It is concluded that, under the conditions of this bioassay, OA was carcinogenic for Fischer 344 rats and B6C3F1 mice of both sexes. (21 refs)

79-3402 Report on the Bioassay of Dicofol for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (90): 1-46; 1978.

The carcinogenicity of dicofol (DC), a synthetic organochlorine acaricide, was investigated by chronic feeding studies in groups of 50 Osborne-Mendel rats and B6C3F1 mice of each sex. DC, dissolved in corn oil, was mixed with the diet at a time-weighted av concentration of

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471 or 942 ppm in male rats, 380 or 760 ppm in female rats, 264 or 528 ppm in male mice, and 122 or 243 ppm in female mice for 78 wk, followed by 34 wk of observation in rats and 14-15 wk in mice. Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. Survival among low-dose (LD) and high-dose (HD) rats, respectively, at 100 wk was 32 and 36 for males and 46 and 44 for females; the respective figures for mice at 92-93 wk were 38 and 38 for males and 42 and 48 for females. Although there were several different tumors in both the dosed and control groups, the only statistically significant incidence compared with controls was that of hepatocellular carcinoma in the HD male mice (35/47 vs 3/18). The overall tumor incidences in LD, HD, and control animals, respectively, were 23/49, 16/47, and 10/19 male rats, 28/49, 28/49, and 16/20 female rats, 34/48, 38/47, and 5/18 male mice, and 5/20, 6/44, and 8/50 female mice. It is concluded that, under the conditions of this bioassay, DC was carcinogenic in male B6C3F1 mice, but not carcinogenic in female B6C3F1 mice or in male or female Osborne-Mendel rats. (26 refs)

79-3403 Bioassay of Hydrazobenzene for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (92): 1-58; 1978.

The carcinogenicity of hydrazobenzene (HB) were investigated by chronic feeding studies in groups of 50 male and 47-50 female Fischer 344 rats and B6C3F1 mice. The male rats received a time-weighted av concentration of 0.008% or 0.03% HB and the female rats received 0.004 or 0.01% HB in their diets for 78 wk, followed by 28-30 wk of observation before sacrifice. The male mice received a time-weighted av concentration of 0.008% or 0.04% HB and the female mice received 0.004 or 0.04% HB in their diets for 78 wk, followed by 17-18 wk of observation. Survival was adequate in all groups for the animals to be at risk for late-developing tumors. Five rats in each group and five mice in each high-dose group were sacrificed at 78 wk. At 100 wk, the survival in the low- and high-dose rat groups, respectively, was 39 and 32 males and 37 and 25 females; at 95-96 wk, the respective survival in the mouse groups was 44 and 39 males and 37 and 26 females. The only statistically significant tumor incidences in comparison with controls were those of hepatocellular carcinoma in low-dose male rats (5/49 vs 0/47), hepatocellular carcinoma (31/49 vs 1/48) and squamous cell carcinoma of zymbal's gland (5/49 vs 0/48) in high-dose male rats, mammary gland adenocarcinoma (6/50 vs 0/50) and neoplastic liver nodules (6/50 vs 0/50) in high-dose female rats, and hepatocellular carcinoma in high-dose female mice (20/43 vs 1/50). The overall tumor incidences in the low- and high-dose test groups and the low- and high-dose control groups, respectively, for animals examined histopathologically were 48/49, 47/49, 47/47, and 44/48

for male rats, 39/50, 36/50, 42/47, and 38/50 for female rats, 18/47, 17/46, 23/50, and 22/49 for male mice, and 19/39, 29/43, 18/47, and 10/50 for female mice. It is concluded that, under the conditions of this bioassay, HB was carcinogenic to Fischer 344 rats of both sexes and to female B6C3F1 mice, but that it was not carcinogenic to male B6C3F1 mice. (22 refs)

79-3404 Bioassay of Phenazopyridine Hydrochloride for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (99): 1-98; 1978.

To investigate the carcinogenicity of phenazopyridine hydrochloride (PPH), chronic feeding studies were undertaken in groups of 35 Fischer 344 rats and B6C3F1 mice of each sex. The animals received 3,700 or 7,500 ppm (rats) or 600 or 1,200 ppm (mice) in their diets 5 days/wk for 78 wk and were observed until 104-107 wk. Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. The numbers of survivors at the end of the study in the low-dose (LD) and high-dose (HD) groups, respectively, were 25 and 28 male rats, 30 and 28 female rats, 26 and 25 male mice, and 26 and 21 female mice. Adenocarcinomas of the large intestine (colon and rectum) occurred in male and female rats at statistically significant frequencies in comparison with controls (4/34, 9/33, and 0/14 males and 3/33, 5/32, and 0/14 females for LD, HD, and control groups, respectively). The other tumors that were found in dosed rats were not significant in comparison with controls. The overall tumor incidences in the LD, HD, and control rats, respectively, were 29/34, 17/35, and 14/14 males and 10/33, 13/32, and 5/15 females. None of the tumor incidences in male mice was significant compared with controls. In LD female mice there was a dose-related trend in the incidence of hepatocellular adenomas and carcinomas (11/34) compared with controls (1/15), and in the HD females the incidence of these tumors (21/32) became significant. The overall tumor incidences in female mice were significant. The overall tumor incidences in LD, HD, and control mice, respectively, were 17/35, 20/34, and 7/15 male mice and 20/34, 27/32, and 8/15 female mice. It is concluded that, under the conditions of this bioassay, PPH was carcinogenic in Fischer 344 rats, causing adenocarcinomas of the large intestine in males and females, and in female B6C3F1 mice, causing hepatocellular adenomas and carcinomas, but that it was not carcinogenic in male B6C3F1 mice. (17 refs)

79-3405 Bioassay of Anilazine for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (104): 1-99; 1978.

The carcinogenicity of the fungicide anilazine (AAZ) was investigated by chronic feeding studies in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex given 500 or 1,000 ppm AAZ dissolved in a small amount of acetone and added to their diets for 103 wk. Surviving animals were sacrificed at 103-104 wk (rats) or 107-109 wk (mice). Survival was adequate in all groups for the animals to be at risk for late-developing tumors. At 2 yr, survival in the low- and high-dose groups, respectively, was 37 and 37 for male rats, 37 and 34 for female rats, 40 and 44 for male mice, and 33 and 41 for female mice. The survival data and the fact that only the dosed male mice exhibited a decreased gain in mean body wt indicate that the rats and the female mice may have been able to tolerate higher doses of AAZ. There was a variety of tumors in both the experimental and control groups, but none of the tumors in the dosed animals occurred at incidences that were significantly higher than those in the corresponding controls. Respective tumor incidences in the low-dose, high-dose, and control animals that were examined histopathologically were 50/50, 50/50, and 24/25 in male rats, 42/50, 43/50, and 22/25 in female rats, 20/49, 25/50, and 15/23 in male mice, and 22/47, 18/50, and 4/25 in female mice. It is concluded that, under the conditions of this bioassay, AAZ was not carcinogenic in Fischer 344 rats and B6C3F1 mice. (31 refs)

79-3406 Bioassay of m-Cresidine for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (105): 1-54; 1978.

The carcinogenicity of m-cresidine (MC), a dyestuff intermediate, was investigated in groups of 49-50 Fischer 344 rats and B6C3F1 mice of each sex. MC was dissolved in corn oil and administered by gavage 5x/wk: rats received 0.08 or 0.16 g/kg for 77 wk; mice received 0.08 or 0.16 g/kg for 32 wk and then 0.02 or 0.04 g/kg, respectively, for 21 wk (the time-weighted av doses were 0.06 and 0.11 g/kg, respectively). Rats were observed for an additional 32-33 wk, mice for an additional 25-41 wk. Survival was adequate in all groups but the male mice for animals to be at risk for late-developing tumors. At the end of the study, there were 31 and 24 male rats and 28 and 22 female rats alive in the low-dose (LD) and high-dose (HD) groups, respectively, with 5 animals having been sacrificed in each HD group in wk 78. Median survival time in LD and HD male mice, respectively, was 52 and 26 wk; 15 LD mice were alive at 93 wk, and all HD mice were dead by 78 wk. At 94 wk, there were 43 and 30 LD and HD female mice alive, respectively. None of the tumor incidences in mice was statistically significant compared with controls; overall tumor incidences in LD, HD, and pooled controls, respectively, were 10/45, 0/36, and 29/73 males and 10/50, 15/46, and 15/74 females. None of the tumor incidences in rats was statistically significant compared with controls,

but the incidences of papillary transitional-cell carcinomas of the bladder in HD males (5/44), LD females (1/46), and HD females (2/44) compared with pooled controls (0/148) and the rarity of this tumor in historical controls indicate that this tumor was MC-related. The overall tumor incidences in LD, HD, and pooled control groups, respectively, were 41/47, 36/45, and 73/75 males and 38/49, 19/45, and 43/73 females. It is concluded that, under the conditions of this bioassay, MC was carcinogenic to Fischer 344 rats of both sexes, was not carcinogenic to female B6C3F1 mice, and cannot be evaluated for carcinogenicity to male B6C3F1 mice because of their high mortality while receiving the compound. (17 refs)

79-3407 Bioassay of Trichlorofluoromethane for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (106): 1-46; 1978.

The carcinogenicity of trichlorofluoromethane (TCFM), widely used aerosol propellant and refrigerant, was investigated in rats and mice that received TCFM by gavage as a 37.5-42.5% soln (rats) or a 20-40% soln (mice) in corn oil on 5 consecutive days/wk for a total of 78 wk. Groups of 50 Osborne-Mendel rats and B6C3F1 mice of each sex were dosed as follows: 425 or 850 mg/kg/day for 12 wk followed by 500 or 1,000 mg/kg/day, respectively, for 66 wk in male rats; 750 or 1,500 mg/kg/day for 12 wk followed by 500 or 1,000 mg/kg/day, respectively, for 66 wk in female rats; 1,580 or 3,160 mg/kg/day for 7 wk followed by 2,000 or 4,000 mg/kg/day, respectively, for 71 wk in both male and female mice. The time-weighted av doses in male and female rats were 488 and 977 and 538 and 1,077 mg/kg/day, respectively, those in mice were 1,962 and 3,925 mg/kg/day. Survival in rats was inadequate for the animals to be at risk for late-developing tumors; only 20 and 15 males and 31 and 17 females in the low- and high-dose groups, respectively, were alive at 52 wk, and <10% of the rats in any group survived to the end of the test (111 wk, except that the low-dose male test ended at 106 wk). Hence the rat tumor data are inconclusive; tumor incidences in the low-dose, high-dose, and control groups for animals examined histopathologically were 4/50, 1/50, and 11/40 in males and 4/49, 7/50, and 16/40 in females. The survival of the treated mice was adequate to evaluate risk from late-developing tumors; at the end of the study (91 wk), the respective low- and high-dose group survival was 41 and 29 in male and 37 and 32 in females. There was a variety of tumors in both treated and control mice, but the tumors in the dosed mice did not occur at significant incidences compared with controls. The respective tumor incidences in low-dose, high-dose, and control mice examined histopathologically were 22/50, 16/47, and 11/37 in males and 11/50, 9/49, and 6/37 in females. No conclusion can be drawn with regard to the carcinogenicity of TCFM in Osborne-Mendel rats. It is concluded that, under the

conditions of this bioassay, TCFM was not carcinogenic to male or female B6C3F1 mice. (31 refs)

79-3408 Report on the Bioassay of 5-Nitro-o-Toluidine for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (107): 1-50; 1978.

The carcinogenicity of the azo dye intermediate 5-nitro-o-toluidine (NT) was investigated by chronic feeding studies in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex. The rodents received a time-weighted av dose of 0.005% or 0.01% (rats) or 0.1% or 0.12% (mice) in their diets for 78 wk and were observed for an additional 30-31 wk (rats) or 19-20 wk (mice). Five animals in each group were sacrificed at 78-79 wk; survivors were sacrificed at the end of the observation periods. Adequate numbers in all test groups survived to the end of the study for the animals to be at risk for late-developing tumors. Survivors in the low-dose (LD) and high-dose (HD) groups, respectively, were 27 and 28 male rats, 28 and 33 female rats, 41 and 38 male mice, and 42 and 36 female mice. A variety of tumors occurred in all treated and control animals. None of the tumor incidences in dosed rats were statistically significant compared with controls; tumor incidences in the LD, HD, and control rat groups, respectively, were 37/46, 42/47, and 47/47 males and 33/47, 35/50, and 42/47 females. In mice, only the incidence of hepatocellular carcinoma was statistically significant when the HD groups were compared with controls (29/45 vs 12/50 in males and 20/45 vs 2/47 in females). The combined incidence of hemangiomas and hemangiosarcomas in HD male mice (4/48) and the incidence of hemangiosarcomas in LD female mice (5/47) were considered attributable to NT because, although these incidences were not significant in comparison with the untreated controls, they were significant in comparison with historical controls in the same laboratory (5/350). The overall tumor incidences in the LD, HD, and control mice, respectively, were 20/46, 31/48, and 23/50 males and 19/47, 25/47, and 18/47 females. It is concluded that, under the conditions of this bioassay, NT was not carcinogenic in Fischer 344 rats, but it caused hepatocellular carcinomas in both male and female B6C3F1 mice as well as an increased incidence of hemangiomas and hemangiosarcomas in male B6C3F1 mice and hemangiosarcomas in female B6C3F1 mice. (26 refs)

79-3409 Bioassay of 3-Amino-4-ethoxyacetanilide for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (112): 1-52; 1978.

The carcinogenicity of 3-amino-4-ethoxyacetanilide (AEA), an azo dye intermediate, was investigated by chronic feeding studies in groups of 48 or 50 Fischer 344 rats and B6C3F1 mice. AEA was administered to the animals at 0.4% or 1.5% (rats) or 0.4% or 0.8% (mice) of their diet for 78 wk. Rats were observed for an additional 28-35 wk, mice for an additional 16-18 wk. Survival was adequate in all groups for animals to be at risk for late-developing tumors. At the end of the study, the low-dose (LD) and high-dose (HD) group survival, respectively, was 32 and 41 in male rats, 32 and 37 in female rats, 48 and 38 in male mice, and 48 and 38 in female mice. None of the tumor incidences in rats was statistically significant compared with controls. The only statistically significant result in mice was the higher incidence of follicular cell carcinoma of the thyroid in HD male mice (7/45) compared with controls (0/45). The overall tumor incidences in LD, HD, LD controls, and HD controls, respectively, were 46/46, 46/50, 44/48, and 42/47 male rats, 37/46, 30/49, 38/50, and 42/47 female rats, 28/49, 19/47, 22/49, and 19/46 male mice, and 31/50, 21/47, 10/50, and 17/47 female mice. Many of the confidence intervals in the statistical analyses of tumor incidence in rats have an upper limit >1, indicating a theoretical possibility of tumor induction in rats by AEA that was not established by this test. It is concluded that, under the conditions of this bioassay, AEA was carcinogenic for male B6C3F1 mice, but that there was insufficient incidence to establish carcinogenicity in male or female Fischer 344 rats. (22 refs)

79-3410 Bioassay of 2,3,5,6-Tetrachloro-4-Nitroanisole for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (114): 1-54; 1978.

The carcinogenicity of 2,3,5,6-tetrachloro-4-nitroanisole (TCNA) was investigated in chronic feeding studies in groups of 50 Fischer 344 rats and groups of 55 B6C3F1 mice of each sex. Dose levels, established in prior sub-chronic toxicity tests, were 0.006% or 0.012% for 104 wk. Because the high-dose male rats were incorrectly sexed, a second group of 25 rats was treated at the high dose level for 104 wk to give a total of 49 male rats subjected to this regimen. Survivors were sacrificed 105-107 wk after the initiation of feeding. Survival was adequate in all groups for the animals to be at risk for late-developing tumors; at the termination of the study, there were, in the low- and high-dose groups, respectively, 46 and 38 male rats, 48 and 39 female rats, 49 and 50 male mice, and 47 and 46 female mice. Tumor incidences in the low-dose, high-dose and control animals in each group that were examined histopathologically were 48/49, 43/48, and 42/49 male rats, 34/50, 28/45, and 27/50 female rats, 38/54, 36/52, and 43/55 male mice, and 37/54, 38/52 and 34/55 female mice. The only statistically significant results, compared

with controls, were the incidences of interstitial cell testis tumors in male rats and the combined incidence of leukemia and malignant lymphoma in male mice. However, these lesions were considered to be unrelated to the administration of TCNA because such tumors occur spontaneously in these strains with a high and variable incidence. It is concluded that, under the conditions of this bioassay, dietary administration of TCNA was not carcinogenic in Fischer 344 rats or B6C3F1 mice of either sex. (19 refs)

79-3411 Bioassay of p-Anisidine Hydrochloride for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (116): 1-52; 1978.

To investigate the carcinogenicity of p-anisidine (AD), a chronic feeding study was undertaken in groups of 55 Fischer 344 rats and B6C3F1 mice of each sex, after the max tolerated dose had been determined in a subchronic toxicity test. Rats were fed 0.6% or 0.3% and mice were fed 1.0% or 0.5% AD in their diet for 103 wk, followed by an observation period of 2-3 wk for rats and 3 wk for mice. Adequate numbers of animals in all groups survived sufficiently long to be at risk for late-developing tumors. Animals alive at the end of the experiment consisted of 43 low-dose and 45 high-dose male rats, 43 low-dose and 50 high-dose female rats, 48 low-dose and 50 high-dose male mice, and 42 low-dose and 43 high-dose female mice. There was a variety of tumors in all treated animals and controls. Tumor incidences were 50/54, 50/55, and 54/54 male rats, respectively, in the low-dose, high-dose, and control animals examined histopathologically. Corresponding values were 38/43, 42/50, and 52/54 female rats, 29/54, 36/50, and 43/55 male mice, and 33/54, 24/50, and 34/55 female mice. In male rats, there were significant positive associations between AD administration and the incidences of both squamous cell carcinomas of the skin and alveolar/bronchiolar adenomas, but none of the Fischer exact comparisons supported these findings. The only statistically significant result in male rats was the Fisher exact comparison of the low-dose and control groups with respect to the combined incidence of adenomas and carcinomas of the preputial gland. There were no significant positive associations in any mice between compound administration and any tumor incidence. It is concluded that AD was not carcinogenic in B6C3F1 mice under the conditions of this bioassay, and that the evidence is insufficient to establish the carcinogenicity of AD in Fischer 344 rats. (20 refs)

79-3412 Bioassay of Dioxathion for Possible Carcinogenicity. (Eng) National Cancer In-

stitute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (125): 1-43; 1978.

The carcinogenicity of dioxathion (DX), an organophosphorus insecticide, was investigated in groups of 50 Osborne-Mendel rats and B6C3F1 mice of each sex in chronic feeding studies. The initial dose was increased after 31 wk in rats and after 17 wk in mice so that the time-weighted av dose received over 78 wk was 90 or 180 ppm in male rats, 45 or 90 ppm in female rats, 284 or 567 ppm in male mice, and 467 or 935 ppm in female mice. These doses were selected on the basis of prior subchronic toxicity tests to determine max tolerated doses. Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. When the rat study was terminated at 111 wk, 23 and 25 males and 31 and 34 females were alive in the low- and high-dose groups, respectively. The corresponding figures for the mouse groups at 90-91 wk were 35 and 37 males and 46 and 45 females. A variety of neoplasms was observed in test animals of both species, but none of the tumors was histologically unusual, and no incidence was statistically significant in comparison with controls. Tumor incidences for low-dose, high-dose, and control groups, respectively, were 23/50, 21/49, and 28/49 in male rats, 27/47, 29/48, and 36/50 in female rats, 10/49, 13/49, and 7/19 in male mice, and 6/50, 6/48, and 2/18 in female mice. It is concluded that, under the conditions of this bioassay, dietary administration of DX was not carcinogenic in Osborne-Mendel rats or B6C3F1 mice. (17 refs)

79-3413 Bioassay of 2,5-Toluenediamine Sulfate for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (126): 1-50; 1978.

The carcinogenicity of 2,5-toluenediamine (TDA: sulfate form) was evaluated in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex. The doses used in these chronic feeding studies were determined by the results of prior subchronic toxicity tests. Male rats were fed either 0.05% TDA for 14 wk, followed by 0.06% for 64 wk, or 0.2% for 78 wk and observed for an additional 28 and 30 wk, respectively. Female rats were fed according to the same regimens and observed for an additional 107 or 109 wk, respectively. Male and female mice were fed 0.06% or 0.1% TDA for 78 wk; low-dose animals were observed an additional 16 wk, high-dose animals an additional 19 wk. Survival was adequate in all test animals; 42 and 45 males and 42 and 43 females lived at least 85 wk in the low- and high-dose rat groups, respectively, and 47 and 37 males and 39 and 33 females lived to the end of the study in the low- and high-dose mice groups. Tumor incidence in the low-dose, high-dose, and two control groups, respectively, was 45/48, 48/49, 22/25, and 34/46 in male rats, 34/50, 33/50, 19/23,

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and 32/49 in female rats, 19/48, 27/49, 21/45, and 17/48 in male mice, and 15/43, 22/46, 22/46, and 20/47 in female mice. The incidence of interstitial cell neoplasms of the testis in the male rats (43/48 low-dose and 47/49 high-dose rats) was statistically significant compared with controls (19/25 and 33/46), but it was not attributed to the administration of TDA because the spontaneous incidence of these tumors is high and variable in Fischer 344 rats. None of the results was statistically significant in the female rats, compared with controls. None of the results in test mice was statistically significant compared with controls except for an increased incidence of lung tumor in the high-dose females (8/46 vs 1/45), but this evidence was not considered convincing because the test and control mice were shipped and housed under different conditions. It is concluded that, under the conditions of this bioassay, sufficient evidence was not obtained to demonstrate the carcinogenicity of TDA in Fischer 344 rats or B6C3F1 mice. (23 refs)

79-3414 Bioassay of Aniline Hydrochloride for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (130): 1-55; 1978.

The carcinogenicity of aniline hydrochloride (AH), a dye intermediate, was investigated by chronic feeding studies in groups of 50 Fischer 344 rats and groups of 49-50 B6C3F1 mice of each sex. The animals were fed 0.3% or 0.6% (rats) or 0.6% or 1.2% (mice) AH in their diets for 103 wk and observed for an additional 4-5 wk. Survival was adequate in all groups for the animals to be at risk for late-developing tumors. At the end of the study, survival in the low-dose (LD) and high-dose (HD) groups, respectively, was 34 and 27 in male rats, 44 and 41 in female rats, 43 and 41 in male mice, and 37 and 41 in female mice. There was a variety of tumors in the dosed and control mice, but none of the tumor incidences in dosed mice was statistically significant in comparison with controls. Tumor incidences in LD, HD, and control mice, respectively, were 27/49, 22/49, and 24/39 males and 22/48, 28/49, and 21/49 females. In comparison with controls, the incidence of spleen hemangiosarcoma (19/50 vs 0/25) in LD male rats and the incidences of spleen fibrosarcoma or sarcoma (9/46 vs 0/25), spleen hemangiosarcoma (20/46 vs 0/25), and fibrosarcoma or sarcoma of multiple organs of the body cavities (9/48 vs 0/25) in HD male rats were statistically significant. None of the tumor incidences in LD female rats was statistically significant in comparison with controls, but in HD female rats, the combined incidence of sarcoma and fibrosarcoma of the spleen and of multiple organs of the body cavities (8/50 vs 0/24) was considered dose-related to AH. The overall tumor incidences in LD, HD, and control rats, respectively, were 27/49, 22/49, and 24/39 males and 22/48, 28/49, and 21/49 females. It is concluded that, under the conditions of this

bioassay, AH was carcinogenic to male and female Fischer 344 rats but not to B6C3F1 mice. (20 refs)

79-3415 Report on the Bioassay of Triphenyltin Hydroxide for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (139): 1-41; 1978.

The carcinogenicity of triphenyltin hydroxide (TPTH), a fungicide and insect-controlling compound, was investigated in chronic feeding studies in groups of 50 Fischer 344 rats and B6C3F1 mice of each sex. Animals were fed diets containing 37.5 or 75 ppm TPTH for 78 wk and observed for an additional 26 wk, at which time all survivors were sacrificed. Survival was adequate in all groups for the animals to be at risk for late-developing tumors; the survivors in the low-dose (LD) and high-dose (HD) groups, respectively, at 104 wk were 38 and 42 male rats, 40 and 45 female rats, 37 and 33 male mice, and 36 and 38 female mice. In male mice, there was a significant positive association between dose and mortality. In female mice and male rats, there was a slight depression of mean body wt gain, but no compound-related mean body wt depression was observed. In female rats there were no significant signs of toxicity due to TPTH. Thus, it is possible that TPTH was not administered at the max tolerated doses. There was a variety of tumors in both the control and treated animals, but no tumors occurred at a significantly higher incidence in dosed animals than in controls. The respective tumor incidences in LD, HD, and control animals examined histopathologically were 49/50, 50/50, and 18/20 male rats, 39/50, 31/50, and 12/20 female rats, 19/44, 22/45, and 10/20 male mice, and 15/42, 18/48, and 10/18 female mice. It is concluded that, under the conditions of this bioassay, TPTH was not carcinogenic in Fischer 344 rats or B6C3F1 mice. (17 refs)

79-3416 Bioassay of Pivalolactone for Possible Carcinogenicity. (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (140): 1-48; 1978.

The carcinogenicity of pivalolactone (PVL), an intermediate in polymer synthesis, was investigated in groups of 50 Fischer rats and B6C3F1 mice of each sex that were treated with PVL in distilled water 3x/wk by gavage for 103 or 102 wk, respectively. The rats received 150 or 300 mg/kg PVL as a 1.5%-3.0% soln; the mice received 75 or 150 mg/kg as a 0.68%-1.36% soln. Among rats given the high PVL dose, 62% of the males and 56% of the females were alive at the end of 103 wk; corresponding survival rates in the low-dose group were 76% and 86%. Survival

rates for mice were 90% of males and 72% of females in the high-dose group and 84% of males and 72% of females in the low-dose group. The rats were observed for an additional 2 wk and the mice for 1 wk before sacrifice. PVL administration was associated with an increased incidence of squamous cell papillomas (scp's) and squamous cell carcinomas (scc's) of the forestomach in rats but not in mice. No other rare or unusual tumors were observed in either species. In the high-dose rats, the increase in forestomach tumors was statistically significant in both males and females. Scp's or scc's developed in 21/48 high-dose rats vs 6/49 low-dose rats and 0/19 controls. In female rats, the combined incidence of these tumors was 11/50 high-dose animals, 2/50 low-dose animals, and 0/20 controls. The results indicate that PVL is carcinogenic for Fischer 344 rats of both sexes but not for B6C3F1 mice. (21 refs)

- 79-3417 Report on the Bioassay of 1-Phenyl-3-methyl-5-pyrazolone for Possible Carcinogenicity.** (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (141): 1-43; 1978.

The carcinogenicity of 1-phenyl-3-methyl-5-pyrazolone (PMP) a widely used dye intermediate, was investigated by chronic feeding studies in groups of 49 or 50 male and 50 female Fischer 344 rats and B6C3F1 mice. The animals received 2,500 or 5,000 ppm (rats) or 7,500 or 15,000 ppm (mice) in their diet for 102-103 wk and were observed for 2 additional wk. The only toxic effect observed was a compound-related mean body wt depression in mice; hence, the compound may not have been administered to rats at the max tolerated dose. Survival was adequate in all test groups for the animals to be at risk for late-developing tumors. The numbers of survivors in the low-dose (LD) and high-dose (HD) groups, respectively, at the end of the study were 29 and 37 for male rats, 44 and 44 for female rats, 40 and 43 for male mice, and 38 and 34 for female mice. A variety of tumors was found in both treated and control animals, but none of the tumors in dosed animals occurred at a significant frequency compared with controls. Tumor incidences in LD, HD, and control animals, respectively, were 49/49, 49/50, and 19/19 male rats, 36/50, 25/50, and 12/20 female rats, 20/49, 14/50, and 11/18 male mice, and 19/49, 15/46, and 9/20 female mice. It is concluded that, under the conditions of this bioassay, there was no evidence for the carcinogenicity of PMP in Fischer 344 rats or B6C3F1 mice. (18 refs)

- 79-3418 Bioassay of 3-Chloro-p-toluidine for Possible Carcinogenicity.** (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer

Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (145): 1-43; 1978.

To investigate the carcinogenicity of 3-chloro-p-toluidine (CT), a dye intermediate and avicide, a chronic feeding study in rodents was undertaken using dose levels determined in a prior subchronic toxicity test. Groups of 50 Fischer 344 rats and 50 B6C3F1 mice of each sex were used. Male and female rats received time-weighted av doses of 1,635 or 3,269 ppm CT for 78 wk and were observed for an additional 25 wk (except the high-dose males, which were observed for 24 wk). Male mice received 600 or 1,200 ppm and female mice received 300 or 600 ppm CT for 78 wk and were observed for an additional 12 wk. Animals were sacrificed when moribund or at the end of the observation period and subjected to histopathologic examination; 47-50 animals in each group were evaluated. A large variety of tumors was found in all dosed and control animals, with no significant differences between the groups. Tumor incidences in rats were 49/49, 50/50, and 20/20 for low-dose, high-dose, and control males, respectively, and 22/50, 28/50, and 7/19 for low-dose, high dose, and control females respectively. Tumor incidences in mice were 15/49, 14/49, and 5/20 in low-dose, high-dose, and control males, respectively, and 6/50, 11/47, and 5/20 in low-dose, high-dose, and control females, respectively. Moreover, adequate numbers of animals in all groups survived sufficiently long to be at risk for late-developing tumors. It is concluded that, under the conditions of this bioassay, there was no convincing evidence for the carcinogenicity of CT. (18 refs)

- 79-3419 Report on the Bioassay of Mexacarbate for Possible Carcinogenicity.** (Eng) National Cancer Institute (Carcinogenesis Testing Program, Div. Cancer Cause and Prevention, NIH, Bethesda, MD 20014). *Natl Cancer Inst Carcinog Tech Rep Ser* (147): 1-49; 1978.

To investigate the carcinogenicity of the pesticide mexacarbate (MCB), chronic feeding studies were undertaken in Osborne-Mendel rats and B6C3F1 mice using groups of 50 animals of each sex. The chronic study comprised 78 wk of feeding plus 33-34 wk of additional observation in rats, 14-15 wk in mice. Male rats received 188 or 375 ppm MCB for 17 wk, followed by 215 or 430 ppm, respectively, for 61 wk; female rats received 300 or 600 ppm for 17 wk, followed by 350 or 700 ppm for 61 wk, respectively. The time-weighted av concentrations administered to these four groups were 209, 418, 339, and 678 ppm. Male mice received 225 or 450 ppm for 6 wk, followed by 275 or 550 ppm for 14 wk and then 350 or 700 ppm for 58 wk, respectively; female mice received 37 or 74 ppm for 6 wk, followed by 50 or 100 ppm for 14 wk and then 75 or 150 ppm for 58 wk, respectively. The time-weighted av concentrations administered to these four groups were 327, 654, 68, and 135 ppm. Survival was adequate in all experimental groups so

that the animals were at risk for late-developing tumors. There were no significant differences in the incidences of any tumor in the rats compared with controls. Female mice may not have received the max tolerated dose of MCB, because the compound had no significant effect on survival or body wt. The numbers of tumor-bearing rats were 21/49, 16/48, and 6/20 for low-dose (LD), high-dose (HD), and control males, respectively, and 33/50, 28/50, and 12/20 for the respective female groups. The respective results for tumor-bearing mice were 24/46, 28/47, and 0/15 males and 16/48, 13/48, and 5/20 females. In male mice that survived ≥ 56 wk, the Cochran-Armitage test showed significant associations between dietary concentration of MCB and the incidences of hepatocellular carcinoma, sc fibrosarcomas, and skin fibromas, but the Fischer exact test did not support any of these results. It is concluded that, under the conditions of this bioassay, insufficient evidence was obtained to establish the car-

cinogenicity of MCB to Osborne-Mendel rats or B6C3F1 mice. (20 refs)

See also:

*(Rev.): 79-3001, 79-3002, 79-3003, 79-3004, 79-3005,
79-3006, 79-3007, 79-3008, 79-3009, 79-3010,
79-3011, 79-3012, 79-3013, 79-3014, 79-3015,
79-3016, 79-3026, 79-3034, 79-3045, 79-3046,
*(Phys.): 79-3421, 79-3422, 79-3423, 79-3425, 79-3426,
79-3428, 79-3432.

*(Viral): 79-3483.

*(Immun.): 79-3505, 79-3517, 79-3524, 79-3525, 79-3530.

*(Path.): 79-3556, 79-3559, 79-3560, 79-3561, 79-3564.

*(Epid.-Biom.): 79-3568, 79-3571, 79-3572, 79-3573,
79-3575, 79-3581, 79-3582, 79-3583,
79-3585.

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- 79-3420 Ultraviolet B Phototherapy for Psoriasis in Sunlight-responsive Patients (Letter to Editor).** (Eng) Boer, J. (Dept. Dermatology, Univ. Hosp., Leiden, Netherlands); Schothorst, A. A.; Suurmond, D. *Lancet* 1(8119): 773; 1979.

UV B phototherapy [wavelength range 290-320 nanometers (nm)] has several advantages over PUVA therapy [psoralen + long-wave (320-400 nm) UV A light] for the treatment of psoriasis. These include the avoidance of po administration of photosensitizers and shorter exposure times. However, just as for PUVA treatment, the long-term risk-benefit ratio (oncogenic aspects) of UV B therapy must be investigated extensively before it can be recommended for use on a large scale. (3 refs)

- 79-3421 Dithranol or Photochemotherapy for Psoriasis? (Letter to Editor).** (Eng) Howell, D. R. (Victoria General Hosp., Halifax, Nova Scotia, Canada). *Lancet* 1(8119): 772-773; 1979.

A previous paper in which dithranol therapy is compared with combined psoralen and long-wave UV (PUVA) treatment for chronic psoriasis is discussed. Some workers have reported that recurrences of psoriasis are relatively common after PUVA treatment; many patients require booster treatments every 1-2 wk, increasing the amount of exposure to long-wave UV light, with its attendant potential hazards (including skin cancer). (no refs)

- 79-3422 Topical Methoxsalen Administration and Sunlamp Fluorescent Irradiation in Psoriasis.** (Eng) Petrozzi, J. W. (Dept. Dermatology, 111-P, Philadelphia Veterans Admin. Hosp., University and Woodland Aves., Philadelphia, PA 19104); Barton, J. O.; Kligman, A.; de los Reyes, O. *Arch Dermatol* 115(4): 436-439; 1979.

Total resolution of psoriasis was achieved in 17/20 patients with corticosteroid-resistant disease treated with topical administration of psoralens and fluorescent light from a sunlamp. Resolution was achieved after an av of 18 treatments, and adverse blistering phototoxic reactions and excessive hyperpigmentation were not encountered. However, the potential risk of photocarcinogenicity makes this treatment experimental, and it should be reserved for recalcitrant cases. (15 refs)

- 79-3423 Effect of Ultraviolet Light and 8-Methoxypsoralen on Epidermal Melanocytes in Organ Cultures of Human Skin (Meeting Abstract).** (Eng) Kamen, P. (Lab. Electron Microscopy, Harvard Sch. Dental Medicine, Boston, MA); Garcia, T.; Szabo, G. *In Vitro* 15(3): 173-174; 1979 (no refs)

- 79-3424 Correlation of In Vitro Transformation with In Vivo Tumorigenicity in 10T1/2 Mouse Cells Exposed to UV Light.** (Eng) Chan, G. L. (Lab. Radiology, Harvard Univ. Sch. of Public Health, Boston, MA 02115); Little, J. B. *Br J Cancer* 39(5): 590-593; 1979.

The correlation between tumorigenicity in vivo and the acquisition of anchorage independence for growth in vitro was studied using UV-irradiated (254 nanometers) C3H mouse embryo-derived 10T1/2 cells. Eight independent transformed foci were selected for cloning; the clones were scored as Types II or III. The criss-crossing cell morphology was seen only at the periphery of Type III foci. A correlation was demonstrated between Type III morphology and the ability to grow in soft agar; Type II cells required attachment to a solid substrate for growth. Type III cells also formed tumors following sc injection into C3H/HENMTV- mice. When 2×10^6 cells were injected, there was a 100% tumor take. The Type II foci failed to cause tumors in vivo. The results indicate that the Type III foci were malignant transformants. The transformation of a normal cell to a fully malignant one appears to be a multistep process, with the loss of contact inhibition of growth being an earlier step than morphologic change, anchorage independence, and tumorigenicity. (13 refs)

- 79-3425 UV-induction of Diphtheria Toxin-resistant (DTr) Mutations in Xeroderma Pigmentosum (XP) Fibroblasts (Meeting Abstract).** (Eng) Trosko, J. E. (Dept. Human Development, Michigan State Univ., East Lansing, MI 48824); Glover, T. W.; Chang, C. C.; Liu, P. *Proc Am Assoc Cancer Res* 20: 4; 1979 (no refs)

- 79-3426 Morphologic Transformation of Hamster Cells by Ultraviolet Irradiation (UV) and Repair of Photo-induced DNA Damage (Meeting Abstract).** (Eng) Doniger, J. (NCI, Bethesda, MD 20014); DiPaolo, J. A. *Proc Am Assoc Cancer Res* 20: 64; 1979 (no refs)

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- 79-3427 Cell Killing by Ultra-Low Dose Rate Radiation: Population Survival Effects and Interaction with Conventional Radiation and Chemotherapeutic Agents in Chinese Hamster Cells (Meeting Abstract). (Eng) Shipley, W. U. (Dept. Radiation Medicine, Massachusetts General Hosp., Boston, MA 02114); Ling, C. C.; Gerweck, L.; Jennings, M. *Proc Am Assoc Cancer Res* 20: 289; 1979 (no refs)
- 79-3428 Evidence for a Common Rate Limiting Step in the Repair Process of Ultraviolet Light (UV) and *N*-Acetoxyacetylaminofluorene (N-AcO-AAF) Induced Damage in the DNA of Human Fibroblasts (Meeting Abstract). (Eng) Levinson, J. W. (Michigan State Univ., East Lansing, MI 48824); Konze-Thomas, B.; Maher, V. M.; McCormick, J. J. *Proc Am Assoc Cancer Res* 20: 105; 1979 (no refs)
- 79-3429 Abnormal Sensitivity to Near UV Light in Cultured Fibroblasts from Patients with Various Syndromes (Meeting Abstract). (Eng) Smith, P. J. (Atomic Energy Canada Limited, Chalk River, Ontario, K0J 1J0, Canada); Paterson, M. C. *Proc Am Assoc Cancer Res* 20: 88; 1979 (no refs)
- 79-3430 Factors Influencing Light-induced DNA Crosslinks in Cultured Cells (Meeting Abstract). (Eng) Gantt, R. (NCI, Bethesda, MD 20014); Jones, G. M.; Stephens, E. V.; Baeck, A. E.; Sanford, K. K. *Proc Am Assoc Cancer Res* 20: 251; 1979 (1 ref)
- 79-3431 Mucosal Histology after Gastric Surgery: Longterm Outcome of Billroth I (BI) and Billroth II (BII) Resection (Meeting Abstract). (Eng) Malchow, H. (Dept. Medicine, Univ. Tübingen, Tübingen, W. Germany); Mikulla, A.; Fischbach, H.; Schenzle, D.; Dietz, K. *Gastroenterology* 76(5, part 2): 1192; 1979 (no refs)
- 79-3432 Effects of TPA and Protease Inhibitors on X-Ray Induced Oncogenic Transformation (Meeting Abstract). (Eng) Kennedy, A. R. (Sch. Public Health, Harvard Univ., Boston, MA 02115); Little, J. B. *Proc Am Assoc Cancer Res* 20: 45; 1979 (1 ref)
- 79-3433 A Dosage Response Curve for the One Rad Range: Adult Risks from Diagnostic Radiation (Meeting Abstract). (Eng) Bross, I. D. (Roswell Park Memorial Inst., Buffalo, NY 14263); Ball, M.; Falen, S. *Proc Am Assoc Cancer Res* 20: 4; 1979 (no refs)
- 79-3434 Normal Serum Prevents Enhancement of Mammary Carcinoma Growth in Rats Caused by Whole Body X-Irradiation (Meeting Abstract). (Eng) Moroson, H. (New York Medical Coll., New York, NY 10029); Schechter, M.; Rivenson, A. *Proc Am Assoc Cancer Res* 20: 73; 1979 (1 ref)
- 79-3435 Protein Kinase Activities in X-Radiation-induced Adenocarcinoma of Rat Small Bowel. (Eng) Stevens, R. H. (Radiation Res. Lab., 14 Medical Labs., Univ. Iowa, Iowa City, IA 52242); Osborne, J. W.; Shaner, J. P.; Oberley, L. W. *Radiat Res* 76(1): 95-104; 1978.
- Cyclic AMP (cAMP)-dependent and cAMP-independent protein kinase activities were measured in cellular homogenates prepared from an x-radiation-induced mucoid adenocarcinoma of the Holtzman rat small bowel. These activities were compared with those in similar cell preparations of normal unirradiated small bowel tissues and with those in irradiated intestines that did not develop visible lesions. A significantly lower degree of cAMP-dependent endogenous and exogenous protein (histone) phosphorylation was observed in the cancer cells compared with that in other tissues. The activities of the enzymes in the tumor tissues, but not the other tissues, appeared to be independent of homogenate concentration. Similarly, cAMP stimulated only very slightly the malignant protein kinases, but significant stimulation occurred with the exogenous kinases of unirradiated small intestines and also of irradiated intestines without visible lesions. The results suggest that (1) the radiation-induced cancer consists of cells that no longer respond normally to extracellular signals that are transmitted intracellularly by the cAMP system or (2) greater phosphoprotein phosphatase activities exist in the cancer cells. (25 refs)
- 79-3436 Correlation Between Radiological, Scintigraphic and Histological Changes in Bone Following Irradiation of Rabbits with Single and Fractionated Doses. (Ger) Burgener, F. A. (Dept. Radiology, Univ. Rochester Medical Center, Rochester, NY 14642); King, M. A.; Weber, D. A. *ROEFO* 130(3): 359-366; 1979.
- The results of radiologic, scintigraphic (SG), and histologic analyses of bone alterations in irradiated rabbits were compared. Male New Zealand albino rabbits were randomized into one of three treatment groups: (1) controls (4 rabbits),

(2) 1,750 rads to the hind leg (8 rabbits), and (3) 4,650 rads to the hind leg fractionated over 3 wk (8 rabbits). X-rays of the contralateral and the radiated legs were obtained up to 12 mo postirradiation (PI). Signs of radiogenic bone atrophy were first noticed 6 mo PI. They had developed gradually and they consisted of nonhomogeneous, spotty mineralization; endosteal scalloping; and pagetoid bone in 13/16 Group 2 and 3 rabbits. The findings were similar in both groups. SG studies with technetium-99m pyrophosphate showed that there was an increased uptake of label in Group 2 irradiated legs 1 day PI compared with control levels. One mo later, this difference was not detectable. Another large increase in uptake was found 3 mo later, and a slight increase remained at 6 mo. However, there was a decrease in uptake in most irradiated legs 1 yr PI. Uptake reached a peak at 3 wk in Group 3 irradiated legs and then fell rapidly, so that at 2 mo uptake was lower than that in control legs. Another uptake peak was found 3-4 mo PI. At 11-12 mo PI, uptake in the irradiated legs was decreased. In some Group 2 and 3 animals, a third uptake peak occurred during the second half of the first yr PI that was found to coincide with tumor appearance. Histological findings at 1 yr were similar for Groups 2 and 3. Bone sarcomas (5 proximal tibia, 1 distal femur) were found in four Group 2 rabbits and in two Group 3 rabbits. The four osteoblastic sarcomas were detectable 3-6 mo earlier by SG studies than by the x-ray studies. These tumors were characterized by the formation of new bone on the x-rays. Evidence for the two fibroblastic sarcomas was first seen by x-rays as bone destruction that progressed rapidly. SC evidence of these tumors first appeared 1-3 mo after x-ray evidence. The results indicate that the combined use of x-ray and SG studies should result in an earlier diagnosis of radiation-induced changes in bone than that obtained with either study alone. (15 refs)

79-3437 Risks of Exposure to X-Rays in Patients Undergoing Long-Term Treatment for Scoliosis. (Eng) Nash, C. L. (Univ. Hosps. Cleveland, 2065 Adelbert Road, Cleveland, OH 44106); Gregg, E. C.; Brown, R. H.; Pillai, K. *J Bone Joint Surg [Am]* 61A(3): 371-374; 1979.

A study was made to determine the current genetic and carcinogenic risks of x-radiation to scoliosis patients under treatment and to evaluate ways in which these risks could be minimized. Thirteen healthy teenage girls with idiopathic adolescent scoliosis were studied using multiple thermoluminescent dosimeters while undergoing standard diagnostic roentgenograms. Av organ doses for each anteroposterior and lateral examination were calculated for bone marrow, gastrointestinal tract, lungs, breast tissue, and gonads. Given an av of 22 roentgenograms over a 3-yr Milwaukee brace-treatment program, the increase in organ carcinogenic risk due to x-radiation ranged from 3.4 to 15/million (1.3%-7.5%), except for breast tissue, which increased from 140 to 290/million (110%). When

posteroanterior exposures were used instead of anteroposterior exposures, the increased risk was reduced to 5.3/million (3.8%). The genetic risks of scoliosis roentgenographic studies were considered to be negligible, especially with gonadal shielding and infrequent roentgenograms made every 3-4 months. Good technique and judicious ordering of roentgenograms added significantly to the safety of the patient. (8 refs)

79-3438 Conditions for Inhibiting and Enhancing Effects of the Protease Inhibitor Antipain on X-Ray-induced Neoplastic Transformation in Hamster and Mouse Cells. (Eng) Borek, C. (Radiological Res. Lab., Dept. Radiology, Columbia Univ. Coll. Physicians and Surgeons, New York, NY 10032); Miller, R.; Pain, C.; Troll, W. *Proc Natl Acad Sci USA* 76(4): 1800-1803; 1979.

The effect of the protease inhibitor antipain on x-ray-induced neoplastic transformation was investigated in two in vitro systems in short-term cultures of freshly explanted hamster embryo cells and in the heteroploid C3H mouse embryo cell line 10T1/2, clone 8. Although there was no effect on cell survival in both cell systems compared with irradiated controls, the addition of 6 µg/ml antipain to the cultures 24 hr prior to irradiation resulted in enhanced transformation, compared with the frequency in cultures exposed to radiation alone. In contrast, the addition of antipain to cultures 10 min after irradiation resulted in a decreased transformation rate. This decrease was not found when antipain was added to the mouse cells 24 hr after irradiation or to the hamster cells 48 hr after irradiation. These results suggest that the protease inhibitor antipain has more than one mechanism of action in modulating the fixation and expression of transformation by x-radiation, possibly by the modification of DNA repair. (32 refs)

79-3439 Radiation Carcinogenesis and Radiological Protection. (Eng) Dolphin, G. W. (Natl. Radiological Protection Board, Harwell, Didcot, Oxon OX11 0RQ, England). *Environ Res* 18(1): 140-146; 1979.

Theoretical and pragmatic methods of setting radiation dose limits are described. With the theoretical approach, it is assumed that the relationship between cancer risk in an organ or tissue and the radiation dose to that organ or tissue is linear and that there is no dose threshold. The slope of the line is believed to be adequately determined from the sparse data on cancer incidence observed in irradiated humans. The pragmatic approach is based on the fact that 25% of all deaths are due to cancer and that cancer is present in an even greater percentage of the population at death. The object of setting dose limits in this approach is to ensure that radiation exposure does not significantly increase the risk of dying from cancer and that

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a worker exposed to radiation is not significantly more at risk than other workers. The risks to radiation workers are established by keeping records of accumulated film badge doses and accumulated internal exposures that are eventually correlated with the cause of and age at death. These data are then compared with similar data for other individuals. (20 refs)

79-3440 Implantation of Transitional Tumor Cells on the Cauterized Murine Urothelial Surface (Meeting Abstract). (Eng) Soloway, M. S. (Univ. Tennessee Center Health Sciences, Memphis, TN 38163); Masters, S. *Proc Am Assoc Cancer Res* 20: 256; 1979 (no refs)

79-3441 Assessment and Significance of the Skeletal Doses Due to ^{210}Po in Radioactive Spa Workers. (Eng) Clemente, G. F. (Laboratorio Radiotossicologia, CSN, Casaccia CNEN, C.P. 2400, 00100 Rome, Italy); Renzetti, A.; Santori, G. *Environ Res* 18(1): 120-126; 1979.

Studies of 15 workers at a spa in which patients bathe in radioactive springwater demonstrated that the urinary ^{210}Po concentrations of 10 workers were significantly higher than those of unexposed individuals. Urinary ^{210}Pb excretion was more variable, and it was higher in more recently exposed workers. (20 refs)

79-3442 Tumorigenic Hazard of Particulate α Activity in Syrian Hamster Lungs. (Eng) Anderson, E. C. (Health Div., Los Alamos Scientific Lab., Univ. California, Los Alamos, NM 87545); Holland, L. M.; Prine, J. R.; Smith, D. M. *Radiat Res* 78(1): 82-97; 1979.

Syrian hamsters were exposed to lung irradiation by three modalities that differed in degree of localization of the radiation dose and in the fraction of lung exposed. An object of these studies was to measure as directly as possible lung tumor yield as a function only of the degree of dispersion of radioactivity. This was accomplished by using microspheres that were uniform in size and radioactivity. The activity per particle and the number of particles were varied independently. Experimental conditions were chosen to determine the relative tumorigenicity of diffuse vs localized irradiation of the lung. Intratracheal instillation of ^{210}Po soln gave nearly uniform α irradiation of the entire lung, iv injection of large numbers of ZrO_2 microspheres loaded with ^{147}Pm gave whole lung exposures to low LET (linear energy transfer) radiation, and iv injection of Pu-loaded microspheres provided a series of focal α exposures with varying numbers and specific activities of particles. The Po and Pm exposures were highly

tumorigenic, whereas the Pu microspheres produced tumors only when a large fraction of the lung was exposed to large doses. Exposures of 2,000 $\text{PuO}_2\text{-ZrO}_2$ spheres per hamster, irradiating about 1% of the lung, gave no significant tumor incidence even at doses up to 118 nanocuries/g of lung tissue. It is concluded that, for a given lung burden of radioactivity, the most hazardous distribution is the most diffuse and that particulate α activity is an ineffective modality of irradiation. The importance of supplemental insults (ie, saline instillation) in determining tumor yield is shown. (35 refs)

79-3443 Morphological Changes in Rat Endocrine Organs During Chronic Internal Irradiation with ^{90}Sr . (Rus) Berezovskii, B. S. (Res. Inst. Biological Testing Chemical Compounds, Moscow, USSR). *Med Radiol (Mosk)* 24(4): 47-53; 1979.

Random-bred albino rats were given a single ip injection of ^{90}Sr (0.05 $\mu\text{Ci/kg}$) and then followed for 210 days. Only 75% of the males and 78% of the females survived to day 210 (compared with 95% and 97% of the controls). Tumors were detected in 17.3% of the male and 35.9% of the female survivors. Bone sarcomas and tumors of the hypophysis, adrenals, thyroid, testis, ovaries, mammary gland, and uterus were recorded. (9 refs)

79-3444 Effect of Small Doses of Americium-241 on Dogs and Rats. (Rus) Rudnitskaia, E. I. (No affiliation given); Moskalev, I. I. *Radiobiologiya* 19(2): 310-316; 1979.

The long term effects of exposure to a small dose of ^{241}Am were studied in dogs and random-bred albino rats. The isotope was given iv (dogs) at 0.1-7.5 $\mu\text{Ci/kg}$ or ip (rats) at 0.01-5.0 $\mu\text{Ci/kg}$. ^{241}Am was significantly more toxic for dogs than for rats; the av duration of survival of dogs inoculated with 2.5, 5.0 or 7.5 $\mu\text{Ci/kg}$ was 459, 233, and 204 days, respectively, compared with 4,778 days in controls. At 5.0 $\mu\text{Ci/kg}$, ^{241}Am caused an insignificant decrease in the av duration of survival of rats (460 days, compared with 559 days in controls). Osteosarcomas were detected in three of the four surviving dogs. The incidence of osteosarcomas in rats ranged from 4.35% at 0.01 $\mu\text{Ci/kg}$ to 12.5% at 2.5 $\mu\text{Ci/kg}$. (13 refs)

79-3445 Transfer of Actinides from the English Channel into the Southern North Sea. (Eng) Murray, C. N. (Chemistry Div., Commission European Communities, JRC Ispra Establishment, 21020 Ispra (Varese), Italy); Kautsky, H.; Eicke, H. F. *Nature* 278(5705): 617-620; 1979.

Isotopes of three actinide elements, plutonium, americium, and curium, were measured in the waters of the English Channel. On the basis of expected fallout ratios, an attempt was made to distinguish the probable source and dispersion of actinides entering the southern North Sea from the English Channel. The variations of activity concentrations in the Channel between March and October 1975 demonstrated the pulse-like nature of actinide contamination entering the North Sea. The major input of the isotopes was from a French nuclear fuel reprocessing plant at La Hague. Although plutonium water activities probably only represent <5% of the total released in low-level wastes, this fraction showed a high degree of mobility. Measurements of curium indicate that any support by americium would be minimal. It is necessary to develop methods for the analysis of the chemical forms of these elements. Actinide activities in the North Sea may be able to reflect increases in European nuclear generating and reprocessing capacities over the next few decades. (18 refs)

79-3446 Late Development of Colorectal Cancer Subsequent to Pelvic Irradiation. (Eng)

O'Connor, T. W. (Dept. Colon and Rectal Surgery, Cleveland Clinic Foundation, Cleveland, OH); Rombeau, J. L.; Levine, H. S.; Turnbull, R. B. *Dis Colon Rectum* 22(2): 123-128; 1979.

Two cases of probable radiation-induced colorectal cancer are reported. A 45-yr-old woman was treated for squamous cell carcinoma of the cervix by insertion of eight radon seeds (1.3 Ci) and by 3,000 rads of external radiation. She did well until 18 yr later, when a diagnosis of radiation proctitis and radiation stricture was made. She remained symptom-free until 12 yr after that, when she had severe tenesmus and hematochezia. The sigmoid colon was resected and a fungating, infiltrating, moderately differentiated adenocarcinoma was identified in the resected segment. A polyp proximal to the mass was identified as a tubular adenoma with mild dysplasia. A 46-yr-old woman with Stage II papillary adenocarcinoma of the endocervix received 2,800 rads of external radiation therapy followed 2 mo later by two implantations of 80 mg of radium seeds for a total of 7,680 mg-hr radiation. She remained well until 16 yr later, when a fixed pelvic mass identified as a moderately differentiated rectal adenocarcinoma was found. The fibrosis produced by the therapeutic irradiation involved the ureter and caused adhesions of the colon, uterus, and ureter. A segment of ureter was resected at colectomy, and the development of a ureterocolic fistula complicated the postoperative course. Patients with radiation-induced proctocolitis should be followed for life, and high-risk patients should have barium-enema examinations at regular intervals. The risk of radiation-associated tumors may increase in men given radiotherapy for adenocarcinoma of the prostate. (17 refs)

79-3447 Microdistribution of Thorotrast and Dose to Cellular Structures. (Eng) Kaul, A. (Freie Universitat Berlin, Klinikum Steglitz, Berlin 45, W. Germany); Foll, U.; Haase, V. A.; Palme, G.; Riedel, W.; Stolpmann, H. J. *Environ Res* 18(1): 13-22; 1979.

Thorium dioxide is distributed inhomogeneously within the tissues of Thorotrast patients. To calculate tissue and cellular dose levels in humans, experiments were conducted in female CF₁ mice and male rabbits to determine the accumulation of ThO₂ particles as a function of time after Thorotrast exposure and self-absorption of α -particles within Thorotrast aggregates of various sites. Colloidal ThO₂ was injected iv into female mice (25, 125, and 250 μ l) or administered intravascularly to male rabbits (2 ml). The animals were sacrificed between 1 and 100 or 392 days, respectively, and tissue samples of the liver and spleen were analyzed by histoautoradiography and light and electron microscopy with respect to the size distribution of the aggregates. For dose calculations, the aggregates were assumed to be spherical, and they were classified into groups within the radius range 0.045-20 μ m. Dose rate estimates were made with respect to cellular structures, cells adjacent to Thorotrast granules, and liver tissue in general, with self-absorption of α -particles being considered a function of Thorotrast particle size and duration of Thorotrast body burden. According to the results, local dose rates may vary from about 4,000 rads/yr (cell membranes) to about 30 rads/yr (mean dose rate to nuclei of liver cells). The mean dose rate to cells within the range of α -particles emitted from the aggregates will be of the order of 0.2 mrad/yr to 2 rads/yr. (6 refs)

79-3448 Evolution of Thorotrast-induced Hepatic Angiosarcomas. (Eng) Telles, N. C. (Bureau Radiological Health, Food and Drug Admin., Rockville, MD 20857); Thomas, L. B.; Popper, H.; Ishak, K. G.; Falk, H. *Environ Res* 18(1): 74-87; 1979.

Pathological findings in 25 cases of Thorotrast-induced angiosarcoma are presented. Characteristic antecedent or precursor changes similar to previously described changes present in hepatic angiosarcoma secondary to vinyl chloride, or arsenicals or of unknown etiology were found. The antecedent or precursor change consisted of areas with simultaneous activation of both the hepatocytes and sinusoidal cells and associated lesions in the sinusoidal and perisinusoidal spaces. The hepatic cell plates surrounding these areas were compressed, with subsequent development of fibrous septa at the interface between the areas of mixed hyperplasia and the areas of compression. In these multiple areas, multicentric angiosarcomas developed in close approximation to the portal tracts but not to the Thorotrast deposits. (24 refs)

- 79-3449 Thorotrast Dosimetric Study in Japan. (Eng) Kato, Y. (Nat. Inst. Radiological Sciences, Chiba, Japan); Mori, T.; Kumatori, T. *Environ Res* 18(1): 32-36; 1979.

The results of a Japanese study concerning the amounts of Thorotrast absorbed by the liver, spleen, and bone marrow of 39 exposed individuals are presented and compared with those of a similar German study. Steady-state activity ratios and the self-absorption of α particles in $^{232}\text{ThO}_2$ aggregates were determined in autopsy specimens using α - and γ -ray spectrometry. Twenty-four of the 39 individuals died of liver cancer (17 with cholangiocarcinoma, 5 with hemangioendothelioma, 2 with liver cell carcinoma). Dose absorbed by the liver in 23 of these individuals was 939 rads. The mean absorbed doses in the spleen and bone marrow in all 24 subjects were 5,760 and 3,087 rads, respectively. Causes of death in the remaining subjects were cirrhosis of the liver (6), aplastic anemia (2), and osteosarcoma, acute myeloid leukemia, erythroleukemia, lung carcinoma, sarcoma at injection site, stomach ulcer, and miliary tuberculosis (1 each). Mean absorbed doses in the liver, spleen, and bone marrow of these individuals were 893, 4,501, and 1,042 rads, respectively. (6 refs)

- 79-3450 Microdistribution of Thorotrast Conglomerates in Lymph Nodes and Radiation Exposure of Single Lymphocytes. (Eng) Steinstrasser, A. (Institut für Biophysik der Universität des Saarlandes, Boris Rajewsky-Institut, D-6650 Homburg, W. Germany); Kemmer, W.; Muth, H. *Environ Res* 18(1): 23-31; 1979.

The microdistribution of Thorotrast (TT) aggregates was studied in autopsy specimens of the lymph nodes of exposed individuals and in the lymph nodes of experimentally exposed Chinese hamsters. Some lymph nodes of exposed patients were almost free of TT, but others were not able to function because of the high TT level. The aggregates either formed a wall in the outer region of the follicle or were evenly distributed throughout the follicle. Dose rates in lymph nodes with low TT levels were similar, averaging about 210 rads/yr. Dose rates in lymph nodes with high TT levels ranged from an av of 645 rads/yr (intermediary sinus and trabeculae) to 1,352 rads/yr (marginal sinus, corresponding region of cortex) to 2,319 rads/yr (follicle wall). (16 refs)

- 79-3451 Osteosarcoma Induced by Short-lived Bone-seeking α Emitters in Mice: The Role of Age. (Eng) Luz, A. (Institut für Biologie, Gesellschaft für Strahlen- u. Umweltforschung mbH München, D-8042 Neuherberg, W. Germany); Müller, W. A.; Gossner, W.; Hug, O. *Environ Res* 18(1): 115-119; 1979.

The incidence of osteosarcoma was compared in weanling

(1-mo-old) and adult (5- to 6-mo-old) female NMRI-Neuherberg mice injected ip with isotonic solns of the α emitters ^{224}Ra and ^{227}Th . The osteosarcoma incidences in weanling and adult mice that received one injection of 25 $\mu\text{Ci/kg}$ ^{224}Ra were 18% and 9%, respectively; the incidences in mice that received one injection of 5 $\mu\text{Ci/kg}$ ^{227}Th were 42% and 19%, respectively. The incidences in animals receiving 0.28 $\mu\text{Ci/kg}$ ^{227}Th every 2 wk for 9 mo were similar, 84% and 76%, respectively. Age-corrected osteosarcoma incidences were not significantly different in the two age groups in any experiment. However, the av latent periods in the weanling mice that received a single injection of ^{224}Ra (553 days), a single injection of ^{227}Th (545 days), and long-term ^{227}Th treatment (375 days) were significantly longer than those in the adult mice (476 days, $p \leq 0.05$; 432 days, $p \leq 0.005$; and 331 days, $p \leq 0.0005$, respectively). The lower latent period in the adults may be related to age. (14 refs)

- 79-3452 Comparative Investigations on the Biokinetics of Colloidal Thorium, Zirconium, and Hafnium Dioxides in Animals. (Eng) Riedel, W. (Klinikum Steglitz, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin 45, W. Germany); Hirschberg, R.; Kaul, A.; Schmier, H.; Walter, U. *Environ Res* 18(1): 127-139; 1979.

The biokinetics of colloidal thorium dioxide (Thorotrast, TT), zirconium dioxide, and hafnium dioxide were compared in female Wistar rats. A thorium dioxide aquasol whose particle size distribution corresponded to that of commercial TT and dextrin-stabilized aquasols of zirconium and hafnium dioxide were administered to the rats. TT kinetics were also studied in mice, rabbits, and 1 dog. The animals received 60-600 μl of one of the aquasols, and they were sacrificed 1-100 days later. The whole-body kinetics of the thorium and hafnium dioxides were studied by single whole-body counting. Both the distribution of the colloids and the activity ratios between ^{232}Th and its daughter products were determined. The results indicate that the biological behavior of thorium dioxide colloid in animals is comparable to that in humans. With increasing amounts of the three colloids, the storage capacity of the hepatic and Kupffer cells was progressively limited. (15 refs)

- 79-3453 Thorium Dioxide and the Liver--Scintigraphic Aspects. (Eng) Oliveira, E. A. (Servico de Medicina IV, Hospital de Santa Maria, Lisbon, Portugal); Saragoca, A.; Oliveira, M. P.; Tavares, M. H.; da Silva Horta, J. *Environ Res* 18(1): 216-217; 1979.

^{131}I -rose bengal or ^{99}Tc -sulfur colloid liver scans were carried out in 24 patients who had been injected with thorium dioxide colloid (Thorotrast) 26 to 50 yr earlier. Only two

scans were considered normal. The observations were consistent with fibrosis in five and with cirrhosis in two. Of 15 patients with a scan showing single or multiple space-occupying lesions, 10 died with a clinical picture of primary liver cancer. Besides poor patient cooperation, it is felt that neither clinical examination nor biochemical and scintigraphic testing are helpful in the early diagnosis of liver malignancy in persons exposed to Thorotrast. (4 refs)

79-3454 Chromosome Radiation-induced Aberrations in Patients Injected with Thorium Dioxide.

(Eng) Teixeira-Pinto, A. A. (Laboratorio de Citogenetica, Instituto de Histologia e Embriologia, Faculdade de Medicina de Lisboa, Lisbon, Portugal); Azevedo e. Silva, M. C. *Environ Res* 18(1): 225-230; 1979.

Cytogenetic studies were carried out in 22 subjects who had been injected with thorium dioxide colloid (Thorotrast) 21-43 yr previously. No significant differences in the number of abnormal cells or chromosome breaks were found when nine patients who received <24 ml of Thorotrast intraarterially and four patients who received >24 ml of Thorotrast intraarterially were compared. The percentages of abnormal cells and breaks in the 13 intraarterially injected patients were 8.78% and 15.58%, respectively. In the seven patients who had received only 2 ml of Thorotrast for perinasal sinus visualization, the incidences of acentrics, dicentrics, and breaks were 5.34%, 0.89%, and 7.12%. The percentages of acentrics and dicentrics in the systemically injected patients, who received 7-36 ml Thorotrast, were 8.02% and 3.58%, respectively. (12 refs)

79-3455 Movement of Thorotrast Aggregates in the Bone Marrow.

(Eng) da Silva Horta, J. (Dept. Pathology, Faculty Medicine, Lisbon, Portugal); Moura Nunes, J. *Environ Res* 18(1): 184-191; 1979.

Morphologic and autoradiographic studies of the distribution of thorium dioxide colloid (Thorotrast, TT) in autopsy specimens of bone from 23 exposed individuals demonstrated that the greatest amounts accumulated in or near the trabeculae. There was little accumulation in the periosteum and in the channels and spaces of Havers. TT aggregates were much more abundant in active bone marrow than in adipose marrow. These and further observations suggest that TT aggregates are attracted to the endosteal surfaces of bone. (11 refs)

79-3456 Chromosome Aberrations as a Biological Dosimeter in Thorotrast Patients: Dosimetric Problems.

(Eng) Kemmer, W. (Bundesministerium des Innern, D-5300 Bonn 1, W. Germany); Steinstrasser, A.; Muth, H. *Environ Res* 18(1): 178-183; 1979.

Cytogenetic studies of lymphocytes from 68 Thorotrast-exposed subjects showed dicentric chromosome aberrations in all subjects. However, there was no clear dose-response relationship between chromosome aberration rate and calculated whole-body Thorotrast exposure or between chromosome aberration rate and estimated dose absorbed by the reticuloendothelial system. (11 refs)

79-3457 Pathological Changes of Lymph Nodes in Thorotrast Patients. Pathoanatomical, Autoradiographical, and Quantitative Investigations.

(Eng) Wegener, K. (Pathological Inst., Municipal Hosp. Ludwigshafen, Bremserstrasse 79, 6700 Ludwigshafen/Rhein, W. Germany); Wesch, H. *Environ Res* 18(1): 245-255; 1979.

Pathological, autoradiographic, and quantitative studies of postmortem organ specimens of 38 patients with thorotrastosis demonstrated that the highest ^{232}Th concentrations were in the lymph nodes of the liver hilus, those in the area behind the pancreas, and those around the hilus of the spleen. The latency period, ie, the time between Thorotrast injection and death, ranged from 24 to 38 yr. (35 refs)

79-3458 Biological Activity In Vitro of Chrysotile Compared to Its Quarried Parent Rock (Platy Serpentine).

(Eng) Frank, A. L. (Environmental Sciences Lab., Dept. Community Medicine, Mount Sinai Sch. Medicine, New York, NY 10029); Rohl, A. N.; Wade, M. J.; Lipkin, L. E. *J Environ Pathol and Toxicol* 2(4): 1041-1046; 1979.

The biological activities of the fibrous serpentine mineral chrysotile, asbestos and the platy serpentines quarried at Rockville, Maryland, were evaluated via an in vitro bioassay using the malignant mouse macrophagelike cell line P338D₁. Crushed stone from the quarry is used for roads, playgrounds, and other surfaces. Air samples taken in the vicinity of roads paved with the stone showed that chrysotile concentrations were approx 1,000 times greater than those found in typical urban areas of the US. The chrysotile (100 $\mu\text{g}/\text{ml}$) was cytotoxic at 72 hr, whereas the ground rock sample (platy serpentine) had no effect on cell growth. Although there are at present no scientific data to relate cytotoxicity to carcinogenicity directly, the fact that there is significant biological activity in the extracted chrysotile from the quarried rock brings the Rockville Quarry problem into sharper focus. (8 refs)

79-3459 Studies on In Vitro Asbestos-Cell Interaction.

(Eng) Wade, M. J. (NCI, Park Bldg. Rm. 417, 9000 Rockville Pike, Bethesda, MD 20014); Lipkin, L. E.;

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Frank, A. L. *J Environ Pathol and Toxicol* 2(4): 1029-1039; 1979.

The effects of filtration, prior leaching, and exposure time to the cytotoxic effects of amosite and chrysotile B asbestos on malignant mouse macrophagelike (P388D₁) cells and human lung (WI-38) cells were studied. Unfiltered chrysotile (100 µg/ml) was cytotoxic to P388D₁ cells, whereas the chrysotile filtrate had no such effect. Unfiltered amosite also caused cytotoxicity, but not the amosite filtrate. Cytotoxicity correlated well with duration of exposure; after 12-24 hr, no additional cytotoxic effect was noted. However, all the asbestos could not be removed by washing, and some residual asbestos was present. Phagocytosis of the asbestos did not block the progression of P388D₁ cells through the cell cycle, unless fibers were present in sufficient concentration or positioned to cause irreversible cell damage. WI-38 cells exposed to amosite showed little morphological change, but cells exposed to chrysotile appeared to elongate and contract, becoming narrower and more fibrous with the appearance of intercellular spaces. (17 refs)

79-3460 Determination of Microgram Quantities of Asbestos by X-Ray Diffraction: Chrysotile in Thin Dust Layers of Matrix Material. (Eng) Lange, B. A. (Center Disease Control, Natl. Inst. Occupational Safety and Health, US Dept. Health, Education, and Welfare, 4676 Columbia Parkway, Cincinnati, OH 45226); Haartz, J. C. *Anal Chem* 51(4): 520-525; 1979.

A precise, accurate, and rapid x-ray diffraction method was developed to determine microgram quantities of chrysotile (serpentine asbestos). The method can measure microgram amounts of either pure chrysotile or small quantities of chrysotile (typically, 1%-10% by wt) in the presence of large amounts of matrix material. Detection limits as low as 2 µg/cm² (on a filter) are cited. In the development of this method, phase analysis procedures, method sample preparation, and a technique for x-ray absorption corrections were evaluated. The primary drawback of the method is interference by minerals such as antigorite, lizardite, the kaolinities, and, possibly, chlorite. Sample pretreatment and x-ray line profile analysis may reduce the adverse effects of interferences. (28 refs)

See also:

- *(Rev.): 79-3012, 79-3016, 79-3017, 79-3018, 79-3019, 79-3020, 79-3026, 79-3034, 79-3037.
- *(Chem.): 79-3050, 79-3051, 79-3066, 79-3089, 79-3099, 79-3188, 79-3217, 79-3228, 79-3229, 79-3250, 79-3291, 79-3318, 79-3353, 79-3357.
- *(Viral): 79-3494.
- *(Path.): 79-3546, 79-3550.
- *(Epid.-Biom.): 79-3570, 79-3571, 79-3572, 79-3573, 79-3574, 79-3580.

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- 79-3461 High Frequency Spontaneous Induction of Ecotropic Endogenous Retrovirus Correlates with Generation of Preleukemia in Granulopoietic Long Term Mouse Bone Marrow Cultures (Meeting Abstract). (Eng) Greenberger, J. S. (Dept. Radiation Therapy, Joint Center Radiation Therapy, Boston, MA 02115); Donahue, D.; Sakakenny, M.; Berendtsen, K. *Proc Am Assoc Cancer Res* 20: 13; 1979 (no refs)
- 79-3462 Non-Helper Virus Sequences of Moloney-MSV: Localization on Linear Unintegrated Viral DNA and Relationship to Other BALB/c-derived Sarcoma Viruses (Meeting Abstract). (Eng) Canaani, E. (Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014). *Proc Am Assoc Cancer Res* 20: 190; 1979 (2 refs)
- 79-3463 Infection of Cultured Human Cells with Murine Ecotropic Retroviruses: Studies by DNA Transfection (Meeting Abstract). (Eng) Yang, D. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Yang, W. K.; Myer, F. E.; Tennant, R. W. *Proc Am Assoc Cancer Res* 20: 248; 1979 (no refs)
- 79-3464 In Vitro Culture of Transplantable Lymphomas Induces Reexpression of Viral Antigens (Meeting Abstract). (Eng) Sabbath, M. (Dept. Pathology, Lenox Hill Hosp., New York, NY 10021); Moll, B.; Joachim, H. L. *Proc Am Assoc Cancer Res* 20: 198; 1979 (no refs)
- 79-3465 Horizontal Transmission of Gross Leukemia by Preleukemic Organ Grafts (Meeting Abstract). (Eng) Mariani, T. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455); Landucci, G. *Proc Am Assoc Cancer Res* 20: 236; 1979 (no refs)
- 79-3466 A Rapid Bioassay for Detecting Murine Leukemogenesis by Cellular gp71 Binding (Meeting Abstract). (Eng) Reed, C. D. (Frederick Cancer Res. Center, NCI, Frederick, MD 21701); Fowler, A. K.; Hellman, A. *In Vitro* 15(3): 224-225; 1979 (no refs)
- 79-3467 Copy Numbers and Chromatin Structures of Integrated Leukemia Proviral Gene Sequences in AKR Mice (Meeting Abstract). (Eng) Garcia, H. D. (Univ. Texas Graduate Sch. Biomedical Sciences at Houston, Houston, TX 77030); Saunders, G. F. *Proc Am Assoc Cancer Res* 20: 211; 1979 (no refs)
- 79-3468 Further Characterization of B-Tropic Murine Leukemia Viruses Isolated from C57BL Mice (Meeting Abstract). (Eng) Benade, L. (Cancer Biology Program, Frederick Cancer Res. Center, Frederick, MD 21701); Ihle, J. *Proc Am Assoc Cancer Res* 20: 191; 1979 (no refs)
- 79-3469 Generation of Rapidly Infecting Variants of Mouse Mammary Tumor Viruses In Vitro (Meeting Abstract). (Eng) Howard, D. K. (NCI, Bethesda, MD 20014); Schlom, J. *In Vitro* 15(3): 171; 1979 (no refs)
- 79-3470 C3H/HeNf Mouse Mammary Tumor Cell Lines as Sources of Endogenous Mouse Mammary Tumor Virus (Meeting Abstract). (Eng) Arthur, L. O. (Frederick Cancer Res. Center, Frederick, MD 21701); Schochetman, G.; Fine, D. L. *Proc Am Assoc Cancer Res* 20: 142; 1979 (no refs)
- 79-3471 The Possible Role of Murine Mammary Tumor Virus in Chemical Transformation of Whole Mouse Mammary Gland In Vitro (Meeting Abstract). (Eng) Tonelli, Q. J. (Inst. Cancer Res., Philadelphia, PA 19111); Long, C. A.; Vaidya, A. B. *Proc Am Assoc Cancer Res* 20: 260; 1979 (1 ref)
- 79-3472 The Human Breast Carcinoma Antigen Is Immunologically Related to the Polypeptide of the Group-specific Glycoprotein of the Mouse Mammary Tumor Virus (Meeting Abstract). (Eng) Ohno, T. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia Univ., New York, NY 10032); Ramanarayanan, M.; Spiegelman, S.; Feigelson, P. *Proc Am Assoc Cancer Res* 20: 277; 1979 (no refs)

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79-3473 Immunological Reactivities of Normal C3H/HeN Mice Infected with Mouse Mammary Tumor Virus (MTV) (Meeting Abstract). (Eng) Tagliabue, A. (NIH, NCI, Bethesda, MD 20014); Herberman, R. B.; Lavrin, D. H.; McCoy, J. L. *Proc Am Assoc Cancer Res* 20: 66; 1979 (no refs)

79-3474 Heterogeneity of MMTV Antigen Expression and Induction in Mouse Mammary Tumor Cells (Meeting Abstract). (Eng) Hager, J. C. (Dept. Medicine, Roger Williams General Hosp., Providence, RI 02908); Dexter, D. L.; Calabresi, P.; Heppner, G. H. *Proc Am Assoc Cancer Res* 20: 61; 1979 (1 ref)

79-3475 Increased Incidence of Immunohistochemically Detectable Mouse Mammary Tumor Virus-related Antigen in Breast Carcinoma Tissues of Patients with a Family History of this Disease (Meeting Abstract). (Eng) Mesa-Tejada, R. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia Univ., New York, NY 10032); Branwood, A. M.; Keydar, I.; Fenoglio, C. M.; Spiegelman, S. *Proc Am Assoc Cancer Res* 20: 276; 1979 (no refs)

79-3476 Regulation of Feline Sarcoma Virus (FeSV) Expression in FeSV Transformed Non-producer Mink Lung Cells (Meeting Abstract). (Eng) Porzig, K. J. (Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014); Robbins, K. C.; Aaronson, S. A. *Proc Am Assoc Cancer Res* 20: 283; 1979 (no refs)

79-3477 Herpesvirus Oncogenesis: Properties of Cells Transformed by Inactivated Virus or Defective Interfering Particles (Meeting Abstract). (Eng) Robinson, R. A. (Univ. Mississippi Medical Center, Jackson, MS 39216); Henry, B. E.; Vance, R. B.; O'Callaghan, D. J. *Proc Am Assoc Cancer Res* 20: 139; 1979 (no refs)

79-3478 Association Between an Epstein-Barr (EBV)-like Virus and Spontaneous Lymphoma in Baboons (Meeting Abstract). (Eng) Neubauer, R. H. (Frederick Cancer Res. Center, Frederick, MD 21701); Rabin, H.; Strnad, B. C.; Lapin, B. A.; Indzie, E.; Agrba, V. Z. *Proc Am Assoc Cancer Res* 20: 3; 1979 (no refs)

79-3479 Functionally Conserved Determinants on gp70s of Distinct Endogenous Primate

Retroviruses (Meeting Abstract). (Eng) Fine, D. L. (Frederick Cancer Res. Center, Frederick, MD 21701); Arthur, L. O.; Schochetman, G. *Proc Am Assoc Cancer Res* 20: 142; 1979 (no refs)

79-3480 Late Transcription and Simultaneous Replication of Simian Adenovirus 7 DNA as Revealed by Spreading Lytically Infected Cell Cultures. (Eng) Matsuguchi, M. (Dept. Microbiology, Faculty Medicine, Kyushu Univ., Fukuoka 812, Japan); Puvion-Dutilleul, F.; Moyne, G. *J Gen Virol* 42(3): 443-456; 1979.

Miller's technique of spreading transcription complexes was used to study virus replication and transcription in simian adenovirus 7 (SA7)-infected green monkey kidney cells. Electron micrographs demonstrated that transcription and replication occurred both separately and simultaneously on the same adenovirus DNA molecule late in the lytic cycle. Virus DNA from infected cells was identified by the presence of molecular species not observed in control cells: duplex DNA of limited length, almost smooth, carrying replication forks and ribonucleoprotein fibrils. Each one of these features was sufficient to characterize a novel kind of deoxyribonucleoprotein morphologically. The observation of numerous replication forks demonstrated that the single-stranded molecule remains bound to the DNA duplex during the spreading procedure. Three main forms of replication complexes were distinguished: Y-shaped replication forks, linear molecules in transition from single- to double-strandedness, and linear molecules that were entirely single- or double-stranded. There were pronounced discrepancies between the present measurements of the SA7 genome length and theoretical values estimated from the mol wt of a linear, double-stranded molecule. This may be explained by spreading or artifactual breaks of the virus DNA molecules, but a compaction of the DNA by proteins cannot be excluded. No differences in morphology between transcription of adenovirus DNA and transcription of non-nucleolar mammalian cell DNA, as studied by Miller's technique, were observed. (31 refs)

79-3481 Prevention of Primary Simian Adenovirus Type 7 (SA7) Tumors in Hamsters by Adoptive Transfer of Lymphoid Cells: Role of Different Cell Types (Meeting Abstract). (Eng) Datta, S. K. (Baylor Coll. Medicine, Houston, TX 77030); Trentin, J. J.; McCormick, K. J. *Proc Am Assoc Cancer Res* 20: 100; 1979 (no refs)

79-3482 Comparison of SV40 Induced Mouse and Hamster Tumor Specific Transplantation Antigen (Meeting Abstract). (Eng) Coggin, J. G. (Univ. South

Alabama, Mobile, AL 36688). *Proc Am Assoc Cancer Res* 20: 3; 1979 (no refs)

79-3483 The Interaction of N-Acetoxy-2-acetylaminofluorene (AAAF) with the SV40 Genome (Meeting Abstract). (Eng) Poupko, J. (Univ. Miami Sch. Medicine, Miami, FL 33101); Scott, W. A. *Proc Am Assoc Cancer Res* 20: 176; 1979 (no refs)

79-3484 SV40 Integration Sites and Growth Properties of Variant Cell Lines Derived from Mouse Kidney Cells Transformed by SV40tsA207 (Meeting Abstract). (Eng) Otsuka, H. (Baylor Coll. Medicine, Houston, TX 77030); Dubbs, D. R.; Kit, S. *Proc Am Assoc Cancer Res* 20: 276; 1979 (no refs)

79-3485 Isolation and Characterization of BK Virus-transformed Rat and Mouse Cells. (Eng) Seehafer, J. (Dept. Biochemistry, Univ. Alberta, Edmonton, Alberta T6G 2H7, Canada); Downer, D. N.; Salmi, A.; Colter, J. S. *J Gen Virol* 42(3): 567-578; 1979.

Four groups of BK virus (BKV)-transformed rat embryo fibroblast (RE) and mouse kidney (MK) cells were isolated and characterized. They consisted of (1) 7 RE lines transformed with a BKV pool containing a high proportion of defective virions, and (2) 16 RE, (3) 14 BALB/c-MK, and (4) 2 Swiss ICR-MK lines transformed (at different input multiplicities), with a pool of BKV free of defective virions. None of the lines produced BKV, all contained BKV tumor antigen, and all grew to higher saturation densities and had higher plating efficiencies than the corresponding control cells. Cells of the RE lines transformed with the BKV pool containing defective virions formed colonies in soft agar and produced tumors in irradiated weanling rats, but cells of the RE lines transformed with the defective virion-free pool did neither. Cells of the BALB/c-MK lines, but not of the ICR-MK lines, were tumorigenic, although cells of both groups formed colonies in soft agar. In general, lines transformed at higher multiplicities expressed the biological properties associated with transformation more strongly than those transformed at lower multiplicities. (12 refs)

79-3486 Differential Susceptibility of Epstein-Barr Virus Early Antigen Expression to Different DNA Synthesis Inhibitors (Meeting Abstract). (Eng) Lidin, B. (Dept. Microbiology and Surgery, Birmingham Veterans Admin. Hosp., Univ. Alabama in Birmingham, Birmingham, AL 35294); Lamon, E. W. *Proc Am Assoc Cancer Res* 20: 159; 1979 (no refs)

79-3487 Epstein-Barr Virus (EBV) DNA Synthesis Induced by Superinfection: P3HR-1 Virus and Various Cell Lines (Meeting Abstract). (Eng) Ecklund, P. S. (Wayne State Univ., Detroit, MI 48201). *In Vitro* 15(3): 171; 1979 (no refs)

79-3488 Epstein-Barr Virus (EBV)-Positive Burkitt Lymphoma (BL) in a Japanese: Virology, Cell Culture, Cytogenetics and Heterotransplantation (Meeting Abstract). (Eng) Miyoshi, I. (Dept. Medicine, Okayama Univ. Medical Sch., Okayama 700, Japan); Hikita, T.; Tanaka, T. *Proc Am Assoc Cancer Res* 20: 23; 1979 (1 ref)

79-3489 Transcription of Epstein-Barr Virus Genome in (Super)-infected Lymphoblastoid Cells (Meeting Abstract). (Eng) Lau, R. Y. (Lab. Molecular Virology, Life Sciences Biomedical Res. Inst., St. Petersburg, FL 33710); Tanaka, A.; Nonoyama, M. *Proc Am Assoc Cancer Res* 20: 44; 1979 (no refs)

79-3490 Temporal Acquisition of Phenotypic Alterations and Tumorigenicity in Cells Exposed to DNA of Herpes Simplex Virus Type 2 (Meeting Abstract). (Eng) Jariwalla, R. J. (Johns Hopkins Univ., Baltimore, MD 21205); Aurelian, L.; Ts'o, P. O. *Proc Am Assoc Cancer Res* 20: 153; 1979 (2 refs)

79-3491 Characterization of Type C Virus Particles in ESP-1 Cell Line by Immunoelectron Microscopy (IEM) (Meeting Abstract). (Eng) Ohtsuki, Y. (Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Myers, B.; Dmochowski, L.; Bowen, J. M. *Proc Am Assoc Cancer Res* 20: 278; 1979 (no refs)

79-3492 Virus and Cell DNA Synthesis in Non-proliferating Hepatocytes Infected with Herpes Simplex Viruses Types 1 or 2 (HSV-1, HSV-2) or Human Cytomegalovirus (HCMV) (Meeting Abstract). (Eng) Isom, H. C. (Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033). *Proc Am Assoc Cancer Res* 20: 281; 1979 (no refs)

79-3493 Hepatitis B Virus and Primary Hepatocellular Carcinoma: Family Studies in Korea (Meeting Abstract). (Eng) Hann, H. L. (Inst. Cancer Res.,

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Philadelphia, PA 19111); London, W. T.; Whitford, P.; Kim, C. Y.; Blumberg, B. S. *Proc Am Assoc Cancer Res* 20: 432; 1979 (no refs)

RNA Tumor Viruses in Somatic Cell Hybrids (Meeting Abstract). (Eng) Wright, C. E. (Roswell Park Memorial Inst., Buffalo, NY 14263); Shows, T. B. *Proc Am Assoc Cancer Res* 20: 158; 1979 (no refs)

79-3494 In Vitro Isolation and Characterization of Retroviruses from ^{90}Sr -induced Murine Osteosarcomas (Meeting Abstract). (Eng) Lee, C. K. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439); Chan, E. W.; Reilly, C. A.; Finkel, M. P.; Rockus, G.; Pahnke, V. A. *In Vitro* 15(3): 171; 1979 (no refs)

79-3497 An Electron Microscopic Examination of Canine Mammary Tumors (Meeting Abstract). (Eng) Hinson, E. (Auburn Univ., Auburn, AL 36830). *Diss Abstr Int [B]* 39(10): 4763; 1979 (no refs)

79-3495 In Vivo Expression of a C-type Virus in Rat Ventral Prostate Epithelial Cells (Meeting Abstract). (Eng) Anderson, K. M. (Section Medical Oncology, Dept. Medicine, Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 60612); Seed, T. M. *Proc Am Assoc Cancer Res* 20: 246; 1979 (no refs)

See also:

*(Rev.): 79-3002, 79-3021, 79-3022, 79-3023, 79-3024, 79-3034, 79-3040, 79-3041, 79-3046.

*(Chem.): 79-3193, 79-3269, 79-3291.

*(Immun.): 79-3507, 79-3509, 79-3515, 79-3519, 79-3526, 79-3528.

*(Path.): 79-3532, 79-3553.

*(Epid.-Biom.): 79-3566.

79-3496 Identification and Chromosome Assignments of Human Genes Involved in the Release of

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- 79-3498 Identification of a Human Acute Lymphocytic Leukemia Associated Antigen (Meeting Abstract).** (Eng) Veit, B. C. (St. Jude Children's Res. Hosp., Memphis, TN 38101). *Proc Am Assoc Cancer Res* 20: 279; 1979 (no refs)
- 79-3499 Effect of 3M-KCl Soluble Cell Surface Antigens on the Incidence of Spontaneous Mouse Mammary Tumors (Meeting Abstract).** (Eng) Lasfargues, E. Y. (Inst. Medical Res., Camden, NJ 08103); Lasfargues, J. C.; Reed, L. *Proc Am Assoc Cancer Res* 20: 87; 1979 (no refs)
- 79-3500 High Incidence of Migration Inhibition Reactivity to Lung Tumor-associated Antigens by Normal Donors in Close Contact with Lung Cancer (Meeting Abstract).** (Eng) Suslov, I. (NCI, Bethesda, MD 20014); McCoy, J. L.; Cannon, G. B.; Herberman, R. B. *Proc Am Assoc Cancer Res* 20: 234; 1979 (no refs)
- 79-3501 Isolation of a 70,000 Molecular Weight Tumor-associated Antigen from Novikoff Hepatoma Cell Cytosol (Meeting Abstract).** (Eng) Taylor, C. W. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030); Yeoman, L. C.; Busch, H. *Proc Am Assoc Cancer Res* 20: 34; 1979 (no refs)
- 79-3502 Humoral Immune Response Augmentation by Synthetic Glycolipids Does Not Cause Tumor Enhancement (Meeting Abstract).** (Eng) Chopra, C. (Faculty Medicine, Univ. Sherbrooke, Sherbrooke, Quebec J1H 5N4, Canada); Nigam, V. N.; Brailovsky, C. A. *Proc Am Assoc Cancer Res* 20: 51; 1979 (no refs)
- 79-3503 Common Surface Antigens of Human Melanoma Cell Lines Detected by Rabbit Xenantisera (Meeting Abstract).** (Eng) Rahman, A. F. (McMaster Univ., Hamilton, Ontario, Canada). *Proc Am Assoc Cancer Res* 20: 237; 1979 (no refs)
- 79-3504 Are Human Melanoma Associated Antigens Modified Histocompatibility Antigens (Meeting Abstract)?** (Eng) McCabe, R. P. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037); Galloway, D.; Indiveri, F.; Curry, R.; Reisfeld, R. A. *Proc Am Assoc Cancer Res* 20: 237; 1979 (no refs)
- 79-3505 Activity of NP-40 Solubilized and Enriched TATA from an MCA-induced BALB/c Sarcoma Containing Alien H-2k Antigens (Meeting Abstract).** (Eng) DuBois, G. C. (NCI, Bethesda, MD 20014); Appella, E.; Law, L. W.; Parmiani, G. *Proc Am Assoc Cancer Res* 20: 126; 1979 (no refs)
- 79-3506 Cytotoxic Potential of Antibody to Oncofetal Antigen in Sera from Melanoma Patients (Meeting Abstract).** (Eng) Sidell, N. (Div. Oncology, UCLA Sch. Medicine, Los Angeles, CA 90024); Irie, R. F.; Irie, K. *Proc Am Assoc Cancer Res* 20: 216; 1979 (2 refs)
- 79-3507 T-T Cell Synergy in the In Vitro Generation of Secondary Cell-mediated Cytotoxicity Against Syngeneic SV-40 Transformed Cells (Meeting Abstract).** (Eng) Glaser, M. (George Washington Univ. Medical Center, 2300 Eye St., N.W., Washington, DC 20037). *Proc Am Assoc Cancer Res* 20: 226; 1979 (no refs)
- 79-3508 Macroglobulinemia and Autoimmune Disease in a Family (Meeting Abstract).** (Eng) Blattner, W. A. (NIH, Bethesda, MD 20014); Garber, J.; Mann, D. L.; Fisher, W.; Bauman, A. W.; Fraumeni, J. F. *Proc Am Assoc Cancer Res* 20: 413; 1979 (no refs)
- 79-3509 General and Cell-mediated Immune Depression in Mice Bearing Progressively Growing Simian Virus 40-induced Sarcoma: Evidence for Suppressor Cell Activity (Meeting Abstract).** (Eng) Padarathsingh, M. L. (Litton Bionetics, Inc., Kensington, MD 20795); Dean, J. H.; Northing, J. W.; Keys, L.; Law, L. W. *Proc Am Assoc Cancer Res* 20: 83; 1979 (no refs)

- 79-3510** Lymphoreticular Malignancies in Patients with Genetically-determined Immunodeficiencies: Evaluation by Surface Markers and/or Histology (Meeting Abstract). (Eng) Spector, B. D. (Immunodeficiency-Cancer Registry, Univ. Minnesota, Minneapolis, MN 55455). *Proc Am Assoc Cancer Res* 20: 98; 1979 (1 ref)
- 79-3511** Genetic Control of Immunity to Transplantable Murine Neuroblastoma (Meeting Abstract). (Eng) Epstein, R. H. (Univ. Pennsylvania Sch. Medicine, Philadelphia, PA 19104); Elkins, W. L. *Proc Am Assoc Cancer Res* 20: 178; 1979 (no refs)
- 79-3512** Macrophage-Lymphocyte Interaction and the Regulation of the Immune Response to Murine Melanoma (Meeting Abstract). (Eng) Stelzer, G. T. (Dept. Microbiology and Immunology, Univ. Louisville Medical Sch., Health Sciences Center, Louisville, KY 40201); Shellhaas, J. L.; Wallace, J. H. *Proc Am Assoc Cancer Res* 20: 12; 1979 (no refs)
- 79-3513** Quantitation of Human Thymus/Leukemia-associated Antigen (HTHy-L) by Radioimmunoassay in Hematopoietic Cell Lines (Meeting Abstract). (Eng) Chechik, B. E. (Res. Dept., Mount Sinai Hosp., Toronto, Canada); Minowada, J. *Proc Am Assoc Cancer Res* 20: 5; 1979 (no refs)
- 79-3514** Heterogeneity of Cell Lineages in Adult L3 Leukemias (Meeting Abstract). (Eng) Koziner, B. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Mertelsmann, R.; McKenzie, S.; Gee, T.; Good, R. A.; Clarkson, B. *Proc Am Assoc Cancer Res* 20: 112; 1979 (1 ref)
- 79-3515** Subpopulations of Tumor Antigen Sensitized Mouse Splenic Lymphocytes Responding to Antigens and Mitogens under Syngeneic and Xenogeneic Culture Conditions (Meeting Abstract). (Eng) Laing, C. A. (Dept. Oncology/Cancer Center, Howard Univ., Washington, DC 20060). *Proc Am Assoc Cancer Res* 20: 38; 1979 (2 refs)
- 79-3516** Selective Inhibition of Natural Killer Cell Activity Against Human Diffuse Histiocytic Lymphoma Cell Lines by IgG (Meeting Abstract). (Eng) Miller, R. A. (Stanford Univ. Medical Center, Stanford, CA 94305); Fry, K.; Kaplan, H. S. *Proc Am Assoc Cancer Res* 20: 33; 1979 (no refs)
- 79-3517** Efficacy of Thymosin Pre-incubated Tumor Enhancing T-Lymphocytes to Control Lewis Tumor Growth in Mice (Meeting Abstract). (Eng) Serrou, B. (Tumor Immunopharmacology Lab., Centre Paul Lamarque, C.R.L.C., B.P. 5054, 34 033, Montpellier Cedex, France); Rosenfeld, C.; Cupissol, D.; Goldstein, A. *Proc Am Assoc Cancer Res* 20: 32; 1979 (no refs)
- 79-3518** Sinclair Swine Melanoma Grows in the Hamster Cheek Pouch (Meeting Abstract). (Eng) Berkelhammer, J. (Cancer Res. Center, Univ. Missouri, Columbia, MO 65201). *Proc Am Assoc Cancer Res* 20: 69; 1979 (no refs)
- 79-3519** Prevention of Leukemia in Lethally Irradiated AKR Mice by CBA/H Marrow Transplantation (Meeting Abstract). (Eng) Tanaka, T. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Obata, Y.; Fernandes, G.; Onoe, K.; Stockert, E.; Good, R. A. *Proc Am Assoc Cancer Res* 20: 114; 1979 (no refs)
- 79-3520** Biological Properties of a Human Colonic Adenocarcinoma (LoVo) Grown in Athymic Mice (Meeting Abstract). (Eng) Stragand, J. J. (M. D. Anderson Hosp., Houston, TX 77030); Hickey, R. C.; Bergerat, J. P.; Hokanson, J.; Drewinko, B. *Proc Am Assoc Cancer Res* 20: 141; 1979 (no refs)
- 79-3521** Tumorigenesis of Nude Mouse Stroma from Human Tumor Xenografts (Meeting Abstract). (Eng) Goldenberg, D. M. (Div. Experimental Pathology, Univ. Kentucky Medical Center, Lexington, KY 40536); Pavia, R. A. *Proc Am Assoc Cancer Res* 20: 94; 1979 (no refs)
- 79-3522** Factors Affecting Growth Characteristics of Small Cell Carcinoma of the Lung in Nude Athymic Mice (Meeting Abstract). (Eng) Pettengill, O. S. (Dartmouth Medical Sch., Hanover, NH 03755); Curphey, T. J.; Cate, C. C.; Sorenson, G. D. *Proc Am Assoc Cancer Res* 20: 152; 1979 (no refs)

79-3523 Malignant Transformation of Murine Fibroblasts Propagated from a Human Cervical Squamous Cancer Xenografted in Nude Mice (Meeting Abstract). (Eng) Pavia, R. A. (Div. Experimental Pathology, Univ. Kentucky Medical Center, Lexington, KY 40536); Goldenberg, D. M. *In Vitro* 15(3): 227; 1979 (no refs)

79-3524 Take Rate and Growth of Human Colorectal Tumor Xenografts (Meeting Abstract). (Eng) Lapis, K. (1st Inst. Pathology, Semmelweis Medical Univ., Budapest, Hungary); Kopper, L.; Hegedus, C.; Hanh, T. V. *Proc Am Assoc Cancer Res* 20: 4; 1979 (no refs)

79-3525 In Vitro Assay for Immunosuppressive Activity of Environmental Toxicants (Meeting Abstract). (Eng) Thomson, M. A. (Iowa State Univ., Ames, IA 50011); Svec, H. J.; Quinn, L. Y. *In Vitro* 15(3): 219-220; 1979 (no refs)

79-3526 Paul-Bunnell Antigen Studies in Human and Murine Malignancy (Meeting Abstract). (Eng) Fjelde, A. (Roswell Park Memorial Inst., Buffalo, NY 14263). *In Vitro* 15(3): 171; 1979 (no refs)

79-3527 Inhibition of Antibody-induced Lysis of Human Melanoma Cells by Fibrinolysin (Plasmin) (Meeting Abstract). (Eng) Nathanson, S. D. (Div. Oncology, UCLA Sch. Medicine, Los Angeles, CA 90024); Morton, D. L.; Sarna, G. P. *Proc Am Assoc Cancer Res* 20: 217; 1979 (no refs)

79-3528 Blocking of Natural Killer Cell Mediated Cytotoxicity with Normal Serum-antibody (Meeting Abstract). (Eng) Kelloff, G. J. (NCI-NIH, Bethesda, MD 20014); Knott, W.; Dobbs, J. *Proc Am Assoc Cancer Res* 20: 106; 1979 (no refs)

79-3529 Suppressive Effect of Seminal Plasma on Tumour-associated Immunity in Prostatic Carcinoma (Meeting Abstract). (Eng) Ablin, R. J. (Cook County Hosp., Chicago, IL 60612); Bhatti, R. A.; Guinan, P. D.; Bush, I. M. *Proc Am Assoc Cancer Res* 20: 287; 1979 (2 refs)

79-3530 Granulomas in Autopsy Material of Melanoma Patients Treated with BCG Immunotherapy (Meeting Abstract). (Eng) Hatzitheofilou, C. (Div. Surgical Oncology, UCLA Sch. Medicine, Los Angeles, CA 90024); Obenchain, D.; Porter, D.; Holmes, E. C.; Morton, D. L. *Proc Am Assoc Cancer Res* 20: 329; 1979 (no refs)

See also:

*(Rev.): 79-3025, 79-3027, 79-3028, 79-3029, 79-3030, 79-3031, 79-3032, 79-3033, 79-3035, 79-3041.
*(Chem.): 79-3075, 79-3076, 79-3092, 79-3197, 79-3208, 79-3211, 79-3237, 79-3297, 79-3306, 79-3355, 79-3356.

*(Phys.): 79-3434.

*(Viral): 79-3473, 79-3478, 79-3481, 79-3482.

*(Path.): 79-3531, 79-3538, 79-3543, 79-3554, 79-3559.

*(Epid.-Biom.): 79-3567, 79-3578.

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79-3531 A Histopathological Study of Lymphoid Tissue Reaction to Metastatic Nasopharyngeal Carcinoma in Nude Mice. (Eng) Chen, H. C. (Dept. Immunology, Inst. Medical Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Kawamura, A.; Murata, M.; Hamajima, K.; Osono, M.; Suzuki, K.; Sudo, K. *IARC Sci Publ* 20: 65-84; 1978.

Lymphoid tissue reactions to metastatic nasopharyngeal carcinoma (NPC) were studied in nude mice. Successful transplants were obtained with 2/7 human NPC cultured cell lines, NPC-204 and NPC-501. Lymphatic metastases occurred during generations 11-15, and 24-30 with NPC-204 and during generations 9-14 with NPC-501. Only the regional nodes were involved. During the first few passages (prometastatic phase), large numbers of wandering macrophages appeared in the dilated medullary sinuses in response to the tumors. As the number of wandering macrophages in the medullary sinuses gradually decreased, diffuse hyperplasia of the lymphocytes occurred in regional lymph nodes. The spleen underwent extensive change, as manifested by the collapse of the splenic cords and the formation of septa studded with granulomas. These changes occurred in the absence of tumor cells, and they are regarded as a defense reaction of the reticuloendothelial system. Neighboring lymph nodes were also often studded with epithelioid cell granulomas, formed by the aggregation of macrophages around nuclear debris in the subcortical areas. It is assumed that the clumps of debris are the remnants of metastatic cancer cells killed by macrophages or sensitized lymphocytes. It would appear, therefore, that macrophages can replace T lymphocytes in killing tumor cells and in protecting the lymph nodes from the spread of metastases. (36 refs)

79-3532 Histopathological Types of Nasopharyngeal Carcinoma in a Low-Risk Area: Japan. (Eng) Sugano, H. (Dept. Pathology, Cancer Inst., 1-37 Kami-Ikebukuro, Toshima-ku, Tokyo 170, Japan); Sakamoto, G.; Sawaki, S.; Hirayama, T. *IARC Sci Publ* 20: 27-39; 1978.

Biopsy material from 816 Japanese patients with nasopharyngeal carcinoma (NPC) was examined histopathologically. A comparative study of NPC among the Taiwanese and an immunoserological study were also performed. Malignant nasopharyngeal tumors in Japanese patients were characterized by a predominance of squamous cell carcinoma, especially of the poorly differentiated type, and a relatively high frequency of malignant

lymphoma. Of 731 NPC cases, 635 were poorly differentiated and 96 were well-differentiated squamous cell carcinomas. The former included 45.8% spindle-polygonal cell carcinomas, 26.6% transitional cell carcinomas, and 14.3% lymphoepitheliomas. Well-differentiated carcinomas were infrequent in both the Japanese and Taiwanese, especially in the latter. Poorly differentiated carcinomas, especially transitional cell carcinomas and lymphoepitheliomas, were more frequent among the Taiwanese. In 84 Japanese NPC patients tested, the anti-Epstein Barr viral capsid antigen (VCA) antibody titer was very high in patients with lymphoepitheliomas, 80% of whom showed positive reactions. Positive rates for other types ranged from 33% (well-differentiated carcinoma) to 47% (spindle-polygonal cell and transitional cell carcinomas). However, in a similar study of 205 Taiwanese NPC patients, anti-VCA antibody titers and positivity rates were almost the same in all subgroups of poorly differentiated carcinoma. The serological results obtained with the Japanese study suggest that EBV genomes in NPC cells vary with the grade of differentiation of NPC. (18 refs)

79-3533 Paraneoplastic Acrokeratosis. (Ger) Bazex, A. (Clinique Dermatologique, Hopital de Purpan, F-31000 Toulouse, France). *Hautarzt* 30(3): 119-123; 1979.

The general clinical aspects of paraneoplastic acrokeratosis (PA) are described. PA is a distinctive dermatosis occurring exclusively in patients with primary malignant tumors of the upper respiratory and digestive tracts and in those with cervical and mediastinal metastases. The primary tumor is usually a spindle cell carcinoma, at times an anaplastic carcinoma, and, on rare occasions, an adenocarcinoma. PA can precede the symptoms of tumor. PA is characterized by centripetally spreading erythematous, squamous, and hyperkeratotic plaques that heal only after removal of the carcinoma. PA may be caused by an allergic or toxic skin reaction to substances produced by the tumor cells. (37 refs)

79-3534 Bazex-Dupre Paraneoplastic Acrokeratosis. (Fre) Jeune, R. (Clinique Dermatologique, Hopital E.-Herriot, F 68374 Lyon Cedex 2, France); Thivolet, J.; Chabbeau, G.; Descos, L. *Lyon Med* 241(4): 235-237; 1979.

Bezex-Dupre paraneoplastic acrokeratosis (PA) was

diagnosed in two men aged 65 and 66 yr. An epithelioma was found in the piriform sinus of one patient, a poorly differentiated malpighian epithelioma in the thymus of the other. Bazex-Dupre PA is often concomitant with and a precursor of cancer of the digestive and respiratory tracts. (4 refs)

- 79-3535 Dysphagia.** (Eng) Castell, D. O. (Digestive Diseases Branch, Internal Medicine Service, Natl. Naval Medical Center, Bethesda, MD 20014); Knuff, T. E.; Brown, F. C.; Gerhardt, D.C.; Burns, T. W.; Gaskins, R. D. *Gastroenterology* 76(5, part 1): 1015-1024; 1979.

The case history of a patient with long-standing gastroesophageal reflux resulting in peptic stricture and dysphagia is presented. Metaplastic changes typical of Barrett's esophagus occurred in the esophageal mucosa, with the subsequent development of midesophageal adenocarcinoma (mid-EAC). The patient had three recurrences of dysphagia. The third recurrence also affected the ingestion of liquids, which is unusual for a peptic stricture. Coupled with a 33-lb wt loss, it suggested the possibility of a malignant process. Literature review indicates that stricture formation occurs frequently in the setting of a columnar-lined lower esophagus. Twenty-eight of 304 patients with a columnar-lined lower esophagus developed EA. Multiple biopsies of the esophagus at the time of previous stricture formation might have revealed a columnar-lined lower esophagus, and a closer surveillance program might have detected the EA in an earlier stage. Patients with Barrett's esophagus also have a propensity to develop EA. Esophageal metaplasia of the type characteristic of Barrett's esophagus is recognized as a premalignant condition for EA. Esophageal carcinoma is occurring with increasing frequency in the American population, and it has an extremely grave prognosis. (49 refs)

- 79-3536 Malignant Melanoma of the Small Intestine Arising from a Regressive Skin Lesion.** (Fre) Saby, R. (Services de chirurgie, Hopital de Libourne, 33500 Libourne, France); Chate, M.; Hypousteguy, D.; Baste, J. C. *Chirurgie* 104(10): 913-920; 1978.

A large malignant melanoma of the small intestine was removed from a 51-yr-old man undergoing laparotomy for acute abdomen. Mesenteric lymph node metastases were also removed. The patient is in good condition 4 yr after surgery and chemotherapy. He had had a small brownish tumor under the right ear that regressed spontaneously within a few months 3 yr prior to discovery of the melanoma. Biopsy from the tumor site suggested a regressed nevus. Thus, the intestinal melanoma may have been a primary tumor or one that was secondary to the skin melanoma. (no refs)

- 79-3537 Recurrent Stupor, Diabetes, Hypothyroidism and Liver Disease in an Elderly Woman.** (Eng) Axelrod, L. (Massachusetts General Hosp., Boston, MA); Vickery, A. L. *N Engl J Med* 300(17): 969-976; 1979.

The case report of a patient with idiopathic hemochromatosis plus hepatic failure, hepatic encephalopathy, diabetes mellitus, cardiomyopathy, and, possibly, pituitary involvement manifested as hypothyroidism is presented. The patient was a 71-yr-old woman with a 10-yr history of recurrent confusion, stupor, and coma. At autopsy, micronodular cirrhosis and hepatocellular carcinoma were evident. (30 refs)

- 79-3538 Hypothyroidism Significantly Prolongs the Survival of Rats Bearing Morris Hepatoma #44 (Meeting Abstract).** (Eng) Mishkin, S. (Dept. Medicine, Royal Victoria Hosp., McGill Univ. Clinic, Montreal H3A 1A1, Canada); Huang, S. N.; Morris, H. P.; Yalovsky, M.; Murthy, P. V. *Proc Am Assoc Cancer Res* 20: 74; 1979 (1 ref)

- 79-3539 Nephroblastoma of the Renal Pelvis Mixed with Sarcoma Botryoides.** (Ger) Heising, J. (Urologische Universitätsklinik, Universität Köln, D-5000 Köln 41, W. Germany); Engelking, R.; Bohr, M.; Lennartz, K. J.; Fuhrmann, U.; Huls, W.; Rottinger, E. M. *Urologe [A]* 18(2): 68-72; 1979.

Nephroblastoma of the renal pelvis with a sarcoma botryoides component was found in a 4-yr-old girl. The histogenesis of this dysontogenetic tumor is probably due to the altered growth and differentiation of the metanephrogenic blastoma and, partly, of the ureteral bud. (10 refs)

- 79-3540 Fibrosarcoma in a Renal Oncocytoma.** (Ger) Blessing, M. H. (Pathologisches Institut der Krankenhauser der Stadt Köln, Ostmerheimer Strasse 200, D-5000 Cologne 91, W. Germany); Burkert, E.; Lehmann, H. D. *Urologe [A]* 18(2): 99-101; 1979.

A tumor of the left kidney was found in a 69-yr-old man. He agreed to undergo surgery 3.5 yr later. A large tumor consisting of mixed oncocytic adenoma and fibrosarcoma was found. The malignant tumor is believed to have originated from necrotic parts of the benign adenoma as a result of excessive regeneration. (15 refs)

- 79-3541 Malignant Transformation of Aneurysmal Bone Cysts in Childhood.** (Ger) Angerpoint-

PATHOGENESIS

ner, T. (Kinderchirurgische Klinik, Dr. v. Haunersches Kinderspital, Lindwurmstrasse 4, 8000 Munich 2, W. Germany); Engert, J.; Konrad, E.; Meister, P. *Z Kinderchir* 26(2): 143-149; 1979.

An aneurysmal bone cyst of the right tibia was diagnosed in a 3.5-yr-old girl in connection with a pathological fracture. Even though the patient was not irradiated, clinical and histological signs of malignancy increased rapidly, forcing amputation 8 mo later. Polynuclear giant cells, spindle cells, and increased cellular atypia were seen. The tumor was diagnosed as a poorly differentiated osteosarcoma. The patient received chemotherapy after the amputation, and she is in good health 4 yr later. The findings suggest there may be a malignant aneurysmal bone cyst with special biological properties. (26 refs)

79-3542 Medullary Carcinoma of the Thyroid: Study of a Kindred. (Fre) Allannic, H. (Service de Clinique Therapeutique C, Hopital Pontchaillou, F 35000 Rennes, France); Lorcy, Y.; Cornec, A.; Le Marec, B.; Calmettes, C. *Ann Endocrinol (Paris)* 40(1): 61-62; 1979.

Two proved and two probable cases of medullary carcinoma of the thyroid (MCT) without pheochromocytoma, hyperparathyroidism, or Cushing's disease were found in one family. Four other members suffered from intestinal occlusion in childhood. The findings indicate that MCT is transmitted as an autosomal dominant trait with a high degree of penetrance. (2 refs)

79-3543 Immunochemical Biological Markers and Hereditary Cancer Risk. (Eng) Guirgis, H. A. (Dept. Community and Environmental Medicine, Univ. California Sch. Medicine, Irvine, CA 92717); Lynch, H. T.; Harris, R. E.; Vandevoorde, J. P. *Scand J Immunol [Suppl]* 8(8): 647-652; 1978.

Several putative biological markers of cancer risk (carcinoembryonic antigen, α -fetoprotein, aryl hydrocarbon hydroxylase, percentage of T and B lymphocytes and IgA, IgM, and IgG) were studied in relatives at high genetic risk for cancer within a kindred manifesting the cancer family syndrome. Thirteen of the 19 individuals studied were unaffected progeny of affected direct genetic line parents. The remaining six individuals had cancer. A predictive index of cancer susceptibility that incorporates five of the putative markers was derived. The number of individuals manifesting a significant index score was compared with the number expected to carry the cancer-predisposing gene among the 13 unaffected progeny of affected parents. The observed number of aberrant index scores agreed precisely with that expected based upon gene segregation and the age distribution of the sample. The proposed index appears to provide predictability of cancer risk status in accord with

mathematical expectations for a simple genetic model. (16 refs)

79-3544 11p Chromosome Deletion in Four Patients with Aniridia and Wilms' Tumor (Meeting Abstract). (Eng) Bader, J. L. (NCI, Bethesda, MD 20014); Li, F. P.; Gerald, P. S.; Leikin, S. L.; Randolph, J. G. *Proc Am Assoc Cancer Res* 20: 210; 1979 (no refs)

79-3545 Chromosomal Characterization of Children with Neuroblastoma and Other Neoplasias (Meeting Abstract). (Eng) Moorhead, P. S. (Children's Cancer Res. Center, Philadelphia, PA 19104); Evans, A. *Proc Am Assoc Cancer Res* 20: 42; 1979 (no refs)

79-3546 Fibroblasts from Patients with Hereditary Retinoblastoma are Abnormally Radiosensitive (Meeting Abstract). (Eng) Weichselbaum, R. R. (Harvard Univ., Sch. Public Health, Boston, MA 02115); Little, J. B.; Nove, J.; Albert, D. *Proc Am Assoc Cancer Res* 20: 73; 1979 (no refs)

79-3547 The Origin of Double Minutes from an HSR-Marker Chromosome in Human Neuroblastoma Cell Hybrids (Meeting Abstract). (Eng) Balaban-Malenbaum, G. (Children's Cancer Res. Center, Philadelphia, PA 19104); Gilbert, F. *Proc Am Assoc Cancer Res* 20: 126; 1979 (1 ref)

79-3548 The Occurrence of Five Paraganglion Tumors in a Three Brother Sibship Over an Eleven Year Period (Meeting Abstract). (Eng) Poster, D. S. (Dept. Medicine A, Roswell Park Memorial Inst., Buffalo, NY 14263); Carlson, R. *Proc Am Assoc Cancer Res* 20: 357; 1979 (no refs)

79-3549 Cerebromeningeal Involvement in Acute Myeloblastic Leukemia and Myeloproliferative Syndromes. Cytological, Histological and Clinical Study of 62 Cases. (Fre) Henin, D. (Service Central d'Anatomie et de Cytologie Pathologiques, Hopital Beaujon, 100 Boulevard du General Leclerc, F 92118 Clichy, France); Slabodsky-Brousse, N.; Renoux, M.; Dhermy, D.; Bernard, J. F. *Nouv Presse Med* 8(10): 751-754; 1979.

Forty-six patients (30 men, 16 women, av age 52.9 yr) with

acute myeloblastic leukemia (AML) and 16 patients (10 men and 6 women, av age 57.6 yr) with myeloproliferative syndromes (MPS) were examined for cerebrospinal involvement (CMI). CMI was proved anatomically in 28/62 patients, but only 18/28 patients had neurological signs; however, patients without CMI had neurological signs. Examination of the cerebrospinal fluid of 22 AML patients and 4 MPS patients revealed blastic infiltration in 15. Autopsy examination of 30 AML patients and 15 MPS patients revealed CMI in 12 and 6 patients, respectively. The arachnoidea was involved in 18 cases, the dura mater in 10, and the cerebral parenchyma in 5. Intracerebral hemorrhages were found at autopsy in 19/27 patients without CMI. (20 refs)

79-3550 Increased In Vitro Radiosensitivity in Some Skin Fibroblast Cultures From a Family with Multiple Cases of Acute Myelogenous Leukemia (AML) (Meeting Abstract). (Eng) Bech-Hansen, N. T. (Atomic Energy Canada Limited, Chalk River, Ontario, K0J 1J0, Canada); Sell, B. M.; Anderson, A. K.; Mulvihill, J. J. *Proc Am Assoc Cancer Res* 20: 78; 1979 (2 refs)

79-3551 Non-random Clonal Evolution in 45 Cases of Chronic Myeloid Leukemia. (Eng) Stoll, C. (Institut de Puericulture, 23 rue de la Porte de l'Hopital, 67000 Strasbourg, France); Oberling, F. *Leuk Res* 3(2): 61-66; 1979.

Chromosome analyses were made of the blood and bone marrow of 52 chronic myeloid leukemia (CML) patients during blastic crisis. Most of the analyses were performed with the use of the RHG-banding technique (analysis of R bands by heat treatment and Giemsa staining). Forty-five patients had other chromosomal aberrations in addition to the Philadelphia chromosome (Ph¹). A second Ph¹ chromosome was found in 28 patients. An additional long arm of chromosome 17 [trisomy 17, or i(17q)] was seen in 29 patients and a trisomy 8 in 13. These three main aberrations occurred alone or together in the same cell. Thirteen patients had two Ph¹ chromosomes and trisomy 17q without trisomy 8. The results indicate that chromosome involvement in the development of CML is nonrandom. Karyotypic evolution usually proceeded by the addition of chromosomes. Seventeen patients had hyperdiploidy, 1 hypodiploidy, and 27 pseudodiploidy. A clonal evolution could be demonstrated in all 45 patients with chromosome changes in addition to Ph¹. Clonal evolution was simple in 32 patients, following a linear pathway; it was the result of successive mitotic nondisjunctions. In 13 patients, clonal evolution was more complex, following a divergent pathway and resulting in several subpopulations. (17 refs)

79-3552 Follicular Mucinosis (Lymphoma) Occurring on the Head and Neck. (Eng) Wilkinson, J. D.

(Slade Hosp., Headington, Oxford OX3 7JH, England); Dawber, R. P.; Ryan, T. J.; Millard, P. R. *J R Soc Med* 72(4): 281-282; 1979.

The case report of a 71-yr-old woman with lymphoma associated with follicular mucinosis of the head and neck is presented. A scaly erythematous rash on the face was progressively replaced by waxy indurated areas with gelatinous plaques, prominent cysts, and follicular plugging. The initial biopsy showed spongiotic vesicles and a patchy infiltrate of mononuclear cells with some pleomorphic forms. A biopsy 5 mo later demonstrated a substantial mononuclear cell infiltrate surrounding the follicles and extending deep into the dermis. Among these cells were pleomorphic atypical forms. (3 refs)

79-3553 Confirmatory Evidence of an Association Between Infectious Mononucleosis and Hematopoietic Malignancy (Meeting Abstract). (Eng) Fjelde, A. (State Univ. New York, Buffalo, NY); Kano, K.; Milgrom, F.; Reynhout, J.; Silverstein, D. *Proc Am Assoc Cancer Res* 20: 102; 1979 (1 ref)

79-3554 Evolution of Giant Lymph Node Hyperplasia (GLNH) to Immunoblastic Sarcoma (IBS) (Meeting Abstract). (Eng) Hopkins, L. A. (Div. Hematology-Oncology, Univ. Cincinnati Coll. Medicine, Cincinnati, OH 45267); Unger, L. M.; Martelo, O. J. *Proc Am Assoc Cancer Res* 20: 352; 1979 (no refs)

79-3555 Skin Metastases of an Ovarian Carcinoma Mimicking Erythema Annulare. (Ger) Bernecker, H. A. (Dermatologische und Allergologische Abteilung, Städtisches Krankenhaus München-Schwabing, Kölner Platz 1, D-8000 Munich 40, W. Germany); Bachmann, W. *Hautarzt* 30(3): 164-165; 1979.

Erythema annulare was found in a 60-yr-old woman 2 mo after the removal of a large ovarian cystoma, at which time tumor cells were found in the ascitic fluid. Biopsy revealed carcinoma cells in the erythema annulare, which was thus a metastasis of an undetected carcinomatous component of the cystoma. (5 refs)

79-3556 Turner's Syndrome: A Six-Year Experience. (Eng) Stenchever, M. A. (Dept. Obstetrics and Gynecology, Univ. Washington Sch. Medicine, Seattle, WA 98195); Parks, K. J.; Stenchever, M. R. *Trans Am Gynecol Soc* 101: 167-172; 1978.

A 6-yr experience of one genetics laboratory with patients

PATHOGENESIS

referred for evaluation of possible Turner's syndrome is reported. One of the 13 individuals with karyotypic evidence of the syndrome developed a well-differentiated endometrial adenocarcinoma while on estrogen replacement therapy. This patient, a 27-yr-old woman, had been treated for 14 yr with diethylstilbestrol (25 days/mo). (19 refs)

79-3557 Increased Frequency of Splenic Metastases in Breast Cancer (Meeting Abstract). (Eng) Buchsbaum, R. (Univ. California Irvine Medical Center, Orange, CA 92668); Souadjian, J. V.; Padova, J. A.; Armentrout, S. A. *Proc Am Assoc Cancer Res* 20: 421; 1979 (no refs)

79-3558 Metastatic Colonisation Potential of Primary Mammary Tumour Cells in Mice (Meeting Abstract). (Eng) Tarin, D. (Royal Postgraduate Medical Sch., London, W12, England); Price, J. E. *Proc Am Assoc Cancer Res* 20: 249; 1979 (no refs)

79-3559 Restoration of Hematogenously Metastasizing Capacity of Rat Mammary Tumors (MT) by Athymic Nude Mice: Immunological Manifestation of Tumor-Host Interaction (Meeting Abstract). (Eng) Kim, U. (Roswell Park Memorial Inst., Buffalo, NY 14263); Shin, S. I.; Freedman, V. H. *Proc Am Assoc Cancer Res* 20: 122; 1979 (no refs)

79-3560 Modulation of the Metastatic Frequency of a Murine Mammary Adenocarcinoma by a Synthetic Cannabinoid Drug (Meeting Abstract). (Eng) Levy, J. A. (Roger Williams General Hosp., 825 Chalkstone Ave., Providence, RI 02908); Heppner, G. H. *Proc Am Assoc Cancer Res* 20: 155; 1979 (no refs)

79-3561 Ultrastructural Study of Membrane Glycocalyx in Primary and Metastatic Rat Mammary Carcinoma (Meeting Abstract). (Eng) Ghosh, L. (Abraham Lincoln Sch. Medicine, Univ. Illinois, Chicago, IL 60612); Nassauer, J.; Faiferman, I. *Proc Am Assoc Cancer Res* 20: 235; 1979 (no refs)

79-3562 Noninvasiveness of Human Tumors in the Athymic (Nude) Mouse (Meeting Abstract). (Eng) DeVore, D. P. (Battelle Memorial Inst., Columbus, OH 43201); Houchens, D. P.; Ovejera, A. A.; Hutson, T. B. *Proc Am Assoc Cancer Res* 20: 5; 1979 (no refs)

79-3563 Possible Role of Lysosomal Enzymes in the Initiation of Metastatic Spread (Meeting Abstract). (Eng) Dobrossy, L. (Dept. Experimental Therapeutics, Roswell Park Memorial Inst., Buffalo, NY 14263); Pavelic, Z. P.; Bernacki, R. J. *Proc Am Assoc Cancer Res* 20: 178; 1979 (no refs)

79-3564 Somatic Genetics of Progressive Neoplastic Transformation (Meeting Abstract). (Eng) Crawford, B. D. (Div. Biophysics, Johns Hopkins Univ., Baltimore, MD 21205); Barrett, J. C.; Ts'o, P. O. *Proc Am Assoc Cancer Res* 20: 135; 1979 (no refs)

See also:

*(Rev.): 79-3002, 79-3032, 79-3034, 79-3035, 79-3036, 79-3037, 79-3038, 79-3044, 79-3047, 79-3048, 79-3049.

*(Chem.): 79-3050, 79-3054, 79-3060, 79-3063, 79-3069, 79-3075, 79-3077, 79-3085, 79-3093, 79-3097, 79-3110, 79-3114, 79-3117, 79-3120, 79-3121, 79-3122, 79-3136, 79-3147, 79-3152, 79-3162, 79-3175, 79-3176, 79-3199, 79-3212, 79-3217, 79-3219, 79-3231, 79-3260, 79-3272, 79-3279, 79-3293, 79-3299, 79-3302, 79-3305, 79-3330, 79-3334, 79-3338, 79-3339, 79-3340, 79-3343, 79-3349, 79-3353, 79-3354, 79-3357, 79-3358, 79-3361, 79-3362, 79-3365, 79-3370.

*(Phys.): 79-3429, 79-3431, 79-3436, 79-3443, 79-3446, 79-3448, 79-3454, 79-3455, 79-3456, 79-3457, 79-3459.

*(Viral): 79-3461, 79-3475, 79-3488, 79-3493, 79-3496, 79-3497.

*(Immun.): 79-3508, 79-3510, 79-3511.

*(Epid.-Biom.): 79-3567, 79-3569, 79-3570, 79-3574, 79-3579, 79-3580, 79-3584.

EPIDEMIOLOGY AND BIOMETRY

- 79-3565 Pathologic and Demographic Studies on Nasopharyngeal Carcinoma (NPC) (Meeting Abstract). (Eng) Levine, P. H. (NCI, Bethesda, MD 20014); Easton, J. M.; Hyams, V. J.; Connelly, R. R. *Proc Am Assoc Cancer Res* 20: 269; 1979 (no refs)
- 79-3566 Epstein-Barr Virus DNA in North American Nasopharyngeal Carcinoma (Meeting Abstract). (Eng) Glaser, R. (Ohio State Univ., Columbus, OH 43210); Nonoyama, M.; Szymanowski, R.; Graham, W. *Proc Am Assoc Cancer Res* 20: 42; 1979 (no refs)
- 79-3567 Etiology of Gastric Cancer (G Ca) (Meeting Abstract). (Eng) Goedert, J. (Vincent T. Lombardi Cancer Res. Center, Georgetown Univ. Hosp., Washington, DC 20007); McKeen, E.; Rosenoff, S.; Schein, P. *Proc Am Assoc Cancer Res* 20: 416; 1979 (1 ref)
- 79-3568 Interrelations of Tobacco, Alcohol and Diet in Contributing to Cancer Incidence among Five Ethnic Groups in Hawaii (Meeting Abstract). (Eng) Hinds, M. W. (Epidemiology Program, Cancer Center of Hawaii, Univ. Hawaii, Honolulu, HI 96822); Kolonel, L. N.; Nomura, A. M. *Proc Am Assoc Cancer Res* 20: 182; 1979 (no refs)
- 79-3569 Malignant Melanoma in Persons under 20 Years of Age: An Epidemiologic Study (Meeting Abstract). (Eng) Bader, J. L. (NCI, Bethesda, MD 20014); Li, F. P.; Miller, R. W.; Roegner, R. *Proc Am Assoc Cancer Res* 20: 316; 1979 (no refs)
- 79-3570 Spectrum and Natural History of Head and Neck (H&N) Neoplasms Following Radiation (RTX) Given for Benign Conditions (Meeting Abstract). (Eng) Khandekar, J. D. (Evanston Hosp., Evanston, IL 60201); Scanlon, E. F.; Murphy, E. D.; Garces, R. M. *Proc Am Assoc Cancer Res* 20: 397; 1979 (2 refs)
- 79-3571 Incidence of Second Malignancies in Hodgkin's Disease (HD) After Various Forms of Treatment (Meeting Abstract). (Eng) Valagussa, P. (Istituto Nazionale Tumori, Milan, Italy 20133); Kenda, R.; Fossati Bellani, F.; Franchi, F.; Banfi, A.; Rilke, F.; Bonadonna, G. *Proc Am Assoc Cancer Res* 20: 360; 1979 (no refs)
- 79-3572 Acute Myeloid Leukemia (AML) Occurring During Complete Remission (CR) in Hodgkin's Disease (Meeting Abstract). (Eng) Pajak, T. F. (Cancer and Leukemia Group B, Scarsdale, NY 10583); Nissen, N. I.; Stutzman, L.; Hoogstraten, B.; Cooper, M. R.; Glowienka, L. P.; Glidewell, O.; Glicksman, A. *Proc Am Assoc Cancer Res* 20: 394; 1979 (no refs)
- 79-3573 The Risk of Subsequent Osteosarcoma in Survivors of Ewing's Sarcoma (Meeting Abstract). (Eng) Strong, L. C. (Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Osborne, B. M.; Chan, R. C. *Proc Am Assoc Cancer Res* 20: 362; 1979 (1 ref)
- 79-3574 Bone Sarcoma (BS) as Second Malignant Neoplasm (SMN) in Children: Influence of Radiation (RT) and Predisposition (Meeting Abstract). (Eng) Meadows, A. T. (Late Effects Study Group (LESG), Philadelphia, PA 19104); Strong, L. C.; Li, F. P.; D'Angio, G. J.; Schweisguth, O.; Freeman, A.; Jenkin, R. D.; Morris-Jones, P. *Proc Am Assoc Cancer Res* 20: 126; 1979 (no refs)
- 79-3575 Multiple Myeloma and Acute Leukemia: Review of 104 Cases (Meeting Abstract). (Eng) Rosner, F. (Queens Hosp. Center Affil. Long Island Jewish-Hillside Medical Center, Jamaica, NY 11432). *Proc Am Assoc Cancer Res* 20: 299; 1979 (no refs)
- 79-3576 Adult Acute Leukemia in New Mexico 1970-1978: A Review of Southwest Oncology Study

Group Results (Meeting Abstract). (Eng) Saiki, J. H. (Univ. New Mexico Sch. Medicine, Albuquerque, NM); Saiers, H. J.; Hardy, W. R.; Wimer, B. M.; Rembe, A. M.; Weiler, R. J.; Shaw, M. T. *Proc Am Assoc Cancer Res* 20: 397; 1979 (no refs)

79-3577 Testicular Carcinoma - An Increased Incidence (Meeting Abstract). (Eng) Jaffrey, I. (Palisades Oncology Assoc., Bardonia, NY 10954). *Proc Am Assoc Cancer Res* 20: 373; 1979 (no refs)

79-3578 Clinical and Laboratory Studies on the Etiology and Control of Rapidly Progressing Breast Cancer in Tunisia (Meeting Abstract). (Eng) Mourali, N. (Inst. Salah Azaiz, Tunis, Tunisia); Tabbane, F.; Muenz, L.; Levine, P. H.; Bekesi, J. G. *Proc Am Assoc Cancer Res* 20: 269; 1979 (no refs)

79-3579 Pragmatic Basis for Identification of Familial Breast Cancer in an Oncology Clinic (Meeting Abstract). (Eng) Lynch, H. T. (Creighton Univ. Sch. Medicine, Omaha, NE 68178); Follett, K. L.; Lynch, P. M.; Albano, W. A.; Mailliard, J. A.; Pierson, R. L.; McKenna, P. J.; Black, L. E.; Novak, B. A.; Lynch, J. F.; Fain, P. R.; Fairman, A. M.; Blair, D. A. *Proc Am Assoc Cancer Res* 20: 315; 1979 (1 ref)

79-3580 Risk Factors Determined for Benign Breast Disease Compared to Those Known for Breast Cancer (Meeting Abstract). (Eng) Buehring, G. C. (Sch. Public Health, Univ. California, Berkeley, CA 94720). *Proc Am Assoc Cancer Res* 20: 137; 1979 (no refs)

79-3581 Human Population with Differing Breast Cancer Risk Exhibit Differences in Rhythms of Prolactin, Aldosterone and Other Hormones Detectable Only at Certain Sampling Times Spaced by Pertinence Rather Than Convenience (Meeting Abstract). (Eng) Haus, E. (Univ. Minnesota, Minneapolis, MN); Halberg, F.; Kawasaki, T.; Lakatua, D. J.; Ueno, M.; Uezono, K.; Omae, T. *Proc Am Assoc Cancer Res* 20: 111; 1979 (no refs)

79-3582 Conjugated Estrogen (CE) Use and the Risk of Breast Cancer in a Prepaid Health Plan (Meeting Abstract). (Eng) Glass, A. (Kaiser Foundation Res. Center, 4610 S.E. Belmont, Portland, OR 97215); Hoover, R.; Finkle, W.; Azevedo, D. *Proc Am Assoc Cancer Res* 20: 424; 1979 (no refs)

79-3583 Spontaneous Genital Cancers in Hens. A Study of Incidence, Morphogenesis, Hormonal Background, and Steroid Receptors. (Meeting Abstract). (Eng) Fredrickson, T. N. (Univ. Connecticut, Storrs, CT 06268); Fournier, D. J.; Esber, H. J.; Okulicz, W. C. *Proc Am Assoc Cancer Res* 20: 243; 1979 (no refs)

79-3584 The Multihit Model of Carcinogenesis: Application to Colon Cancer Data from the Third National Cancer Survey (Meeting Abstract). (Eng) Sutherland, J. V. (Univ. Colorado Medical Center, Denver, CO 80262); Bailer, J. C. *Proc Am Assoc Cancer Res* 20: 77; 1979 (no refs)

79-3585 Amebic Pseudotumors in Pseudobranchs of Pacific Cod, *Gadus Macrocephalus* (Meeting Abstract). (Eng) Dawe, C. J. (NCI, Bethesda, MD 20014); Bagshaw, J.; Poore, C. M. *Proc Am Assoc Cancer Res* 20: 245; 1979 (1 ref)

See also:

*(Rev.): 79-3005, 79-3006, 79-3007, 79-3010, 79-3011, 79-3013, 79-3014, 79-3015, 79-3019, 79-3029, 79-3030, 79-3039, 79-3040, 79-3041, 79-3042, 79-3043, 79-3044, 79-3045, 79-3046.

*(Chem.): 79-3058, 79-3084, 79-3115, 79-3168, 79-3216, 79-3339, 79-3351, 79-3364, 79-3371, 79-3372.

*(Phys.): 79-3431, 79-3445, 79-3460.

*(Immun.): 79-3500.

*(Path.): 79-3543.

MISCELLANEOUS

- 79-3586 Polyamine Content and Release during the Proliferation of Burkitt's Lymphoma Cells In Vitro.** (Eng) Woo, K. B. (Biological Markers Lab., NCI Frederick Cancer Res. Center, Frederick, MD 21701); Perini, F.; Sadow, J.; Sullivan, C.; Funkhouser, W. *Cancer Res* 39(7): 2429-2435; 1979.

The cellular content and excretion of polyamines in relation to the cell cycle and proliferation kinetics of Burkitt's lymphoma cells in vitro were investigated. Quantitative relationships were established between the cellular content of polyamines and the growth kinetic parameters, including specific growth rate, labeling index, and cell viability. The intracellular content of putrescine, spermidine, and spermine correlated significantly with the specific growth rate, suggesting that all the polyamines actively participate in the process of Burkitt's lymphoma cell proliferation. A negative correlation was found between the labeling index and the intracellular content of putrescine ($r = -0.893$); a positive correlation was observed between the labeling index and the ratios of spermidine to putrescine (0.888) and spermine to putrescine (0.855). In addition, the extracellular content of putrescine showed a positive correlation with the labeling index (0.613). The percentage of dead cells determined by trypan blue exclusion exhibited a high positive correlation with the intracellular content of putrescine (0.912). Examination of the distribution pattern of polyamines with respect to the fraction of cells in each cell cycle stage during a 10-day growth period revealed that the accumulation of cells in G_2 was associated with a reduction of intracellular levels of spermidine and spermine. In cultures synchronized by double thymidine blockade, maximal levels of intracellular spermidine and spermine occurred in S phase. Intracellular putrescine levels were lowest when the DNA content was lowest in early and mid-S phase, and they were highest when the DNA content was highest in late S and early $G_2 + M$. (39 refs)

- 79-3587 Differences in the Peripheries of Walker Cancer Cells Growing in Different Sites in the Rat.** (Eng) Weiss, L. (Dept. Environmental Pathology, Roswell Park Memorial Inst., Buffalo, NY 14263); Harlos, J. P. *Cancer Res* 39(7, part 1): 2481-2485; 1979.

Walker 265 carcinosarcoma cells growing in the ascitic form and following direct injection of 10^6 cells in the livers or in sc sites in Sprague-Dawley rats had significantly higher anodic electrophoretic mobilities than did cells derived from the same source but growing in kidneys and

spleens. Following incubation with neuraminidase, the cancer cells from the kidneys and spleen lost significantly less net surface negativity than did cells growing in the other three sites. These kidney- and spleen-associated differences were not demonstrably due to preexisting, electrokinetic subpopulations of cancer cells within the original ascites tumor; they were maintained on organ-to-organ passage and were reversed on reconversion of the tumors to ascitic form. The differences between primary cancers and their metastases are conceivably due to site-induced modulation as distinct from preexisting metastatic subpopulations. There is evidence that cancer cells in some primary tumors are different from those in their metastases, at least in some organs. The possibilities arise that specific organs are selectively seeded by preexisting subpopulations of cancer cells from within the primary tumor or that the seeding is random. In either case, cells in metastases are different because they are located in specific metastatic sites. (38 refs)

- 79-3588 Plasminogen Activation Transforms the Morphology of Quiescent 3T3 Cell Monolayers and Initiates Growth.** (Eng) Whur, P. (Marie Curie Memorial Foundation, Oxted, Surrey, England); Silcox, J. J.; Boston, J. A.; Williams, D. C. *Br J Cancer* 39(6): 718-730; 1979.

Attempts were made to induce transformation-like morphology and growth in confluent quiescent 3T3 cells in tissue culture by activating plasminogen (PM) via the addition of urokinase (UK) and PM or PM-containing acid-treated serum, or plasmin. Cells in medium supplemented with UK alone formed well-ordered confluent monolayers. When UK and PM were both added to PM-free acid-treated horse serum, there was cell elongation and multi-layering followed by the formation of thick cords of cells which left large areas of the growth surface almost devoid of cells. These changes were associated with growth to significantly higher densities than those of the controls. Similar but more rapid growth was seen when acid-treated horse serum was used instead of PM supplementation, and similar but less marked changes were seen in the presence of plasmin. There was a partial reversal of the supplement-induced disruption when the cells were returned to medium containing acid-treated PM-free serum. The data indicate that well-ordered monolayer morphology and quiescence in 3T3 cells at confluence are dependent on the absence of PM activation. (54 refs)

79-3589 Biochemical and Genetic Evidence for Two Extracellular Adenosine 3':5'-Monophosphate Phosphodiesterases in *Dictyostelium purpureum*. (Eng) Tsang, A. S. (Imperial Cancer Res. Fund, Mill Hill Labs., Burtonhole Lane, London NW 1AD, England); Coukell, M. B. *Eur J Biochem* 95(2): 407-417; 1979.

Two extracellular AMP phosphodiesterases (PDE-1 and PDE-2) were purified from growth media of cultures of the cellular slime mold *Dictyostelium purpureum*. Both enzymes have a broad pH optimum of 7.5-8.6, but they differ in mol wt, stability, kinetic behavior, and sensitivity to inactivation by the PDE inhibitor of *D. discoideum*. The polypeptides associated with the two enzyme activities were identified on two-dimensional gels. Under these conditions, the polypeptides associated with PDE-1 activity had a mol wt of 60,000 and an apparent isoelectric point of pH 8.5. Two polypeptides (mol wts 50,000 and 48,000) were associated with PDE-2 activity. These two polypeptides had the same isoelectric point of about pH 7.5 and very similar peptide maps. With Coomassie blue stain, the ratio of the 50,000-mol wt polypeptide to the 48,000-mol wt polypeptide was 3:1; the same polypeptides appeared to be present in a 6:1 ratio when they were stained with PAS reagent. These results suggest that the major difference between the 50,000- and 48,000-mol wt polypeptides is in the size of their carbohydrate moieties. Several developmental mutants were isolated that synthesize the PDE's abnormally. Results of an examination of enzyme production in two of these mutants provided further evidence for the existence of two extracellular AMP PDE's in *D. purpureum*. In addition, these results revealed that PDE-1 is synthesized during vegetative growth, whereas PDE-2 is synthesized toward the end of the growth phase and during early development. (40 refs)

79-3590 Adenosine 3':5' Cyclic Monophosphate and Myeloid Leukemic Cell Proliferation In Vitro (Meeting Abstract). (Eng) Elias, L. (Cancer Res. and Treatment Center, Univ. New Mexico, Albuquerque, NM 87131); Wogenrich, F. J. *Proc Am Assoc Cancer Res* 20: 443; 1979 (no refs)

79-3591 Tumor Lines from Six Human Tumor Categories Established in Nude Mice (Meeting Abstract). (Eng) Fogh, J. (Sloan-Kettering Inst., Rye, NY 10580); Tiso, J.; Orfeo, T.; Sharkey, F. E. *Proc Am Assoc Cancer Res* 20: 246; 1979 (no refs)

79-3592 Morphological Markers of Oncogenic Transformation in Respiratory Tract Epithelial Cells. (Eng) Heckman, C. A. (Biology Div., Oak

Ridge Natl. Lab., Oak Ridge, TN 37830); Olson, A. C. *Cancer Res* 39(7): 2390-2399; 1979.

Changes in cell shape and microvillar density accompanying oncogenic transformation were studied in rat respiratory tract epithelial cells. Two cell lines that became tumorigenic during in vitro culture (1000 W and 165 S) were studied. In relatively late passages, but not in early passages, the lines produced keratinizing squamous cell carcinomas when tested in syngeneic hosts. Small colonies, predominantly of clonal origin, were obtained early and late after initiation of the lines in in vitro culture. Scanning electron microscopic studies showed that preoncogenic and oncogenic populations differed with respect to the shapes of cells within colonies. Differences in cell shape were analyzed further by estimating the height and the ratio of length to width for 20 cell samples from each colony. Each cell was assigned to one of nine classes of cell shape. The frequency with which spindle-shaped cells were observed in colonies increased threefold with oncogenic transformation of the 1000 W and 165 S lines. The frequency did not increase during in vitro culture of a third highly oncogenic cell line, BP 3-0. The frequency of the spindle-shaped cells in the 1000 W line was not decreased by in vivo growth and rederivation. In fact, a tumor-derived subline, (1000 WT), exhibited a fivefold greater frequency of such cells than an early passage of the 1000 W line. The number of colonies with this cell shape also increased fivefold and came to include nearly one-half of the colonies analyzed. Therefore, expression of the spindle shape became prevalent in clonal subpopulations. In early passages of the 1000 W and 165 S lines, most spindle-shaped cells were found at the edges of colonies. This suggested that the spindle shape was assumed in response to forces generated during colony expansion. In general, the 1000 W line, which was more oncogenic than the 165 S line, also showed more pronounced morphological alterations. The prevalence of ruffles was correlated with oncogenicity in the 1000 W line. However, the cell lines differed with respect to the density of microvilli at the cell surface, and this feature did not correlate with oncogenicity. The results indicate that the transformation of epithelial cells is accompanied by cytoskeletal and/or adhesive alterations. (33 refs)

79-3593 Effect of Different Nutrient Media on Diploidy and the Detection of Spontaneous Transformation of Rat Embryo Connective Tissue Cells. (Rus) Eroshkina, A. M. (Cancer Res. Center, Moscow, USSR). *Vopr Onkol* 25(4): 78; 1979.

The effect of various nutrient media on the detection of spontaneous transformation of cells in continuous cultures was studied. Cultures of rat embryo connective tissue were grown in five media. The cultured cells were systematically injected into newborn rats, and tumor development at the injection site indicated that malignant transformation had occurred. Regardless of the type of medium, malignant

transformation was detected within 8-15 mo of culturing. The addition of amniotic fluid or embryonal extract inhibited malignant transformation significantly (1.5- to 2-fold). The culture remained diploid for 6-15 mo of cultivation in media containing amniotic fluid or embryonal extract, 12-20 mo of cultivation in medium 199, 2.5-3 yr of cultivation in Eagle's medium, and for 4-4.5 yr of cultivation in double Eagle's medium. These findings indicate that chromosome aberrations cannot be regarded as a sign of malignant transformation. (no refs)

79-3594 Effect of Ascorbic Acid on the Resistance of the Extracellular Matrix to Destruction by Macrophages and Tumor Cells (Meeting Abstract). (Eng) DeClerck, Y. (Div. Hematology-Oncology, Childrens Hosp. Los Angeles, Los Angeles, CA 90027). *Proc Am Assoc Cancer Res* 20: 69; 1979 (no refs)

79-3595 Basement Membrane Collagen Synthesis in Cloned Mouse Mammary Tumor Cells (Meeting Abstract). (Eng) Roesel, R. A. (Dept. Cell and Molecular Biology, Medical Coll. Georgia, Augusta, GA 30902); Howard, E.; Cutroneo, K. R.; Gay, R. *Proc Am Assoc Cancer Res* 20: 272; 1979 (1 ref)

79-3596 Role of the Nuclear Protein Matrix (NPM) in Granulocyte Pyknosis and Segmentation (Meeting Abstract). (Eng) Eastment, C. E. (Dept. Medicine, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA 23298); Scott, R. B.; Shelton, K. R.; Haar, J. *Proc Am Assoc Cancer Res* 20: 65; 1979 (no refs)

79-3597 Biologically Active Fragments of Fibronectin (Meeting Abstract). (Eng) Ruoslahti, E. (Div. Immunology, City of Hope Natl. Medical Center, 1500 Duarte Rd., Duarte, CA 91010); Hayman, E. G.; Engvall, E. *Proc Am Assoc Cancer Res* 20: 160; 1979 (no refs)

79-3598 Comparative Aspects of Nuclear Proteins and Nuclear Composition of Cultivated Embryonic, Neonatal and Neoplastic Rat Brain Cells of Glial Origin. (Eng) Chilina, A. R. (Dept. Environmental Practice, Univ. Tennessee, Knoxville, TN 37901); Chang, M.; Ives, D. H.; Koestner, A. *Res Commun Chem Pathol Pharmacol* 24(2): 289-305; 1979.

The chemical composition of nuclei isolated from embryonic and neonatal cultured rat brain glial cells and from

cultured neoplastic rat brain glioma (T-9) cells were studied and the results compared with those obtained from HeLa S-3 cell nuclei. The T-9 cell nuclei contained significantly more total and soluble protein than the normal rat brain cell nuclei, and nuclear RNA was much more abundant in the T-9 than in the untransformed nuclei. The embryonic brain cell nuclei contained substantially more nuclear RNA than the neonatal glial cell nuclei. Equal quantities of histones were found in the normal and transformed nuclei, but the T-9 and HeLa nuclei contained more acid-soluble nonhistone proteins than the normal nuclei. The T-9 and nontransformed nuclei contained similar quantities of histone H4, H3, H2A, and H2B, but the transformed cells contained two dominant subspecies of lysine-rich H1 histone whereas only one histone H1 subcomponent was extracted from the normal nuclei. (44 refs)

79-3599 Lipid Composition of Human Neural Tumors. (Eng) Yates, A. J. (Dept. Pathology, Ohio State Univ., Columbus, OH 43210); Thompson, D. K.; Boesel, C. P.; Albrightson, C.; Hart, R. W. *J Lipid Res* 20(4): 428-436; 1979.

Ganglioside, cholesterol, and phospholipid levels were quantitated in the tissues of 11 human neural tumors and in the cells of two human gliomas cultured in vitro. All tumor tissues contained higher water concentrations but lower total lipid concentrations than either human grey or white matter. In general, they contained less cholesterol, sphingomyelin, and serine glycerophospholipid but more choline glycerophospholipid than white matter. Concentrations of total ganglioside sialic acid were intermediate between grey and white matter. Compared with normal brain, all tumors had greater proportions of the structurally less complex gangliosides and smaller proportions of the more complex gangliosides. This was most marked in the rapidly growing tumors; the better differentiated astrocytomas contained the greatest proportions of complex gangliosides. The cultured tumor cells contained amounts of total lipid and total phospholipid similar to those of their parent tissues. However, the cultures had less cholesterol, sphingomyelin, and total ganglioside than their parent tissues. There were significant amounts of choline and ethanolamine plasmalogens in both cultures and parent tissues. The ganglioside patterns of both cultures were complex, but they contained a greater proportion of structurally simpler gangliosides than their parent tissues. (45 refs)

79-3600 Characterization of Post-transcriptional Modification Sites of 18S rRNA of Novikoff Hepatoma (Meeting Abstract). (Eng) Choi, Y. C. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030); Malinowski, J.; Busch, H. *Proc Am Assoc Cancer Res* 20: 276; 1979 (1 ref)

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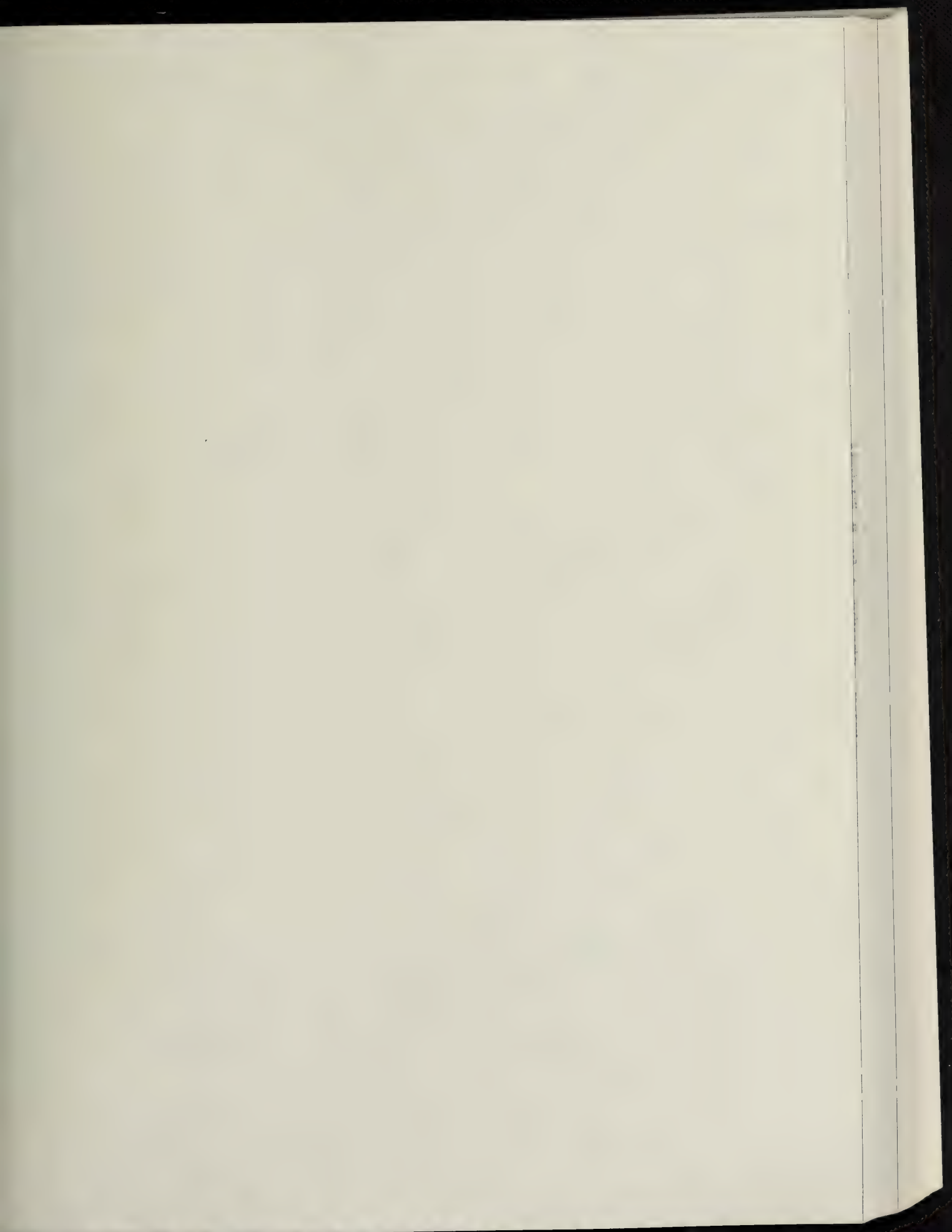
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ABBREVIATIONS

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LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED₅₀	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO₂	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD₅₀	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD₅₀	median lethal dose		
M	molar		
μM	micromolar		

REVIEW

- 79-3601 Genetic Control of Carcinogen Metabolism Leading to Individual Differences in Cancer Risk.** (Eng) Nebert, D. W. (Developmental Pharmacology Branch, Natl. Inst. Child Health and Human Development, NIH, Bethesda, MD 20014). *Biochimie* 60(9): 1019-1029; 1978.

The characteristics of the P-450-mediated monooxygenases and their coordinated enzymes are reviewed, and genetic differences in this model system in mice are examined. The *Ah* locus controls the induction of at least 20 monooxygenase activities and associated cytochromes P₁-450 and P-448 by polycyclic aromatic compounds. One product of the regulatory *Ah* gene is a cytosolic receptor protein, which has a high affinity for polycyclic aromatic inducers and is defective in nonresponsive mice. Regulation of responsiveness probably involves several alleles at more than one locus, but differences between C57BL/6 (responsive, *Ahb*), and DBA/2 (nonresponsive, *Ahd*) mice can be explained by the difference at the *Ah* locus. Heterozygotes (*Ahb/Ahd*) are responsive, but other genetic crosses can result in the expression of additive inheritance or a situation in which a lack of responsiveness is dominant. Compared with *Ahd/Ahd* mice, *Ahb/Ahb* and *Ahb/Ahd* mice have a high inflammatory response to topical application of 7,12-dimethylbenz(a)anthracene, a high susceptibility to 3-methylcholanthrene (3-MC)- and benzo(a)pyrene (BP)-induced sc sarcomas and 3-MC-induced lung tumors, and an increased resistance to 3-MC- or BP-induced leukemia. The *Ahb* allele is associated in vitro with a high mutation rate in *Salmonella typhimurium* by metabolic activation of several chemical carcinogens. Evidence exists that heritable aryl hydrocarbon hydroxylase inducibility occurs in humans, but experimental difficulties have precluded the possibility of determining whether the induction is controlled by a single gene. (35 refs)

- 79-3602 Detoxification or Toxication? Modification of the Toxicity of Foreign Compounds by Conjugation in the Liver.** (Eng) Mulder, G. J. (Dept. Pharmacology, State Univ. Groningen, Groningen, Netherlands). *Trends Biochem Sci* 4(4): 86-90; 1979.

Studies of the modification of xenobiotic toxicity by conjugation in the liver are reviewed. Generally, conjugation terminates the biological activity of endogenous compounds, such as steroid hormones, but there are exceptions. Studies with estrone sulfate suggest that conjugation of certain hormones prevents their elimination and may result in transport forms of these hormones that are specifically labeled for uptake in target cells. Benzo(a)pyrene is converted by microsomal monooxygenases into chemically reactive products that are responsible for

liver cancer. Sulfation of N-hydroxy-2-acetylaminofluorene (N-hydroxy-2-AAF) leads to an extremely reactive species that has been implicated in the carcinogenic action of this compound. N-Hydroxy-2-aminofluorene, N-acetoxy-2-aminofluorene, and, possibly, N-acetoxy-2-acetylaminofluorene are formed in vivo through enzymatic transacetylation in the liver. These compounds may also be involved in the carcinogenicity of N-hydroxy-2-AAF. Evidence for the generation of a reactive intermediate by drug-metabolizing enzymes rests on two indirect assays: covalent binding of a compound to macromolecules and the *Salmonella typhimurium* mutagenicity test. 1,2-dichloroethane is chemically activated to a more potent mutagen by glutathione conjugation. These findings may lead to a reevaluation of conjugation in the toxification of xenobiotics and in the intermediary metabolism of endogenous compounds. (21 refs)

- 79-3603 Tobacco and Health: A Societal Challenge.** (Eng) Wynder, E. L. (Naylor Dana Inst. Disease Prevention, Valhalla, NY 10595); Hoffmann, D. *N Engl J Med* 300(16): 894-903; 1979.

Three aspects of the effort to prevent or control health problems caused by smoking are reviewed: (1) youth antismoking programs; (2) smoking-cessation programs; and (3) development of a less-harmful cigarette. Various known and suspected tumorigenic agents have been identified in tobacco smoke. Numerous cigarettes with <10 mg tar and lower levels of nicotine are on the market, but their acceptance depends on the increased use of flavoring agents derived from tobacco. Assuming that all available safety techniques are used and that none of the flavor additives induce additional toxic effects, some max levels for certain smoke constituents in a less-harmful cigarette are proposed: tar, 8 mg; nicotine, 0.6 mg; carbon monoxide, 8 mg; hydrogen cyanide, 100 µg; and benzo(a)pyrene, 8 nanograms. (91 refs)

- 79-3604 Environmental Cancer: On the Causes of the Main Human Cancers.** (Eng) Weisburger, J. H. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595). *Tex Rep Biol Med* 37: 1-20; 1978.

Causes of the main human cancers are reviewed briefly. Occupationally incurred cancer arises in individuals exposed at work to varied carcinogenic chemicals, and it develops cancer at a site that is determined by the nature of the carcinogen. In the context of the total incidence of

REVIEW

cancer in the US, occupational cancer assumes a minor quantitative role. Nonetheless, provided proper plant design and hygienic measures are developed, these cancers are totally preventable. Most of the remaining human cancers in the US such as cancer of the respiratory and digestive tracts (including the stomach and large bowel) and of the endocrine-sensitive organs (including the breast, prostate, ovary, and endometrium), are related to lifestyle; namely, cigarette smoking and diet. The mechanism whereby lifestyle translates to certain of these diseases is presented. (66 refs)

- 79-3605 Some Monomers, Plastics and Synthetic Elastomers, and Acrolein.** (Eng) IARC Working Group (Unit Chemical Carcinogenesis, IARC, Lyon, France). *IARC Monogr Eval Carcinog Risk Chem* 19: 1-513; 1979.

Monographs published by the International Agency for Research on Cancer (IARC) summarized evidence on the carcinogenic risk of individual chemicals to humans and other relevant information. The analyses are intended to assist national and international authorities in formulating decisions concerning preventive measures. The chemicals selected for evaluation are those to which humans are exposed and for which there is some experimental evidence of carcinogenicity. Using data collected from appropriate tests in animals, it is possible to extrapolate to estimate possible human risk. The individual monographs contain chemical and physical data on each compound; its production, use, occurrence, and means of analysis; biological data relevant to the evaluation of carcinogenic risk in humans; and the critical analyses of the data by the IARC Working Groups. The data analyzed include those from carcinogenicity studies in animals, lethality, toxicity, and metabolism studies; studies and observations of embryotoxicity and teratogenicity; and indirect mutagenicity and other short-term tests. (53 refs)

- 79-3606 Environmental Cancer: Animal Studies and Assay Procedures.** (Eng) Griffin, A. C. (Dept. Biochemistry, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030). *Tex Rep Biol Med* 37: 45-49; 1978.

In developing an animal testing program for determining the carcinogenicity of environmental chemicals, species, strain, age, sex, number, maintenance, and cost of the test animals; dosage and mode and duration of administration of the test chemical; and length of the study must be considered. Evaluation of the findings should include gross evaluations of animals, histopathological studies of tissues and organs, data evaluation and statistical aspects, extrapolation of data to humans, and final evaluations and recommendations involving governmental agencies, labor, and industry. (no refs)

- 79-3607 How Do We Know What Causes Cancer?** (Eng) Shaw, C. R. (Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Leal, N. *Tex Rep Biol Med* 37: 21-25; 1978.

Tests to determine whether a chemical will produce genotoxic or mutagenic effects involve humans, other animals, or bacteria. The most widely used bacterial test is the Ames *Salmonella typhimurium* mutagenicity assay. One problem with this test arises from the fact that most chemicals are not actively toxic in the forms in which they are taken into the body. Other problems involve genetic variations in the ability to metabolize chemicals, variations in the levels of enzymes involved in DNA repair, and individual differences in the degree of immunosurveillance. (3 refs)

- 79-3608 The "Natural" Food Myth.** (Eng) Rhodes, M. E. (Food Lab., Div. Chemistry, Dept. Agriculture and Consumer Services, Tallahassee, FL). *Sciences* 19(5): 11-30; 1979.

Consuming only foods labeled natural does not necessarily eliminate poisons from food, as there are many natural poisons in food. However, the concentrations of these natural poisons are so low that a huge amount would have to be consumed over a long period of time for the toxic effect to become apparent. Food additives such as nitrates and nitrites, as well as sodium benzoate, occur naturally in leafy vegetables and cranberries, respectively. Some natural components of food, such as aflatoxin, which produces liver cancer in laboratory animals, may actually be harmful. (no refs)

- 79-3609 Iatrogenic Complications of Dermatologic Therapy.** *Primum non Nocere.* (Eng) McMeekin, T. O. (300 White Spruce Blvd., Rochester, NY 14623); Moschella, S. L. *Med Clin North Am* 63(2): 441-452; 1979.

Adverse reactions to systemic and topical drugs and to physical modalities in the treatment of skin disease are reviewed. Radiotherapy and systemic azathioprine therapy, the latter of which is used to treat pemphigus vulgaris, may be complicated by the development of malignant disease. The development of keratoses in chronic radiation dermatitis may herald malignant degeneration. There is also an association between external radiation and delayed neoplastic changes in the thyroid. Hazards are also associated with dermatologic use of the cytostatic drugs podophyllin and γ -benzene hexachloride. (98 refs)

- 79-3610 Drugs and the Liver.** (Eng) Rosenoer, V. M. (Dept. Gastroenterology, Lahey Clinic, 605

Commonwealth Ave., Boston, MA 02215); Tornay, A. S. *Med Clin North Am* 63(2): 405-412; 1979.

A review of acute and chronic drug hepatotoxicity includes discussions of metabolite-related drug toxicity, drug hypersensitivity, chronic liver disease, and the association between hepatic tumors and oral contraceptives (OC's). Current evidence implicates OC use in the pathogenesis of benign and malignant liver tumors, and this is supported by the regression of benign tumors upon withdrawal of therapy and recurrences after primary resection in patients who continued to use OC's. (19 refs)

79-3611 Potential Halogenated Industrial Carcinogenic and Mutagenic Chemicals. III. Alkane Halides, Alkanols and Ethers. (Eng) Fishbein, L. (Natl. Center Toxicological Res., Jefferson, AR 72079). *Sci Total Environ* 11(3): 223-257; 1979.

Data on a variety of potentially carcinogenic alkane halides, alkanols, and ethers are reviewed with regard to synthesis, use, stability, distribution, reactivity, populations at risk, exposure levels, metabolism, mutagenicity, and carcinogenicity. The compounds include ethylene dichloride and dibromide, propylene dichloride, dibromochloropropane, 2-chloroethanol, 1-chloro-2-propanol, 2,3-dibromo-1-propanol, bis(chloromethyl)-ether, chloromethylmethylether, bis(2-chloroisopropyl)-ether and bis(2-chloroethyl)ether. They are used as solvents, fumigants, propellants, and intermediates in the production of other chemicals, textiles, plastics, and ion-exchange resins. (199 refs)

79-3612 Potential Halogenated Industrial Carcinogenic and Mutagenic Chemicals. IV. Halogenated Aryl Derivatives. (Eng) Fishbein, L. (Natl. Center Toxicological Res., Jefferson, AR 72079). *Sci Total Environ* 11(3): 259-278; 1979.

Data on a variety of potentially carcinogenic halogenated aryl derivatives are reviewed with respect to synthesis, use, stability, distribution, reactivity, populations at risk, exposure levels, metabolism, mutagenicity, and carcinogenicity. The compounds, which include o- and p-dichlorobenzene, 1,2,4-trichloro- and hexachlorobenzene, benzyl chloride and the bromobenzenes, are used industrially as solvents for pesticides, heat-transfer agents, pesticide intermediates, additives for rubber products, intermediates in organic synthesis, and as insect repellants and deodorants. (116 refs)

79-3613 The Effects of Toxic Agents on Reproduction. (Eng) Stellman, J. M. (Div. Occupational

Health and Toxicology, American Health Foundation, New York, NY). *Occup Health Saf* 48(3): 36-43; 1979.

Aspects of the toxicology of human reproduction are reviewed, with particular emphasis on the effects of toxic agents encountered in the workplace. Potential modes of reproductive dysfunction include organ dysfunction, genetic defects, gestational effects, and postpartum effects. Substances that may affect reproduction include diethylstilbestrol (DES), estrogen, thalidomide, anesthetic gases, vinyl chloride, chloroprene, dibromochloropropane, DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane], copper in intrauterine devices), lead, and nonionizing radiation. (no refs)

79-3614 Mechanisms of Damaging Effect of Beryllium. (Rus) Vasil'eva, E. V. (Inst. Vocational Hygiene and Occupational Diseases, Moscow, USSR); Ivanova, L.A.; Sokolov, V. V. *Gig Sanit* (2): 15-19; 1979.

Current data on beryllium-induced alterations in immunologic homeostasis are reviewed. The main biological feature of Be is its permeation into the cell nucleus, which is followed by the development of humoral and cellular antinuclear reactions. The fact that patients with berylliosis and healthy persons who had been exposed occupationally to Be show sensitization to Be and formation of antibodies against DNA indicate that Be interacts with nuclear DNA. Be exposure increases serum IgG levels and the titer of antitissue antibodies. Small amounts of Be induce epithelial granulomas in lungs and other organs in sensitized patients. (65 refs)

79-3615 Comparative Toxicology of N-Nitroso Compounds and Their Carcinogenic Potential to Man. (Eng) Olajos, E. J. (Inst. Comparative and Human Toxicology, Albany Medical Coll., Albany, NY 12208); Coulston, F. *Ecotoxicol Environ Saf* 2(3/4): 317-367; 1978.

The in vitro and in vivo formation, chemistry, and bioconversion of N-nitroso compounds are reviewed, along with the interaction of N-nitroso compounds with genetic material. Attention is drawn to recent developments regarding the relationship between mammalian metabolism of N-nitroso compounds, mutagenesis, and relative susceptibility to carcinogenesis. The specific organotropic effect of this class of carcinogens is also covered, as are recent developments pertaining to nitrosamine-induced transplacental carcinogenesis. The article concludes with the presumptive role of N-nitroso compounds, preformed as well as derived from precursor amino compounds, in the etiology of human cancer. (no refs)

79-3616 Experimental Stomach Carcinogenesis. (Eng) Sugimura, T. (Natl. Cancer Res. Inst., Tokyo,

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Japan); Kawachi, T. In: *Gastrointestinal Tract Cancer*. Lipkin, M.; Good, R. A., eds. (New York, London: Plenum Medical Book Co.): Sloan Kettering Inst. Cancer Series 602 pp.; 327-341; 1978.

Studies of the production of experimental stomach carcinoma (SC) in dogs and rats by N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) are reviewed. The method is easy, the incidence of SC is fairly high, and the results are reproducible. In one study, administration of MNNG (83 µg/ml in drinking water) to Wistar male rats resulted in shallow erosion in the pyloric region 3 days later. Three weeks later, regenerative hyperplastic changes were found. Adenomatous hyperplasia was observed at 6 mo, adenocarcinoma (AC) showing cellular and structural atypism plus active infiltration through the musculus propria at 12 mo. The SC's that developed were mainly differentiated-type AC's; diffuse- and signet ring cell-type AC were also produced. The pylorus was the preferred site. The different types of SC tended to metastasize to selected sites, similar to the situation in humans. After MNNG treatment, the strict localization of DNA synthesis at the bottom of the pyloric gland and at the neck of the fundic gland was found to be disturbed. In rats, radiographic exams can be carried out repeatedly without sacrificing the animal. Dogs were administered MNNG in drinking water or in dried food pellets soaked with aqueous MNNG soln. As in rats, high MNNG doses and longer exposure periods produced tumors in loci other than the stomach. Dog SC's mainly developed in the antrum and pylorus and in the cardiac portion of the dorsal wall; they were well- and poorly differentiated AC's. Successive changes in the stomach of beagle dogs given MNNG in drinking water ad libitum included hyperemia, erosion, and ulcers at the angulus in the antrum, followed by mucosal atrophy and scar. Subsequently, AC's developed at the ulcer scar. The large size of the dog stomach permits precise radiographic follow-up endoscopic exams. (79 refs)

79-3617 Tumors of the Cardiac Stomach in Rodents as an Experimental Model for Testing the Carcinogenicity of Chemicals Administered Orally. (Rus) Ianysheva, N. Ia. (A. N. Marzaev Res. Inst. General and Communal Hygiene, Kiev, USSR); Chernichenko, I. A.; Balenko, N. V. *Gig Sanit* (1): 77-80; 1979.

Controversial data on the possibility of using tumors of the cardiac stomach in rodents as a model for evaluating the carcinogenicity of various chemicals given po are reviewed. Recent studies show that direct administration of 7,12-dimethylbenz(a)anthracene into the glandular region of the stomach can induce tumors, which might be indicative of the sufficient sensitivity of this part of the stomach to carcinogens. When benzo(a)pyrene was administered po to rats, it accumulated in the cardiac stomach and in the glandular region of the stomach and induced tumors of the glandular region in 34.5% of the animals. (15 refs)

79-3618 Dietary Cholesterol is Co-carcinogenic for Human Colon Cancer. (Eng) Cruse, P. (Surgical Unit, Univ. Coll. Hosp. Medical Sch., London WC1E 6JJ, England); Lewin, M.; Clark, C. G. *Lancet* 1(8119): 752-755; 1979.

Evidence suggesting that dietary cholesterol (CS) may be a cocarcinogen in human colon cancer is summarized, and possible mechanisms of CS cocarcinogenesis are presented. There is a significant correlation between high consumption of CS-containing foods and the worldwide distribution of colon cancer. Patients with colon cancer have significantly higher levels of fecal CS and CS metabolites than patients with other gastrointestinal disease and healthy controls. Dietary status and current fecal metabolic findings correlate better with the presence of colon cancer than do past diet or past fecal biochemistry. This suggests a continuing, concurrent effect rather than a precursor effect and favors a cocarcinogenic role for dietary or fecal CS rather than an initiating one. In every experimental colon cancer study of a CS-containing diet, the greatest percentage of tumor-bearing animals, the highest mean number of colon tumors per animal, and the greatest incidence of metastases occurred in animals on diets containing the most CS. Four mechanisms of CS cocarcinogenesis are considered: (1) it may exert a preparative action on the colonic epithelium; (2) it may be a permissive agent; (3) it may be a tumor promoter; or (4) it may exert a systemic conditional influence. Evidence from human and animal studies supports the third alternative, tumor promotion, as the most likely mechanism for CS cocarcinogenesis. (55 refs)

79-3619 Review of the Genetic Effects of Caffeine. (Eng) Legator, M. S. (Div. Genetic Toxicology, Dept. Prevention Medicine and Community Health, Galveston, TX 77550); Zimmering, S. *J Environ Sci Health* 13(2): 135-188; 1979.

Caffeine (SF) induces chromosome breakage in some plant systems, and it has a synergistic effect on breakage induced by chemical mutagens. Consistent low-level effects of CF on chromosome loss occur in male and female *Drosophila* germ cells. Results from dominant lethal tests in rodents suggest no demonstrable effect of CF in F₁ hybrids; a low-level effect at certain doses in some inbred strains and no consistent effect in combination with known mutagens. In vivo cytogenetic studies provide evidence of a synergistic effect in CF-oxygen and CF-cyclophosphamide combinations. In humans, one experiment testing a single dose of CF in vivo provided no evidence of a break induction effect. (150 refs)

79-3620 Effects of Methylxanthines on the Fetus. (Eng) Soyka, L. F. (Dept. Pharmacology, Univ. Vermont Coll. Medicine, Burlington, VT 05405). *Symp Pharmacol* 6(1): 37-51; 1979.

The pharmacokinetics and mechanism of action of methylxanthines, particularly caffeine (CF), theophylline, and theobromine, are reviewed along with their effects on fetal lung maturation. CF is a weak mutagen in some non-mammalian systems but not in mammalian systems. CF can modify the effects of other agents on mutation, chromosomal change, and DNA repair. There has been no convincing demonstration of CF carcinogenicity in animals, and studies in which animals received <50 mg/kg CF demonstrated no increased teratogenicity. Doses of >20 mg/kg/day decrease fetal wt and increase fetal absorption in rats. CF also inhibits uterine activity. (79 refs)

- 79-3621 **Estrogen Substitution Therapy and Endometrial Carcinoma.** (Dut) Kruijver, G. P. (Sittard, Netherlands). *Ned Tijdschr Geneesk* 123(10): 383-387; 1979.

A critical review is presented of recent statistical investigations showing a correlation between estrogen substitution therapy and endometrial carcinoma. A theory implicating estrone as a specific carcinogen is rejected. Estrogen medication per se does not appear to be the cause of endometrial carcinoma, but it can speed up the manifestation of carcinoma in predisposed patients. Pending further research into the correlation between estrogens and endometrial carcinoma, this therapy should be applied conservatively. (31 refs)

- 79-3622 **Hormonal Factors in Lipogenesis in Mammary Gland.** (Eng) Mayer, R. J. (Dept. Biochemistry, Queen's Medical Centre, Univ. Nottingham, Nottingham, England). *Vitam Horm* 36: 101, 154-163; 1978.

Studies of lipogenic enzyme patterns in mammary tumors are reviewed. Mouse mammary adenocarcinomas synthesize fatty acids from acetate and glucose at much lower rates than do lactating mouse mammary gland, and the proportion of medium-chain fatty acids produced by preneoplastic or neoplastic mammary tissue is much lower than that in normal lactating mammary gland. Pharmacological doses of estrogen increased glucose-6-phosphate dehydrogenase (G6PDH) activity in transplantable rat mammary adenocarcinomas, which indicates that the tumor can undergo some lactogenic differentiation in response to pharmacologic doses of estrogen, although in more slowly proliferating cells. Increased cAMP concentrations may exert an antilipogenic effect in mammary gland and liver and, possibly, in tumors from these tissues. Injection of dibutyl-cAMP into rats bearing MTW9 mammary carcinoma resulted in the early disappearance of microsomal G6PDH activity. Since dibutyl-cAMP arrested the growth of these tumors, it was suggested that the loss of G6PDH may be an early event in the inhibition of

tumor growth in vivo. It has not been determined whether lipogenic enzymes are transformation- or progression-linked discriminants in mammary tumors. However, some mammary tumors may retain the potential for lactogenic responses to hormones, at least when administered in pharmacological doses. (73 refs)

- 79-3623 **Reference to Bile and Other Surfactants as Promoters of Postoperative Gastric Cancer.** (Eng) Domellof, L. (Univ. Hosp., S-901 85 Umea, Sweden). *Med Hypotheses* 5(4): 463-476; 1979.

For reasons that are not known, the incidence of stomach cancer is declining in most countries. In contrast, cancer of the gastric remnant after partial gastrectomy for peptic ulcer disease seems to be increasing. "Gastric stump cancer" has been claimed to be a separate disease entity and has recently been labeled an iatrogenic cancer. Postoperative mucosal degeneration, exposure to surfactants such as bile, deconjugation of bile salts by microflora, increased pH with the possible production of mutagenic compounds, and diet may contribute to the malignant transformation of target cells. The cancer risk seems to be higher in men. Differences in tobacco abuse and/or a sex-related metabolism of carcinogens (repair mechanisms) may explain this observation. Alkaline bile reflux is one important promoting factor, as judged by the occurrence of polyps and cancers in areas most exposed to the reflux. The decline in cancer incidence in the nonoperated stomach has mainly been restricted to intestinal-type cancer. Thus, it is important to note that stump cancer often is of the diffuse type. Different target cells and/or modes of carcinogen exposure may be of etiological importance in these histologically and clinically separate neoplastic diseases. (75 refs)

- 79-3624 **Do Biopsy and Operation Lead to Distant Metastases?** (Ger) Hermanek, P. (Urologische Universitätsklinik, Universitätskrankenhaus Eppendorf, Martinistrasse 52, D-2000 Hamburg 20, W. Germany); Klosterhalfen, H. *Urologe [A]* 18(2): 49-50; 1979.

A hypothesis that states that 30%-90% of all tumor metastases are caused by biopsy and surgery is criticized. As no details are available on the method used to arrive at these figures, the hypothesis cannot be proved. The applications of biopsy shall consequently not be restricted because of this hypothesis. (13 refs)

- 79-3625 **The Nuclear Worker and Ionizing Radiation.** (Eng) Bertell, R. (151 East Street, Buffalo, NY 14207). *Am Ind Hyg Assoc J* (40): 395-401; 1979.

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The ability of workers to tolerate exposure to ionizing radiation from nuclear fission by-products and related industries is reviewed. With regard to the immediate effects of worker exposure, there may be a problem of physical adjustment to chronic low doses of radiation that may involve temporary motor and judgment impairment. With respect to chronic damage to health, theory and observation both give the same caution: namely, that radiation-related cancer deaths have been underestimated by the most widely used predictive models. Data from the Tri-State Leukemia Survey show that exposure to permissible levels of medical x-radiation (1 to 620 millirads) has been associated with an increased frequency of nonlymphatic leukemia. The effects of such low doses of radiation appear to be similar to those of aging. All debilitating long-term chronic effects known to be related to radiation exposure are unmonitored in the nuclear industry. Although it has been assumed that control of radiation-induced cancer is the key to control of all other health effects, different health effects of radiation exposure become dominant at different dose levels and rates of exposure, and death may be caused by complicated interaction of factors. Also, different segments of the human population may be more sensitive to radiation, eg, fetuses and infants. The records of radiation workers should document exposures, medical history, reproductive history and data on offspring. These records should be kept in perpetuity, and data should be pooled across industries involving exposures to the same carcinogens. (21 refs)

- 79-3626 Systematic Review of Thorotrast Data and Facts: Animal Experiments. (Eng) Wegener, K. (Pathological Inst. Municipal Hosp., Bremserstrasse 79, D-6700 Ludwigshafen am Rhine, W. Germany). *Virchows Arch [Pathol Anat]* 381(3): 245-268; 1979.

A comprehensive survey of animal experiments concerning thorium dioxide colloid (Thorotrast: TT) distribution in the reticuloendothelial system (RES) that have been carried out since its introduction as a contrast medium is presented. Stages in the distribution of TT throughout the body following iv injection include splitting off of the carrier component, dextrin; pinocytotic and phagocytotic absorption of protein-coated thorium dioxide particles by Kupffer cells; appearance of TT in cytoplasmic vacuoles; aggregation of TT particles; and storage of aggregates in secondary lysosomes. A short time following injection, the hepatocytes also begin absorbing thorium dioxide particles, although to a lesser degree than do the Kupffer cells. Max accumulation in the liver and spleen is reached quickly; TT then migrates, mainly to the lung. Urinary and fecal elimination occurs only after complete saturation of the spleen and liver. Fibroblastic sarcomas and polymorphous sarcomas have developed in animals following ip injection of TT. The induction of hemangioendotheliosarcomas by TT is apparently less frequent in animals than in humans. Other lesions reported to occur in animals following TT injection include focal liver cell hyperplasia, liver cell

adenoma and carcinoma, bile duct hyperplasia, cholangiocarcinoma, malignant splenic hemangioendothelioma, Kupffer cell hyperplasia, malignant lymphoma, malignant histiocytoma, reticulum cell sarcoma, osteoid sarcoma, pulmonary adenoma, alveolar cell carcinoma, and rhabdomyosarcoma. One experiment demonstrated that the incidence of TT-induced tumors is dose-related. Another investigation showed that the product of concentration and latency period is not constant. None of the experiments have satisfactorily demonstrated the relative importance of the presence of a foreign body or the effects of radiation in the development of TT-related tumors. (70 refs)

- 79-3627 The Growth of Virus Research 1928-1978. (Eng) Andrewes, C. (London, England). *Postgrad Med J* 55(640): 73-77; 1979.

A review of the growth of virus research includes discussions of tissue culture, virus properties, classification, immunity, virus interference, influenza, viruses and tumors, and molecular biology. Oncogenic viruses may be integrated with the genetic apparatus of the host, where they may remain latent until some stimulus activates them. (33 refs)

- 79-3628 Why Cell Biologists Should be Aware of Genetically Transmitted Viruses. (Eng) Weiss, R. A. (Imperial Cancer Res. Fund Lab., P.O. Box 123, Lincoln's Inn Fields, London WC2A 3PX, England). *Natl Cancer Inst Monogr* (48): 183-189; 1978.

The implications of the presence of endogenous tumor virus genes and their products in uninfected cell cultures are reviewed. Endogenous virus expression in these cultures will vary according to the genetic constitution of the cells, the culture conditions, and the presence or absence of extrinsic activating agents. Once the virus has been activated, host genes affecting provirus expression affect the susceptibility of the cells to secondary infection. In the case of the xenotropic endogenous viruses, cross-species transfer to humans might readily occur in laboratory experiments, even when a natural zoonosis is unlikely to occur. Among the situations that would permit amplification of xenotropic viruses with increased likelihood of zoonotic infection are somatic cell hybridization between mouse and human cells and the transplantation of human cells into nude or immunodeficient mice. It is improbable, however, that the xenotropic viruses thus activated would present a great hazard to humans. The activity of endogenous viruses might effect the significance of experimental results, as, for example, in studies of cell-surface antigens, RNA or membrane synthesis, cell morphology, or agglutinability by lectins. Endogenous viruses might also pose problems to virologists as a result of phenotypic mixing, genetic com-

plementation, genetic recombination, and unscheduled reverse transcription. (50 refs)

- 79-3629 Genome Structure and Replication of Oncornaviruses.** (Eng) Kisselev, L. L. (Inst. Molecular Biology, Acad Sciences USSR, Moscow V-334, USSR); Frolova, L. Yu. *Folia Biol (Praha)* 25(1): 1-35; 1979.

The structure of RNA oncornavirus (OV) genomes and their replication are reviewed. The OV genome contains 60S-70S RNA and 4S-10S RNA. The 60S-70S RNA is composed of two identical 30S-40S subunits held together by hydrogen bonds. Electron microscopy has established that the 30S-40S RNA molecules of murine, feline, and simian OV's in the 60S-70S complex form a structure that has the following features: (1) proximity of the 5'-termini of both molecules ('dimer linkage structure'); and (2) a large loop in each chain. The 3' terminus of the 30S-40S RNA has a long polyadenylic sequence, and the 5'-end has a cap. The following gene order has been deduced from a number of studies: (5') pol-env-arc (3'). The first stage of virion replication, which has been proved experimentally, is as follows: initiation of synthesis at the 3'-end of the transfer RNA (tRNA) primer, synthesis of the short DNA transcript complementary to the 5'-end of viral RNA, its interaction with the 3' end of the same or other 30S-40S RNA molecule, and subsequent transcription of the RNA template catalyzed by reverse transcriptase. It is assumed that the covalently closed, double-stranded, supercoiled DNA is the final form of this molecule before it is integrated into the cellular genome. (172 refs)

- 79-3630 Transformation by Viruses: Simian Virus 40 as a Model System.** (Eng) Noonan, C. A. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Butel, J. S. *Natl Cancer Inst Monogr* (48): 227-237; 1978.

This review is devoted to a brief description of the basic properties of the simian virus 40 (SV40) system and to a summary of current understanding of the process by which SV40 mediates cellular transformation. SV40, a DNA-containing tumor virus in the papovavirus group, represents an ideal model system for analyzing viral-induced tumorigenesis because of the small size of its genome and its broad range of oncogenic potential. Viral genes persist and are expressed in SV40-transformed cells. Temperature-sensitive (ts) mutants of the virus have proved to be valuable for the identification and analysis of viral gene expression in transformed cells. These mutants have helped to determine that a specific gene product (A protein) is required to initiate cellular transformation. The role of virus genes in the maintenance of the transformed state was determined by transforming mouse, hamster, and human

cells by ts virus containing A-gene mutations. These cells were then examined under permissive and nonpermissive conditions for the presence of a variety of intracellular and surface alterations commonly associated with neoplastic transformation. These experiments showed that an SV40-specific function is also necessary for the maintenance of at least some of the phenotypic properties of the transformed state. Indirect evidence, derived from a comparison of the biological and biomedical properties of the SV40-induced tumor (T) antigen and the gene A protein, supports the idea that T antigen is a product of the A gene. One model devised to explain the mechanism by which the gene A protein might function as an effector of transformation is presented. (50 refs)

- 79-3631 Replication of Herpesviruses and Latency.** (Eng) Rosenthal, L. J. (Dept. Microbiology, Georgetown Univ., Sch. Medicine, Washington, DC 20007). *Can J Microbiol* 25(3): 239-244; 1979.

Characteristics of herpesviruses (HV's), especially herpes simplex virus types 1 and 2 (HSV-1 and -2) and human cytomegalovirus (CMV), are reviewed. HV's are large enveloped DNA viruses 150-200 nanometers in diameter that bud through the nuclear membrane. They are composed of a centrally located core surrounded by a capsid, tegument, and envelope containing spikes. HV DNA's are linear, double-stranded molecules with a mol wt of $97-99 \times 10^6$ for HSV-1 and -2 and 150.5×10^6 for CMV. HSV-1 and -2 can infect cells from almost any species without regard to cell type, and the replicative cycle is complete within 24 hr. Human CMV replication is characterized by a long eclipse phase of 55 hr and a dependence on human fibroblasts for replication. After fusion, naked CMV capsids migrate across the cytoplasm to the nucleus and become coated with a fibrillar material. This coating, which was not observed with HSV, may account for the long eclipse time. HSV infection inhibits host DNA, RNA, and protein synthesis. CMV infection stimulates cellular DNA, RNA, and protein synthesis and, in permissive cells, results in the production of virus-specific intranuclear antigen as early as 3 hr after infection. HSV-1 and -2 and CMV transform cells in vitro. The biochemical nature of latency and the physical state of the latent virus are unknown. It appears that some form of nonreplicating virus is conserved in latent infection. (43 refs)

- 79-3632 Immune Control of Herpesvirus Latency.** (Eng) Babiuk, L. A. (Dept. Veterinary Microbiology, Western Coll. Veterinary Medicine, Univ. Saskatchewan, Saskatoon, Saskatchewan, Canada S7N 0W0); Rouse, B. T. *Can J Microbiol* 25(3): 267-274; 1979.

The mechanism of herpesvirus latency, the role of immunological control in maintaining virus persistence, and

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the influence of the immune response on the expression of disease are reviewed. Two hypotheses have been proposed to explain the persistence of herpes simplex virus type 1 (HSV-1) in the ganglia: (1) the dynamic state theory, which suggests that virus replication and shedding from infected ganglia occur continuously but at a slow rate, leading to microfoci of infection that can be controlled by an uncompromised immune system; and (2) the static-state theory, which suggests that the virus is present in the ganglia in an unknown nonreplicating state, is activated by some stimulus, and will replicate if uncontrolled by the immune system. The genome is maintained in a latent state by humoral antibody directed against viral-coded membrane antigens. Fluctuations in specific antibody or weakening of Fc-IgG binding results in reactivation of virus in the neurons with subsequent centrifugal migration to the epithelium, where the virus can replicate. As soon as replication occurs, there is rapid infiltration of polymorphonuclear cells, lymphocytes, and macrophages as well as secretion of lymphokines, which can limit replication by killing the infected cells or by limiting virus replication. A defect in any one of these components permits the virus to continue to replicate and results in a lesion. (56 refs)

- 79-3633 Persistence, Reactivation, and Cell Transformation by Human Herpesviruses: Herpes Simplex 1,2 (HSV-1, HSV-2), Cytomegalovirus (CMV), Varicella-Zoster (VZV), Epstein-Barr Virus (EBV). (Eng) Joncas, J. H. (Dept. Microbiology, Univ. Montreal, Montreal, Quebec, Canada). *Can J Microbiol* 25(3): 254-260; 1979.

Evidence for the persistence of human herpesviruses following primary infection is presented, and the effects of this persistence are reviewed. The persistence of herpes simplex virus types 1 and 2 (HSV-1 and -2), Epstein-Barr virus (EBV), Varicella-Zoster virus (VZV), and cytomegaloviruses (CMV) has been demonstrated in a variety of human tissues in vitro, and it is supported by the occurrence of reactivated infections by these viruses in immunosuppressed hosts. A literature review and observations made in ataxia-telangiectasia (AT) and Burkitt's lymphoma (BL) patients and in a family with multiple Burkitt's tumors and nasopharyngeal carcinoma suggest that an important factor in the development of an EBV-associated tumor could be the target cell and its genetics. Studies of two EBV-transformed cell lines of one AT patient disclosed a chromosome 14 translocation that is a marker found exclusively in lymphomatous cells. The response of the EBV antigens in AT cell lines to hydrocortisone was similar to that seen in BL cell lines. BL cell lines (6/9) and AT cell lines (3/3) responded to hydrocortisone by an increase in EBV early antigen (EA) synthesis, compared with 0/18 EBV-transformed lymphoid cell lines established from the peripheral blood of normal individuals. The prevalence of EBV EA antibodies in normal individuals did not exceed 5%-10%, whereas in AT and BL

it was as high as 50%-100%. It is concluded that target cell defectiveness may be a primary factor and immunodeficiency a secondary factor in the increased incidence of tumors in other immunodeficiency syndromes and in families with multiple tumors. (50 refs)

- 79-3634 Some Aspects of the Role of Viruses in Cancer. (Eng) Potter, C. W. (Dept. Virology, Univ. Sheffield Medical Sch., Sheffield, Yorkshire, England). *Postgrad Med J* 55(640): 150-158; 1979.

The role of viruses in human cancer is reviewed. Several papova-, adeno-, and herpesviruses are associated with naturally occurring cancers or produce tumors when inoculated into experimental animals. Tumors induced by adenoviruses or simian virus 40 contain virus-specific DNA, RNA, and tumor (T) antigen, and they induce virus-specific T antibody and transplantation immunity. These features have not been demonstrated in human cancer patients, which suggests that these viruses do not have a biological role in normal human cancers. Herpes simplex virus type 2 (HSV-2) has been implicated in the etiology of cervical carcinoma in humans. The epidemiology of this cancer, the oncogenic potential of herpesviruses in other species, the probable association of Epstein-Barr virus with Burkitt's lymphoma and the ability of HSV-2 to transform human cells in vitro and produce carcinoma of the cervix in animals suggest a possible role of this virus in cervical cancer. However, virus isolation and serologic studies in patients with cervical cancer have not significantly supported this theory. (55 refs)

- 79-3635 Warts: Immunologic Factors of Prognostic Significance. (Eng) von Krogh, G. (Dept. Dermatology, Karolinska Institutet, Sodersjukhuset, S0100 64, Stockholm 38, Sweden). *Int J Dermatol* 18(3): 195-204; 1979.

The classification and immunology of warts and wart viruses (papovaviruses) are reviewed. Available data support the hypothesis that specific cell-mediated immune responses against neoantigens on the surface of tumor cells are of dominant importance in the induction of host resistance to neoplasms. The appearance of human papilloma virus-specific antibodies is probably a secondary phenomenon in wart rejection. (54 refs)

- 79-3636 Epstein-Barr Virus-Regulation Studies on Somatic-Cell Hybrids Derived from the Fusion of Burkitt's Lymphoma and Nasopharyngeal Carcinoma Cells with Human or Mouse Partners. (Eng) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S 104 01 Stockholm 60, Sweden). *IARC Sci Publ* 20: 369-376; 1978.

Studies of Epstein-Barr virus (EBV) regulation using somatic cell hybrids are reviewed. Chromosome patterns and EBV genome persistence were studied in two types of mouse/human hybrid. In mouse/Burkitt's lymphoma (BL) hybrids, loss of chromosome 21 paralleled the loss of EBV. In mouse/nasopharyngeal carcinoma (NPC) hybrids, most of the EBV-carrying hybrids contained chromosome 20 or 21. None of the hybrids showed any spontaneous EBV production, nor were they inducible. Epstein-Barr nuclear antigen (EBNA) was fully expressed. Spontaneous EBV production was eliminated in a hybrid between the EBV-producer line P3HR-1 and an EBV-negative HeLa subline, but it was maintained in a BL/BL hybrid. There was a good correlation in various hybrids between the early antigen (EA) induction responses following iododeoxyuridine treatment or P3HR-1 superinfection, compared with those in parental cells, in spite of the different levels of response to the two treatments. This suggests that the same regulatory mechanisms control the responses of the cell to both agents. Both suppressive and permissive hybrids have been identified, suggesting that the outcome is determined by the actual combinations of target cells. In all human/mouse hybrids in which an EBV-carrying B-lymphoblast line was the human partner and in human/human hybrids derived from the fusion of a B-lymphoid line with nonlymphoid partners, all differentiated (B-cell) markers of the human partner were eclipsed completely. When two B-cell lines were fused, complement receptors, EBV receptors, and Fc receptors showed a dominant expression. Major histocompatibility complex antigens (HLA) and B-cell antigens were expressed codominantly. (22 refs)

79-3637 Discussion Summary. (Eng) Tachibana, T. (Res. Inst. Tuberculosis and Cancer, Tohoku Univ., Sendai, Japan). *IARC Sci Publ* 20: 537-542; 1978.

A discussion of conference papers on the etiology of nasopharyngeal carcinoma (NPC) is summarized. Antibodies to Epstein-Barr virus (EBV) are demonstrated not only in EBV-carrying tumors and EBV-associated infections, but also in non-EBV-carrying tumors (ie, Hodgkin's disease, chronic lymphocytic leukemia, head and neck carcinomas) and in healthy donors. The viral capsid antigen-specific IgG level is highest in those with EBV-associated diseases, intermediate in those with non-EBV-carrying tumors, and lowest in healthy donors. Sera of NPC patients had higher incidences and titers of EBV-specific IgA than patients with other EBV-associated diseases. Killer T cells from NPC biopsies showed specific cytotoxicity for EBV-positive cell lines and for Burkitt's lymphoma (BL) biopsies. The association of a specific HLA profile with NPC has been shown in Chinese living in Singapore, Malaysia, Hong Kong, and the US. Major histocompatibility complex identity is necessary between responding and stimulating cells for T-cell activation. Therefore, in testing cell-mediated immunity (CMI) in NPC or BL pa-

tients in vitro, the target cells should have an HLA identical to that of the patient from whom the sensitized lymphocytes were taken. Mouse immune response (Ir) genes seem to act at the level of stimulator cells. Studies of the Ir genes indicate that cells from patients with a high-risk NPC haplotype could act as stronger stimulators in CMI tests for lymphocytes sensitized to EBV-specific antigens than cells from patients that do not have the high-risk haplotype. (2 refs)

79-3638 Epstein-Barr Virus--Discovery, Properties and Relationship to Nasopharyngeal Carcinoma. (Eng) Epstein, M. A. (Dept. Pathology, Univ. Bristol Medical Sch., University Walk, Bristol BS8 1TD, England). *IARC Sci Publ* 20: 333-345; 1978.

The discovery and properties of Epstein-Barr virus (EBV) and the relationship of this virus to nasopharyngeal carcinoma (NPC) are reviewed. EBV was detected in African Burkitt's lymphoma biopsy samples by electron microscopy and was identified as a member of the herpesvirus family by immunological studies. The virus genome consists of double-stranded DNA with a mol wt of 10^6 daltons. EBV is transmitted horizontally and infects all human populations. It is the etiological agent for infectious mononucleosis, it can transform cells in vitro, and it induces tumors in subhuman primates. The EBV genome and Epstein-Barr nuclear antigen (EBNA) have been detected in the epithelial cells of NPC's from every part of the world. All NPC patients have antibodies to EBV capsid antigen (VCA), usually at high titer. There is evidence that NPC is a monoclonal disease, and the presence of EBV DNA in every tumor cell indicates that the original cell that underwent malignant transformation must have been infected from the beginning. The high incidence of NPC in the southern Chinese and the moderately high incidence in parts of East and North Africa indicate a genetically determined predisposition to NPC, which has been confirmed by studies showing a three times greater risk of NPC associated with particular major histocompatibility complex (HL-A) profiles in southern Chinese. Although the exact mechanisms of the cause of NPC remain obscure, a susceptible genetic constitution, some unknown environmental cofactors, and EBV all play a role. (73 refs)

79-3639 Animal Models for Nasopharyngeal Carcinoma. (Eng) Ablashi, D. V. (Viral Oncology Program, NCI, Bethesda, MD 20014); Easton, J. M.; Glaser, R. *IARC Sci Publ* 20: 85-94; 1978.

Two groups of animal models for nasopharyngeal carcinoma (NPC) are reviewed: (1) naturally occurring tumors of the mouth, pharynx, and nose in domestic animals; and (2) tumors induced in nude mice by human epithelial somatic cell hybrids containing the Epstein-Barr virus (EBV) genome. Of 469 oropharyngeal (OP) malignancies

diagnosed in one study of dogs, cats, horses, and cattle, 84% were in dogs. A significant number of these malignancies were squamous cell carcinomas. The most frequently observed canine OP neoplasm was carcinoma of the tonsil, which resembles the human nasopharynx in that lymphoid tissue is covered by a layer of epithelium. In domestic animals, the relative risk values for intranasal neoplasms and OP cancer tended to increase with age. The variation in the incidence of these cancers among different breeds of dogs was similar to the variation in NPC incidence among different human populations. The laboratory model for NPC involved tumor induction by the somatic cell hybrid D98/HR-1. (HR-1 is a lymphoblastoid line that contains EBV; D98 is a HeLa variant.) Tumors (undifferentiated carcinomas) induced by the D98/HR-1 cells were more aggressive than those induced by the two parental cell lines, which may be related to their association with EBV. Such tumors could serve as EBV-carrying models for Burkitt's lymphoma (HR-1 cells) and anaplastic NPC (D98/HR-1) in humans. Passage in nude mice seems to influence the number of EBV genomes in both the productive and non-productive states. Thus, these tumors could also serve as models for regulation of the EBV genome in Burkitt's lymphoma and NPC. (15 refs)

79-3640 Discussion Summary. (Eng) Henle, W. (Joseph Stokes, Jr. Res. Inst., Children's Hosp. of Univ. of Pennsylvania, Philadelphia, PA 19104). *IARC Sci Publ* 20: 421-423; 1978.

Discussions held at a symposium on nasopharyngeal carcinoma (NPC) are summarized. The conclusion that childhood Epstein-Barr virus (EBV) infections mostly remain silent may be erroneous, since such illnesses may be lost among the numerous upper respiratory tract illnesses that children suffer. The association of carcinomas of the head and neck with EBV seems limited to undifferentiated NPC's. Caucasian NPC patients seen at one clinic had serological patterns comparable to those seen in Chinese and African patients. Differentiation must be made between NPC patients before and after treatment, disease stage at the time of serum collection, elapsed period since initiation of therapy, and the presence of residual or recurrent tumor activity. EBV-related serology alone is insufficient to establish an association of EBV with a given malignancy. The demonstration of EBV DNA has been limited to cases of Burkitt's lymphoma and NPC. Undifferentiated carcinoma cells from NPC biopsies contain complete viral genomes. Cultured Epstein-Barr nuclear antigen (EBNA)-positive NPC cells can be maintained in culture for only a few weeks. Some of them can be induced to synthesize early antigen, viral capsid antigen, and virus particles. It is important to establish permanent lines of NPC cells as well as cultures of their normal epithelial progenitors to study the events involved in the transformation of epithelial cells by EBV. (no refs)

79-3641 Descriptive and Analytical Epidemiology of Nasopharyngeal Cancer. (Eng) Hirayama, T. (Epidemiology Div., Natl. Cancer Center Res. Inst., Tsukiji-5, Chuo-ku, Tokyo, Japan). *IARC Sci Publ* 20: 167-189; 1978.

Data on the descriptive and analytical epidemiology of nasopharyngeal carcinoma (NPC) that have been reported since the first international symposium on the subject in Singapore in 1964 are reviewed. NPC is rare in most countries in the world, with an age-adjusted incidence rate of <1/100,000, and the incidence rate is twice as high in men as in women. The southern Chinese have a uniquely high risk, the incidence rates per 100,000 being 10-20 in men and 5-10 in women. The greater the admixture of southern Chinese blood in a given ethnic group, the more likely it is that the NPC incidence rate in that group will be raised. The incidence in both sexes begins to rise after the ages of 20-24 and reaches a plateau at between 45 and 54. When the logarithm of mortality and morbidity is plotted against the logarithm of age the power of the age that provides the best fit to a straight line on a log-log graph is approx two to four. These figures are lower than those for other cancers. Seroepidemiological case-control studies indicate that both different birthplace and abnormal response to Epstein-Barr virus (EBV) antigen significantly enhance the risk for NPC; when these two factors are combined, the relative risk appears to rise further. The effect of other environmental chemicals, ie, those in cigarette smoke, shown to be significant in several retrospective studies, could explain in part epidemiological phenomena such as sex difference in incidence. The definitive reason for the uniquely high risk in southern Chinese should be further investigated by taking into account the interactions of host factors (birthplace, histocompatibility antigens, etc) and environmental factors (Epstein-Barr virus, chemical carcinogens including nitrosamines, excessive intake of salted fish, nutritional deficiencies, etc). (103 refs)

79-3642 Discussion of Risk Factors for Nasopharyngeal Carcinoma. (Eng) Henderson, B. E. (Dept. Community and Family Medicine, Univ. Southern California Sch. Medicine, Los Angeles, CA); Louie, E. *IARC Sci Publ* 20: 251-260; 1978.

Seven case-control studies of nasopharyngeal carcinoma (NPC) in southern Chinese populations, conducted from 1961-1974, are reviewed. 'Never married' and lower socioeconomic statuses were risk factors in most studies. A prior history of ear and nose disease and of the use of traditional Chinese medication (balms or oils) for nasal symptoms was a relatively consistent finding. Chinese tea was associated with NPC in some studies, but not in others. Alcohol consumption and smoking were not regularly associated with NPC risk. Most of the studies found an increased risk associated with exposure to inhaled smoke, fumes, and/or dust. For US subjects, contact with such

carcinogens would occur primarily at the workplace, whereas, for the Chinese, exposure would occur primarily in the home during childhood. The high risk for NPC in southern Chinese may also be due to exposure to Cantonese salted fish, in which carcinogenic nitrosamines have been detected. One study found a positive association between exposure to salted fish during the weaning period and NPC risk. Experimental carcinoma of the nasal cavity was induced in rats fed a diet of Cantonese salted fish or exposed to N-nitrosodimethylamine by artificial inhalation. Further study of the dietary patterns of the southern Chinese, especially during childhood, are needed. (28 refs)

- 79-3643 Cytologic and Histologic Correlation of Lung Tumors.** (Eng) Lukeman, J. M. (Cytology Section, Dept. Pathology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Texas Medical Center, Houston, TX 70030); Wilson, R. A.; Samuel, R. F. *Prog Cancer Res Ther* 11: 91-116; 1979.

The correlation of histologic and cytologic findings in lung tumor patients is reviewed. The histologic features and cytologic expression of well-, moderately well-, and poorly differentiated squamous carcinoma (SC) and adenocarcinoma, undifferentiated small (oat) cell carcinoma, large cell anaplastic carcinoma, giant cell carcinoma, and mucoepidermoid-carcinoma are described. Cytologic diagnosis correlates well with histologic diagnosis in well-differentiated and moderately well-differentiated SC and adenocarcinoma and in undifferentiated small cell carcinoma. Correlation is more difficult with poorly differentiated SC and adenocarcinoma. It is recommended that these cases be classified as "poorly differentiated carcinoma, type undetermined." Since cells of any tumor may undergo degenerative changes and make recognition and classification in sputum difficult, it is of major importance that diagnostic impressions be withheld until well-defined, intact, and nondegenerated cells have been examined and their cell type determined on the basis of distinctive characteristics. (2 refs)

- 79-3644 Multiple Diseases of the Gastrointestinal Tract. Part 1. Frequency and Classification, Accompanying Diseases, and Combined Diseases.** (Ger) Pusch, H. J. (Medizinische Poliklinik, Universität Würzburg, Klinikstrasse 8, 8700 Würzburg, W. Germany); Franke, H. *Fortschr Med* 97(13): 611-614; 1979.

Studies of multiple diseases and syndromes of the gastrointestinal tract are reviewed. There is as yet no explanation for the high incidence of ulcer in lung cancer patients, of ulcerative colitis in bile duct carcinoma patients, and of diabetes mellitus in pancreatic carcinoma patients. The cutaneous paraneoplastic syndromes associated with gastrointestinal tumors include malignant acanthosis

nigricans, seen in 68% of the patients with tumors of the stomach and palate; psoriasiform acrodermatitis, seen in 100% of the patients with tumors of the stomach and palate; lanuginous hypertrichosis, seen in 100% of the patients with gastrointestinal and pulmonary tumors; and dermatomyositis, seen in 50% of the patients with gastrointestinal and pulmonary tumors that are >40 yr of age. (62 refs)

- 79-3645 Dysplasia, Precancer, and Ulcerative Colitis (Letter to Editor).** (Eng) Nugent, F. W. (Dept. Gastroenterology, Lahey Clinic, 605 Commonwealth Ave., Boston, MA 02215); Haggitt, R. C.; Colcher, H.; Kuteruf, G. C. *Gastroenterology* 76(5, part 1): 1079; 1979.

The recommendation that colectomy be performed in ulcerative colitis patients with moderate or marked dysplasia is clarified. The presence or stage of carcinoma does not always correspond with the degree of dysplasia. Some patients who undergo colectomy following the discovery of severe dysplasia are found to have early lesions, and others have advanced carcinoma in the presence of only mild or moderate dysplasia. Dysplasia should be found in more than one biopsy specimen before colectomy is advised, and colonoscopy should still be used as a screening procedure for patients with long-standing ulcerative colitis. (no refs)

- 79-3646 Epidemiology of Colon Cancer.** (Fre) Audigier, J. C. (Hopital Edouard-Herriot, 69374 Lyon Cedex 2, France); Lambert, R. *Rev Prat* 29(13): 1055-1064; 1979.

The epidemiology and etiology of cancer of the colon are reviewed. The risk of colon cancer increases almost linearly with age in both sexes. The male/female ratio is usually higher than 1, but it is lower in Finland, the Netherlands, Canada, Chile, Venezuela and New Zealand. Mortality (per 100,000 population) is highest in Scotland (15.3), Canada (14.4), among whites in the US (13.7), and New Zealand (13.3). The incidence increases among European and Japanese immigrants in the US, which implicates environmental and, more specifically, dietary factors. Adenomatous polyps, intestinal inflammatory processes (hemorrhagic rectocolitis), and high-fat, low-fiber diets are the major etiological factors in colon cancer. The malignant transformation of Stage III or IV adenomatous polyps takes about 8-11 yr, that of Stage V polyps only 3.6 yr. Dietary fibers exert a protective effect against colon cancer by accelerating intestinal transit times. (13 refs)

- 79-3647 Does the Male Transmit Cervical Cancer?** (Eng) Singer, A. (Dept. Obstetrics-

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Gynecology, Univ. Sheffield, Sheffield, England); Reid, B. L. *Contemp Obstet Gynecol* 13(4): 173-180; 1979.

Evidence implicating men as carriers of an agent that induces cervical cancer (CVC) in their wives is presented. The characteristics of the high-risk man include a tendency toward genital cancer, a history of CVC in other spouses, and a position in the lower socioeconomic class. In a study of CVC in the second and, sometimes, third wives of men whose first wives had developed CVC, the incidence of CVC was 3.5 times that in a control series. In another study, the mortality from CVC for wives of men in the lowest socioeconomic grouping was more than five times that for wives of men in the highest grouping. Recent studies do not support previous suggestions that smegma or herpes simplex virus type 2 are the etiologic agent in CVC. Basic proteins have been extracted from the sperm of 200 subjects and analyzed as part of a study to link variations in the amount of each protein in each individual with his socioeconomic status. A histone:protamine ratio was established that varied widely from man to man but correlated with socioeconomic ranking. The lower the class, the greater the proportion of protamine and the lower the ratio. The association of sperm protein composition was as highly correlated with socioeconomic status as was the mortality of wives from CVC. The results suggest that the transmitted agent in CVC is a basic protein, an arginine-rich histone or a protamine. (15 refs)

- 79-3648 **Psychosomatic Problems in Gynaecology.** (Eng) Dening, F. C. (No affiliation given). *Nurs Mirror* 146(5): 7-9; 1978.

The role of psychosomatic effects in menstrual disorders, leukorrhea, premenstrual tension, prolactin secretion, obstetric and gynecologic pain, and breast and cervical malignancy is discussed. One study demonstrated a positive correlation between psychosociological factors and the characteristics and dimensions of neoplasms in 6,000 women. Separation, either in childhood or adult life, appears to be a predisposing event in cervical carcinoma. (no refs)

- 79-3649 **Early Coitus and Cervical Cancer (Letter to Editor).** (Eng) Sebastian, J. A. (Dept. Obstetrics and Gynecology, Duluth Clinic, Duluth, MN). *Human Sexuality* 13(4): 117; 1979.

Several studies suggest that sexual intercourse before the age of 18 predisposes to cervical cancer and that intercourse with multiple partners per se does not increase the risk of cervical cancer after the age of 18. Early coitus with more than one partner may increase the number of physical factors present that may, in turn, be responsible for the development of cervical cancer. (6 refs)

- 79-3650 **Early Coital Activity and Cervical Cancer (2 Letters to Editor).** (Eng) Eichner, E. (Severance Medical Arts Building, 5 Severance Circle, Cleveland Heights, OH 44118); Sebastian, J. A. *Am J Obstet Gynecol* 133(8): 940-941; 1979.

With respect to a previous article on cervical cancer in prostitutes, the need for considering the length of time that prostitutes with abnormal cytology were in the profession is pointed out. In a response, it is explained that length of employment was considered and that this factor was combined with age at the time of examination to obtain age at first coitus. Prior to analysis of the data, it was believed that greater dysplasia would be related to frequency of intercourse times the length of employment; however, the results suggested that early coitus is the major predisposing factor in cervical cancer. (no refs)

- 79-3651 **Disseminated Intravascular Coagulation and Related Syndromes: Etiology, Pathophysiology, Diagnosis, and Management.** (Eng) Bick, R. L. (2727 Eye St., Bakersfield, CA 93301). *Am J Hematol* 5(3): 265-282; 1978.

The etiology, pathophysiology, clinical and laboratory diagnosis, and management of disseminated intravascular coagulation (DIC) syndrome are reviewed. Other syndromes, not clinically considered in the spectrum of DIC, may actually share a similar or identical pathophysiology to that of DIC, even though they may be organ-specific, as in the hemolytic-uremic or respiratory distress syndromes. DIC may also be associated with solid malignancies and leukemias. (49 refs)

- 79-3652 **Iatrogenic and Factitious Thyroidal Disease.** (Eng) Zellmann, H. E. (Div. Endocrinology, Lahey Clinic, 605 Commonwealth Ave., Boston, MA 02215). *Med Clin North Am* 63(2): 329-335; 1979.

Pitfalls in the therapy of thyroid diseases are reviewed. Factitious hyperthyroidism, self-treatment with iodide, iatrogenic iodide goiter and myxedema, iodide and thyroid hormone therapy of nodular goiter, L-triiodothyronine therapy, misinterpretation of routine serum thyroid tests, induction of Graves' disease, thyroid problems during pregnancy, agranulocytosis, myxedema, and external irradiation are discussed. (11 refs)

- 79-3653 **Statistical and Epidemiological Considerations in Evaluating Environmental Cancer.** (Eng) Buffler, P. A. (Epidemiology Res. Unit, Dept. Preventive Medicine and Community Health, Univ. Texas Medical

Branch, Galveston, TX 77550). *Tex Rep Biol Med* 37: 64-93; 1978.

Three basic questions that should be considered in studies assessing environmental cancer are put forth, and three different approaches used in the study of disease patterns in human populations are reviewed. The first question relates to selecting health outcomes to be measured in monitoring populations for cancer risks. Indicators that may be used include cancer incidence, cancer mortality, childhood malignancies, premature deaths, specific morbidity as measured by certain biochemical parameters, reproductive outcomes, and birth defects. The second question relates to the concern today for the reported increase in US cancer deaths during the past 40 or 50 yr. In 1900, malignant neoplasms were the eighth leading cause of death and accounted for 3.7% of all deaths. In 1975, malignancies were

the second leading cause of death, accounting for 17.5% of all deaths. The significance of increased longevity (due to a decline in mortality from infectious diseases and diseases of infancy and childhood) in the increased cancer mortality is illustrated. The third question concerns the importance of environmental exposures in cancer etiology. Several cancer maps illustrating the geographic distribution of mortality rates from cancer of all sites as well as cancer at several different sites are presented. Three basic approaches used in epidemiology that help identify environmental conditions associated with cancer development, using cancer mortality data as examples, include: (1) correlating mortality rates with the prevalence of suspected factors; (2) observing the mortality experience of cohorts whose exposure to suspected agents has been previously defined; and (3) comparing the past experience or exposures of people with and without cancer. (10 refs)

CHEMICAL CARCINOGENESIS

- 79-3654 Evaluation of Chemical Carcinogenicity by In Vitro Neoplastic Transformation.** (Eng) Bouck, N. (Dept. Microbiology, Univ. Illinois at the Medical Center, Chicago, IL 60680); Di Mayorca, G. *Methods Enzymol* 48: 296-302; 1979.

A rapid and reliable method for evaluating the malignant transformation of cultured mammalian cells by chemical carcinogens is presented. The transformation of a fibroblast line of baby hamster kidney cells (BHK 21/cl 13) after brief treatment with a suspected carcinogen is assayed by testing the ability of the treated cells to form colonies in soft agar. This established BHK cell line grows rapidly (12-hr doubling time at 37 C), transforms with high frequency in response to a variety of carcinogens, clones readily to yield homogeneous populations, and is amenable to culture for periods of time sufficient to carry untreated controls to the end of an experiment. Although the cells are already "immortal", they are not already tumorigenic in vivo at moderate doses and require transformation in vitro for tumorigenicity. The soft agar assay is somewhat more cumbersome than some other assays, but it offers the advantage of single-step selection of cells capable of anchorage-independent growth. This characteristic of the transformed phenotype of a fibroblast cell is the only one consistently correlated with in vivo tumorigenicity. (18 refs)

- 79-3655 Metabolism of [¹⁴C]- and [³⁶Cl]-labeled Vinyl Chloride In Vivo and In Vitro.** (Eng) Guengerich, F. P. (Dept. Biochemistry, Center Environmental Toxicology, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Watanabe, P. G. *Biochem Pharmacol* 28(5): 589-596; 1979.

Studies were conducted to establish the roles of liver microsomal cytochrome P-450 and epoxide hydratase in the biotransformation of vinyl chloride (VC) to metabolites, particularly those bound to protein and nucleic acids. Label from [¹⁴C]VC was covalently bound to protein and nucleic acids in vivo and in vitro in the presence of Sprague-Dawley rat liver microsomal fractions or highly purified cytochrome P-450 and NADPH-cytochrome P-450 reductase preparations. The ratio of bound to total nonvolatile metabolites increased in going from the in vivo to the microsomal to the purified system. [³⁶Cl]VC was metabolized by microsomes and highly purified systems: no label was bound, and most of the metabolized chlorine could be accounted for as chloride ion. Phenobarbital pretreatment of rats did not induce total metabolism of VC in vivo at either the 10- or 250-ppm exposure levels; however, binding to protein and RNA was enhanced at the

10-ppm but not the 250-ppm level. Phenobarbital pretreatment increased the in vitro microsomal conversion of VC to both total and bound metabolites. A sizeable fraction of the label of [¹⁴C]VC metabolized in vivo was recovered in the microsomal fraction of the liver, but sodium dodecyl sulfate-polyacrylamide gel electrophoresis of in vitro incubations indicated that the metabolites were distributed among many microsomal proteins and not localized to cytochrome P-450. Evidence was obtained for the metabolism of the suspected VC metabolite chloroethylene oxide by microsomal epoxide hydratase. However, the epoxide hydratase inhibitor 3,3,3-trichloropropylene oxide, which blocks the microsomal degradation of chloroethylene oxide, did not enhance the level of VC bound to either protein or adenosine. (39 refs)

- 79-3656 On the Oncogenic Activity of Ethylene Oxide and Propylene Oxide in Mice.** (Eng) Dunkelberg, H. (Inst. Hygiene, Univ. Mainz, Hochhaus am Augustusplatz, D-6500 Mainz, Germany). *Br J Cancer* 39(5): 588-589; 1979.

The carcinogenic effects of ethylene oxide (EO: 0.1-1.0 mg/wk, sc) and propylene oxide (PO: 0.1-2.5 mg/wk, sc) in female NMRI mice were studied. Sarcomas appeared at the injection site in EO- and PO-treated mice but not in control animals. The first tumor appeared during the 39th week of PO treatment and during the 50th week of EO treatment. The number of sc tumors increased with increasing dosage, although at the low dosages of PO this effect was not clear. The number of tumors at sites distant from the injection site (mostly lymphomas) did not differ between controls and mice treated with EO or PO. (7 refs).

- 79-3657 The Mutagenic Effect of 1,2-Dichloroethane on *Salmonella typhimurium*. II. Activation by the Isolated Perfused Rat Liver.** (Eng) Rannug, U. (Division Toxicology Genetics, Wallenburg Lab., Univ. Stockholm, Stockholm, Sweden); Beijer, B. *Chem Biol Interact* 24(3): 265-285; 1979.

Isolated Wistar rat liver was perfused with a soln containing 1,2-dichloroethane (DCE), 1,2-dibromoethane (DBE), or 2-chloroethanol (CE), and the mutagenicities of the perfusates for *Salmonella typhimurium* strains TA1530 and TA1535 were tested. Bile samples (diluted 10-fold) produced by DCE-treated livers were strongly mutagenic for TA1535, the greatest values (800 and 600 mutants/plate) being observed 15 or 30 min after addition of DCE [360 micromoles (μ mol)] at 0 and 90 min, respectively. Bile from

DBE-treated livers (1 dose of 12 μ mol DBE) was also mutagenic for TA1535, producing 50 and 60 mutants/plate at 15 and 30 min, respectively. Bile from DCE-treated Sprague-Dawley rats was significantly less mutagenic than that of Wistar rat bile ($p < 0.001$), and the former was clearly more mutagenic after 30 min than after 15 min. CE was not mutagenic in this system. The results with DCE and DBE indicated an activation through conjugation to glutathione with a subsequent excretion through the bile. Bile produced by mice treated ip with DCE (80 mg/kg) was also mutagenic for TA1535, the mutagenicity being greater 30 min after injection than 60 min after injection. S-(2-chloroethyl)-L-cysteine and N-acetyl-S-(2-chloroethyl)-L-cysteine were equally mutagenic for TA1535 in the concentration range 0.2-0.6 μ mol/plate, whereas S-(2-hydroxyethyl)-L-cysteine was not directly mutagenic. Differences and similarities in the metabolism of DCE and vinyl chloride are discussed on the basis of these results. (49 refs)

- 79-3658 **Biological Effects of Polyvinyl Chloride in Rats.** (Rus) Shevchenko, A. M. (Dept. Vocational Hygiene, Medical Inst., Kiev, USSR); Shkurko, G. A.; Medvedev, V. N. *Vrach Delo* (2): 93-95; 1979.

Albino rats were exposed to daily inhalations of polyvinyl chloride (PVC: 100-150 mg/m³, 4 hr/day, for 6 mo). After the end of the experiment, the av content of hydroxyproline in the lung tissue was 69% greater than that in controls. Morphological examination of the lung tissue revealed the following severe disorders: plasmorrhagia, focal hemorrhages, perivascular sclerosis, and foci of emphysema. PVC induced dystrophic changes in the liver, kidneys, and cardiac muscle. (no refs)

- 79-3659 **The Reaction of Guanine with Some Potential Metabolites of 1-Chloropropene.** (Eng) Goldschmidt, B. M. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016); Van Duuren, B. L.; Goldstein, R. C. *Tetrahedron Lett* (14): 1177-1180; 1979.

The bifunctional alkylating agents 2-chloropropanal and 1-chloro-1,2-epoxypropane, two potential metabolites of 1-chloropropene, were shown to react with guanine in dimethyl sulfoxide to yield the monoalkylated product 2-chloro-N-propenyl-2N-guanine. 1-Chloropropene, the simplest homolog of vinyl chloride, is carcinogenic in animals. (22 refs)

- 79-3660 **Two-Year Oral Toxicity and Multigeneration Studies in Rats on Two Chemically Modified**

Maize Starches. (Eng) Truhaut, R. (Faculte des Sciences pharmaceutiques et Biologiques, Centre de Recherches Toxicologiques, 4 Avenue de l'Observatoire, 75006 Paris, France); Coquet, B.; Fouillet, X.; Galland, D.; Guyot, D.; Long, D.; Rouaud, J. L. *Food Cosmet Toxicol* 17(1): 11-17; 1979.

Three generations of Sprague-Dawley rats fed a diet consisting of 62% modified maize starch (acetylated distarch adipate or acetylated distarch glycerol) during a 2-yr period showed no increase in tumor incidence or other pathological changes in comparison with controls fed unmodified starch. Behavior, food consumption, fertility, litter size, embryonic or preweaning mortality, and histological features were also unaltered. (6 refs)

- 79-3661 **Covalent Binding of DBCP to Proteins In Vitro.** (Eng) Kato, Y. (Inst. Environmental Toxicology, Suzukicho 2-772, Kodaira, Tokyo 187, Japan); Matano, O.; Goto, S. *Toxicol Lett* 3(5): 299-302; 1979.

¹⁴C-1,2-Dibromo-3-chloropropane was found to bind covalently to proteins in vitro after activation by microsomal oxidase from a rat liver supernatant. (13 refs)

- 79-3662 **Organics in the Environment.** (Eng) Budde, W. L. (U.S. Environmental Protection Agency, Environmental Monitoring and Support Lab., 26 W. St. Clair St., Cincinnati, OH 45268); Eichelberger, J. W. *Anal Chem* 51(6): 567A-574A; 1979.

Technical and cost considerations affecting the selection of analytical methods to detect and measure organic pollutants are discussed, and a program to establish industrial wastewater effluent limitations for a group of priority pollutants is described. Two general goals affect the method selection process. The target compound (TC) goal requires measurement of the concentration of a particular compound(s). Since the TC is known, sample processing and measurement may be optimized for the TC. The broad spectrum (BS) goal seeks to establish a broad spectrum picture of whatever is present in a sample. The development of the computerized gas chromatography-mass spectrometry system has made this goal a feasible alternative for many types of samples. The most beneficial result of the BS approach is the frequent discovery of significant but previously unrecognized pollutants. The 129 priority pollutants were selected on the basis of known human or animal toxic and carcinogenic effects, and they include 106 specific organic compounds, 9 product formulations that are mixtures of organic compounds, 12 metals, cyanide ion, and asbestos. A program is underway to identify and measure these materials in various industrial wastewaters using gas chromatography-mass spectrometry. (3 refs)

CHEMICAL CARCINOGENESIS

79-3663 Possible Association of Lithium with Chronic Myelocytic Leukemia (Letter to Editor). (Eng)

Jim, R. T. (Dept. Medicine, Univ. Hawaii Sch. Medicine, Honolulu, HI). *Blood* 53(5): 1031; 1979.

A 56-yr-old woman developed clinical features consistent with chronic myelocytic leukemia (CML) after 11 mo of lithium therapy (900 mg/day). The patient presented with hepatosplenomegaly, leukocytosis (WBC 250,000/mm³), intense marrow hypercellularity with myeloid proliferation, and a positive marrow Philadelphia chromosome. Substitution of Li with Myleran resulted in resolution of all symptoms, including hepatosplenomegaly and hematological abnormalities. (no refs)

79-3664 Nickel Sub-Sulphide-induced Leiomyosarcoma in Rabbit White Skeletal Muscle. A Light Microscopical and Ultrastructural Study. (Eng)

Hildebrand, H. F. (Institut de Recherches sur le Cancer de Lille, U. 124 INSERM, Institut de Recherches sur le Cancer de Lille, B.P. no. 3567, 59020 Lille Cedex, France); Biserte, G. *Cancer* 43(4): 1358-1374; 1979.

Three nickel subsulfide (Ni₃S₂)-induced leiomyosarcomas in rabbit white skeletal muscle were studied by light and electron microscopy. The tumors, which appeared at the site of im Ni₃S₂ implantation, had identical histologic features. Two types of areas were observed: one characterized by irregular cords of cells with clear spaces around small pleomorphic nuclei; and another characterized by interlacing fascicles of spindle-shaped smooth muscle cells, often with elongated nuclei. The most common cells were small spindle cells separated by collagen fibrils. The cells contained randomly oriented, closely packed microfibrils averaging 7 nanometers (nm) in thickness. They also contained voluminous nuclei with one or more large nucleoli and one or several nuclear bodies, large areas of Golgi apparatus, some mitochondria, large vesicles, and rough endoplasmic reticulum. The elongated smooth muscle cells were interlaced with the spindle cells, and they contained the same elements as the spindle cells. Desmosomal junctions and some gap junctions were observed. In the tumors, the Ni₃S₂ implants were generally surrounded by a capsule, the major components of which were collagen fibers, degenerated nuclei, and rodlike structures with a transverse periodicity of 15.5 nm. (50 refs)

79-3665 Long-Term Toxicity and Carcinogenicity Studies of the Bread Improver Potassium Bromate. 1. Studies in Rats. (Eng) Fisher, N. (Flour Milling and Baking Res. Assoc., Chorleywood, Rickmansworth, Herts. WD3 5SH, England); Hutchinson, J. B.; Berry, R.; Hardy, J.; Ginocchio, A. V.; Waite, V.

Food Cosmet Toxicol 17(1): 33-39; 1979.

The appearance, behavior, and health of Wistar rats fed

for 104 wk on bread-based diets in which the bread was prepared from potassium bromate-treated flour (50 or 75 ppm, alone or in combination with other commonly-used additives) were similar to those of rats fed bread containing untreated flour. The death rate was lower in test females than in control females, and high-dose males experienced significantly fewer deaths than the other groups taken together. No evidence of carcinogenicity or chronic toxicity was attributable to the compounds tested. (21 refs)

79-3666 Long-Term Toxicity and Carcinogenicity Studies of the Bread Improver Potassium Bromate. 2. Studies in Mice. (Eng) Ginocchio, A. V. (Consultox Labs. Ltd., 188 Brent Crescent, London NW10 7XR, England); Waite, V.; Hardy, J.; Fisher, N.; Hutchinson, J. B.; Berry, R. *Food Cosmet Toxicol* 17(1): 41-47; 1979.

Long-term toxicity and carcinogenicity studies of the oxidative bread improver potassium bromate (PB) were made in mice of Theiller's Original strain. The mice were fed for 80 wk on five bread-based diets in which the bread was prepared from untreated flour or from flour treated with 50 or 75 ppm PB or with one of two mixtures of PB with other commonly used additives. Appearance, behavior, health, and survival were similar in test and control groups. No carcinogenic effects were produced by any of the diets. Anemia was present in all male groups (including controls) except the group receiving 50 ppm PB together with three other flour additives (30 ppm ascorbic acid, 50 ppm benzoyl peroxide, and 15 ppm chlorine dioxide), and in females terminally. There was a dose-related decrease in the RBC count of males at 3 mo, and the neutrophilia seen at 12 and 18 mo was dose-related. Raised blood-sugar levels related to dose were found in females at 3 and 12 mo, but the effect was not significant at 18 mo, and it was not found at any stage in the males. Dose-related differences in heart, pituitary and uterus wts were found; when expressed relative to body wt, values for the heart and uterus were no longer dose-related, but pituitary, brain, kidney, and thyroid wts showed significant dose-related trends. These effects were not associated with pathological changes in the structures of the tissues concerned. (4 refs)

79-3667 Differential Cytotoxic Activity of Potassium Dichromate on Nucleoside Uptake in BHK Fibroblasts. (Eng) Bianchi, V. (Inst. Animal Biology, Univ. Padova, Via Loredan 10, 35100 Padua, Italy); Levis, A. G.; Saggiaro, D. *Chem Biol Interact* 24(2): 137-151; 1979.

In cultures of baby hamster kidney (line BHK21) fibroblasts treated with potassium dichromate (10⁻⁵ to 10⁻³ M for 1-10 hr), nucleic acid and protein syntheses are differentially inhibited, and nucleoside uptake into the in-

tracellular pool is characterized by a stimulation phase followed by an inhibition phase. Different patterns are observed for the uptake of each ribo- and deoxyribonucleoside, with pyrimidine nucleoside (particularly deoxycytidine) uptake reaching the highest stimulation level. The kinetics of the initial uptake of thymidine and deoxycytidine at different exogenous nucleoside concentrations show that $K_2Cr_2O_7$ affects both simple and facilitated diffusion of nucleosides. The time course of thymidine and deoxycytidine pool saturation suggests, however, that the effects of $K_2Cr_2O_7$ on plasma membrane permeability are partially counterbalanced by modifications of pool size due to the concomitant alteration of nucleoside metabolism steps separate from nucleoside uptake. The inhibition of macromolecular synthesis depends on the length of treatment, treatment soln [Eagle's minimal essential medium (MEM) supplemented with 10% calf serum or Hank's balanced salt soln (BSS)], and $K_2Cr_2O_7$ concentration. DNA replication is mainly and immediately affected, whereas RNA and protein syntheses are reduced to a less extent and later. The inhibition of macromolecular synthesis is more marked with BSS; the lower cytotoxic activity displayed by $K_2Cr_2O_7$ in MEM is due to a more pronounced Cr^{6+} reduction, which occurs immediately after dichromate solubilization. (31 refs)

79-3668 Ileal Resection Potentiates 1,2-Dimethylhydrazine-induced Colonic Carcinogenesis. (Eng) Oscarson, J. E. (Surgical Services, Shriners Burns Inst., Boston, MA); Veen, H. F.; Ross, J. S.; Malt, R. A. *Ann Surg* 189(4): 503-508; 1979.

The effect of colonic hyperplasia produced by resection of the distal third of the small bowel (DSBR) on the development of dimethylhydrazine (DMH)-induced colon carcinogenesis was studied in male Sprague-Dawley rats. Neither DSBR nor DMH (10 mg/kg, sc) had a significant effect on wt gain. The mortality rate was 100% in the control group, 94% in the DMH only group, 81% in the DSBR + vehicle group, and 82% in the DSBR + DMH group. DSBR produced adaptive mucosal hyperplasia in the remaining bowel mucosa, with increases in DNA and RNA levels. DMH treatment also increased DNA and RNA levels in the bowel mucosa. In general, the amounts of RNA and DNA in the small bowel were the same with the combined treatments as with either DMH or DSBR alone; quantities of nucleic acids tended to increase only in the transverse and distal colon. After 37 wk, the number of neoplasms per rat was increased six-fold by combining DSBR with DMH. Neoplasms were spread throughout the colon after combined treatment, but they were confined to the ascending colon after DMH alone. Postresectional hyperplasia appeared to increase the incidence and distribution of colon tumors. (26 refs)

79-3669 1-Acetyl-2-phenylhydrazine Carcinogenesis in Mice. (Eng) Toth, B. (Eppley Inst. Res.

Cancer, Univ. Nebraska Medical Center, Omaha, NE 68105). *Br J Cancer* 39(5): 584-587; 1979.

The tumorigenicity of 1-acetyl-2-phenylhydrazine (APH: 0.015% in the drinking water for life) was studied in Swiss albino mice. Blood vessel tumors developed in 32% of the APH-treated females (8 angiomas and 8 angiosarcomas), 24% of the treated males (7 angiomas and 5 angiosarcomas), 8% of the control females (4 angiomas and 4 angiosarcomas), and 5% of the control males (3 angiomas and 2 angiosarcomas). The difference in tumor incidence between treated and control mice was significant. All of the blood vessel tumors in the ADP-treated animals occurred in the liver and, to a lesser extent, the spleen, whereas they were found in the liver, ovary, uterus, lymph node, anal gland, and pararenal fat of the controls. The incidence of other types of tumors in both groups was low, and it was lower in the APH-treated mice than in the controls. (23 refs)

79-3670 Sites of Inhibition of In Vitro DNA Synthesis in Carcinogen- and UV-treated ϕ X174 DNA. (Eng) Moore, P. (Dept. Microbiology, Univ. Chicago, Chicago, IL 60637); Strauss, B. S. *Nature* 278(5705): 664-666; 1979.

The sites of inhibition of in vitro DNA synthesis in ϕ X174 DNA treated with UV radiation or N-acetoxy-2-acetylaminofluorene (AAAF) were investigated. In the reaction with AAAF-treated DNA, a number of bands were seen on polyacrylamide gels that did not appear with untreated DNA. In the three cases in which the bands were identifiable, synthesis had stopped at the base before a guanine in the DNA template. There seemed to be no preferential reaction of AAAF with particular guanines in the section of DNA tested, and there was no indication of lesions blocking synthesis at any position other than guanine. Synthesis on the UV-irradiated template terminated one nucleotide before any pair of pyrimidine bases. All three pyrimidine dimers, ie, cytosine (C)-C, C-thymine (T), and T-T, were blocks to DNA synthesis, although there was some indication that the bands resulting from T-T dimers were more prominent. Guanine appeared to be the major site of reaction for (\pm)7,8-dihydroxy-9,10-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene, but the reaction also occurred at other bases, notably adenine. The data suggest that synthesis in all cases terminated because of the inability of DNA polymerase to incorporate nucleotides opposite a nonpairing lesion. (19 refs)

79-3671 Transplacental and Perinatal Carcinogenicity of 2-Acetylaminofluorene in Rats (Meeting Abstract). (Eng) Pitts, C. M. (Dept. Biology, Univ. Alabama, University, AL 35486); Lindahl, R. *Proc Am Assoc Cancer Res* 20: 1; 1979 (no refs)

CHEMICAL CARCINOGENESIS

79-3672 Contamination of Beer with Trace Quantities of N-Nitrosodimethylamine. (Eng) Spiegelhalder, B. (Institut für Toxikologie und Chemotherapie, Deutschen Krebsforschungszentrum, Heidelberg, W. Germany); Eisenbrand, G.; Preussmann, R. *Food Cosmet Toxicol* 17(1): 29-31; 1979.

Of 158 samples of commercially available draft, bottled, and canned beers analyzed by chemiluminescence for the presence of volatile nitrosamines, 111 were found to contain N-nitrosodimethylamine. The mean concentration was 2.7 ppb, and the max was 68 ppb. N-Nitrosodiethylamine was the only other volatile nitrosamine found, and it occurred in only two samples (at 0.5 and 3.0 ppb). (5 refs)

79-3673 Analysis for Volatile Nitrosamines in Salt-preserved Foodstuffs Traditionally Consumed by Southern Chinese. (Eng) Huang, D. P. (Medical and Health Dept., Inst. Radiology Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Ho, J. H.; Gough, T. A. *IARC Sci Publ* 20: 309-314; 1978.

Because of the prevalence of nasopharyngeal carcinoma (NPC) among the southern Chinese, 6 types of salted fish and 10 other salted food products traditionally consumed by these people were analyzed for the presence of volatile nitrosamines by gas chromatography and high-resolution mass spectrometry. The detection limit in the original raw food was 1 µg/kg. Volatile nitrosamines were not detected in any of the 10 salted food products (pork sausage, goose liver sausage, salt-dried beans, soya bean paste, soy, shrimp paste, oyster sauce, soya bean curd, salt-dried egg yolk, and fish sauce) or in 2/6 types of salted fish. N-Nitrosodimethylamine was detected in salted anchovies, croakers, white herring, and yellow croakers at concentrations of 1-35 µg/kg. However, these concentrations are no higher than those found in cured meats consumed in Europe, where the incidence of NPC is low. (17 refs)

79-3674 DNA Damage Induced In Vivo Evaluated with a Nonradioactive Alkaline Elution Technique. (Eng) Schwarz, M. (Inst. Biochemistry, German Cancer Res. Center, Neuenheimer Feld 280, 6900 Heidelberg, W. Germany); Hummel, J.; Appel, K. E.; Rickart, R.; Kunz, W. *Cancer Lett* 6(4): 221-226; 1979.

A modification of the alkaline filter elution test in which DNA is measured colorimetrically was used to study damage to liver DNA of NMRI mice following treatment with hepatocarcinogens. Diethylnitrosamine (DEN), dimethylnitrosamine (DMN), N-nitrosomorpholine (NMP), and methyl methanesulfonate (MMS) were given ip, and N-acetylaminofluorene (AAF) and 4-dimethylaminoazobenzene (DAB) were given ip or by stomach tube. Liver cell nuclei were prepared and lysed on

top of polyvinyl filters. DNA was eluted, and the amounts remaining on the filter and in the elute were measured colorimetrically. DMN (10 mg/kg), DEN (100 mg/kg), NMP (100 mg/kg), and MMS (150 mg/kg) significantly enhanced DNA passage through the filters, whereas AAF (20-100 mg/kg ip or po), DAB (100 mg/kg ip or po), carbon tetrachloride (2.5 ml/kg po), phenobarbital (0.1% in the drinking water), and halothane (1% by inhalation) did not. Thus, all the alkylating compounds generated DNA strand breaks and enhanced DNA elution under alkaline conditions, whereas AAF, DAB, and CCl₄ gave no indication that alkali-sensitive reaction products were formed. This imposes a limit on the value of the method for large-scale screening of chemical carcinogens. However, the method does allow a balance to be struck between DNA damage and repair in a considerably shortened period of time and does not require radioactive carcinogens or DNA. (12 refs)

79-3675 The Detection of Various Nitrosamines in the Hepatocyte Primary Culture/DNA Repair Test. (Eng) Williams, G. M. (Naylor Dana Inst. Disease Prevention, American Health Foundation, 1 Dana Road, Valhalla, NY 10595); Laspi, M. F. *Cancer Lett* 6(4): 199-206; 1979.

The utility of the hepatocyte primary culture (HPC)/DNA repair test for detecting carcinogens was tested with nine nitrosamines, six of which were carcinogenic and three noncarcinogenic. Simultaneous exposure to the test compound in a range of logarithmic doses and ³H-thymidine (³H-TdR) began after a 1.5-hr attachment interval. Several different protocols were evaluated: (1) simultaneous exposure to the test compound and ³H-TdR for 5 hr; (2) removal of the test compound and ³H-TdR at 5 hr followed by incubation for 15 hr; (3) removal of the test compound and ³H-TdR at 5 hr followed by replacement of ³H-TdR for the next 15 hr; and (4) simultaneous exposure for 20 hr. Autoradiographic measurement of unscheduled DNA synthesis (UDS) indicated that the six carcinogens [dimethylnitrosamine, diethylnitrosamine, nitrosomorpholine, nitrososornicotine, 4-(N-methyl-N-nitrosoamino)-1-(3-pyridyl)-1-butanone, and nitrosopyrrolidine] elicited DNA repair, whereas the noncarcinogens (diphenylnitrosamine, dimethylformamide, and nitrosocarbazole) did not. The background counts in these experiments were variable and not consistently related to any protocol. These results suggest that the HPC/DNA repair test may be a valuable addition to bacterial mutagenesis tests in a screening battery. Satisfactory screening results were obtained with protocols 1, 2, and 4. (15 refs)

79-3676 Carcinoma of the Nasal and Paranasal Regions in Rats Fed Cantonese Salted Marine Fish. (Eng) Huang, D. P. (Medical and Health Dept., Inst.

Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Ho, J. H.; Saw, D.; Teoh, T. B. *IARC Sci Publ* 20: 315-328; 1978.

Cantonese salted fish is suspected on epidemiological grounds to be an etiological factor in human nasopharyngeal carcinoma (NPC). The incidence and morphology of the tumors that developed in the nasal cavities and paranasal regions of WA albino rats and Syrian golden hamsters fed Cantonese salted fish in their diet for 1-2 yr from the age of 1 mo are reported. Three of 20 treated rats, but no control rats and no treated or control hamsters, developed carcinomas (2 adenocarcinomas and 1 undifferentiated carcinoma) in the nasal or paranasal sinus cavities after 12-24 mo treatment. N-Nitrosodiethylamine (NDEA) was given po (0.5 mg/wk) to a similar group of animals as a positive control, and N-nitrosodimethylamine, the only volatile nitrosamine detected in salted fish, was added to the drinking water of the third group (2.5 mg/wk). Three of 14 NDEA-treated rats developed adenocarcinomas in the nasal cavities, but none of the other animals developed nasal or paranasal tumors. These findings suggest that salted fish may contain a carcinogen or procarcinogen that can act systematically on the epithelial cells of the nasal and paranasal cavities. (12 refs)

79-3677 Studies on Lung Tumours. IV. Correlation Between [³H]Thymidine Labelling of Lung and Liver Cells and Tumour Formation in GRS/A and C3Hf/A Male Mice Following Administration of Dimethylnitrosamine. (Eng) De Munter, H. K. (Div. Chemical Carcinogenesis, Antoni van Leeuwenhoek-huis, Netherlands Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam, Netherlands); Den Engelse, L.; Emmelot, P. *Chem Biol Interact* 24(3): 299-316; 1979.

Male GRS/A mice are highly susceptible to lung tumor formation but resistant to liver tumor formation, whereas the opposite relation holds for male C3Hf/A mice. The liver and lung cells of these two mouse strains were studied autoradiographically after ip injection of [³H]dimethylnitrosamine (DMN) and [³H]thymidine (TdR) 1-14 days after treatment of the mice with unlabeled DMN (5 or 7 mg/kg ip). The [³H]DMN and [³H]TdR were injected 1 hr and 30 min prior to sacrifice, respectively. Corresponding cell types in the lungs and livers of these two mouse strains bound similar amounts of [³H]DMN. Among the various types of lung cells, only the alveolar type II cells, from which lung adenomas originate, showed a strain-specific difference in [³H]TdR-labeling indices, with many more cells being labeled in GRS/A mice than in C3H/A mice 3-7 days after DMN administration. Opposite TdR-labeling indices were exhibited by the parenchymal liver cells of the two strains, with C3Hf/A males showing a greater response than GRS/A males. Thus, the TdR-labeling and tumorigenic responses of target lung and liver cells to a carcinogen coincided in the two strains. Sulfur

dioxide and carbon tetrachloride mimicked the effects of DMN on the TdR-labeling indices of the lung alveolar type II and liver parenchymal cells of the two strains. (22 refs)

79-3678 Lung Tumorigenesis in Mice after Chronic Exposure in Early Life to a Low Dose of Dimethylnitrosamine. (Eng) Anderson, L. M. (Memorial Sloan-Kettering Cancer Center, Donald S. Walker Lab., Rye, NY 10580); Priest, L. J.; Budinger, J. M. *J Natl Cancer Inst* 62(6): 1553-1555; 1979.

Strain A female mice were given 10 ppb dimethylnitrosamine (DMN) in the drinking water starting 4 wk before mating and continuing until the progeny were 22 wk old, and the incidence of lung tumors was determined. The only abnormalities in the progeny were lung tumors (adenomas). The incidence of lung tumors among the treated progeny (23%) was significantly higher than that among controls (8%). The male offspring of DMN-treated dams had a significantly higher incidence of lung tumors (32%) than the male offspring of control females (4.3%). The incidence of lung tumors in the female offspring of DMN-treated dams was not significantly higher than that in the control offspring. The results indicate that nitrosamines occurring as part-per-billion contaminants present a real possibility of tumorigenicity. (34 refs)

79-3679 Formation of Relatively Persistent O²-Ethylthymidine by Diethylnitrosamine in Rat Liver DNA. (Eng) Steward, A. P. (Div. Chemical Carcinogenesis, Antoni Van Leeuwenhoek-Huis, Netherlands Cancer Inst., 121, Plesmanlaan, 1066 CX Amsterdam, Netherlands); Scherer, E.; Emmelot, P. *FEBS Lett* 100(1): 191-194; 1979.

The formation of O²-ethylthymidine (OET) in liver DNA of rats treated with diethylnitrosamine (DEN) is reported. Female Sprague-Dawley rats were given [¹⁴C]DEN (10 mg/kg), and the livers were excised 3 hr later. DNA was extracted from the liver and heated before enzymic hydrolysis. The enzyme digest was chromatographed on a Dowex 50 column. One of the peaks was identified as OET by comparison with an authentic sample. Conclusive evidence came from the acidic conversion of OET to O²-ethylthymine. Three hours after DEN treatment, rat liver DNA contained three thymidine groups ethylated at the O²-position per 10⁶ deoxynucleotides, 6% of the total binding of ethyl groups to DNA. Determination of OET levels in rat liver DNA at different times after DEN treatment revealed that OET is persistent in vivo: 2.2 micromoles (μmol)/mol DNA-P were measured after 2 wk, 1.2 μmol/mol DNA-P after 4 wk. This represents a half-life of 20 days for OET in rat liver DNA. Other studies have suggested that DNA synthesis may be hampered at the site of O²-alkylthymine, resulting in an increased error frequen-

cy during replication of O²-alkylthymine-containing DNA. The induction of mutations in vivo would be favored by the persistence of this lesion. Since OET was quite persistent in rat liver DNA, its formation could be relevant for hepatocarcinogenesis. (15 refs)

79-3680 Increased Excretion of Urinary Porphyrins by White Rats Given Intragastrically the Chemical Carcinogens Diethylnitrosamine, Monocrotaline, T-2 Toxin and Ethylmethanesulphonate. (Eng) Schoental, R. (Dept. Pathology, Royal Veterinary Coll., Royal Coll. St., London NW1 0TU, England); Gibbard, S. *Biochem Soc Trans* 7(1): 127-129; 1979.

The effect of chemical carcinogens on the excretion of porphyrins by white rats was studied. In rats given monocrotaline, T-2 toxin ([4 β ,15-diacetoxy-8 α -(3-methylbutyryloxy)-12, 13-epoxytrichothec-9-en-3 α -ol]) or ethyl methanesulphonate intragastrically at approx the LD₅₀, the urinary excretion of porphyrins increased progressively for 2-6 days and then declined to pretreatment values within the next few days. If the drug was readministered, the urinary excretion of porphyrins showed the same pattern. The max urinary excretion of coproporphyrins after carcinogen treatment was up to several times higher than the excretion by untreated rats or rats given aqueous ethanol. Increased porphyrin excretion indicated inhibition of heme formation, especially at the stage of insertion of the metal iron. The results indicate that increased porphyrin excretion may be related to carcinogenic activity. (9 refs)

79-3681 DNA Synthesis with Methylated Poly(dC-dG) Templates. Evidence for a Competitive Nature to Miscoding by O⁶-Methylguanine. (Eng) Abbott, P. J. (Dept. Pharmacology and Experimental Therapeutics, Albany Medical Coll., Albany, NY 12208); Saffhill, R. *Biochim Biophys Acta* 562(1): 51-61; 1979.

The coding properties of methylated cytosine and guanine bases were investigated with the use of an assay system that detects very small amounts of misincorporation into newly synthesized polynucleotide. The alternating copolymer poly(dC-dG) was methylated by dimethyl sulfate (DMS) or N-methyl-N-nitrosourea (MNU), and the levels of the various methylation products were determined. In addition to 3-methylcytosine, 3-methylguanine, and 7-methylguanine (produced by both agents), reaction with MNU also yielded easily detectable amounts of O⁶-methylguanine (OMG) and phosphotriesters. These methylated polymers were then used as templates in an in vitro assay with *Escherichia coli* DNA polymerase I in which the incorporation of complementary [deoxycytosine monophosphate (dCMP) and deoxyguanosine monophosphate] and noncomplementary [dAMP and

deoxythymidine monophosphate (dTMP)] nucleotides was measured. When the DMS-methylated polymer was used as template, there was virtually no detectable incorporation of noncomplementary nucleotides, indicating that no miscoding could be attributed to the presence of 3-methylcytosine, 3-methylguanine, or 7-methylguanine. When the MNU-methylated polymer was used as template, however, there was a specific incorporation of dTMP but not of dAMP. The amount of dTMP incorporated was always less than the level of OMG in the template, and it varied with the relative concentrations of the deoxynucleoside 5'-triphosphates in the assay. As the amount of dCTP decreased, the misincorporation of dTMP increased and approached a level that would have been expected for a one-to-one miscoding by OMG as the concentration of dCTP approached zero. The results indicate that OMG is capable of miscoding with a DNA polymerase, but that the miscoding is competitive with the normal incorporation of dCMP. When the 5'-triphosphate precursors are present in equal amounts, approx one OMG in three miscodes, leading to the incorporation of dTMP. The results support the view that OMG is a promutagenic base when present in DNA. (30 refs)

79-3682 O⁶-Methylguanine Accumulates in DNA of Mammary Glands after Administration of N-Methyl-N-nitrosourea to Rats. (Eng) Cox, R. (Cancer Res. Lab., Veterans Admin. Hosp., 1030 Jefferson Ave., Memphis, TN 38104); Irving, C. C. *Cancer Lett* 6(4): 273-278; 1979.

The methylation of mammary gland DNA following treatment with N-methyl-N-nitrosourea (MNU) was studied in female Sprague-Dawley rats. Rats were injected iv with various doses of MNU and killed 4 hr or 1, 3, or 7 days later. When mammary gland epithelial cells from a control rat lysed and the DNA was sedimented in an alkaline sucrose gradient, the DNA was fragmented, but the major part remained in the bottom third of the gradient. Treatment with MNU (7, 10, or 20 mg) did not cause a shift of the DNA out of the bottom third of the gradient. Methylated products found in the DNA of mammary gland epithelial cells after MNU administration were determined by high-pressure liquid chromatography. 3-Methyl-adenine was not detected at any time after MNU treatment. O⁶-Methylguanine (OMG) levels decreased at 4 hr but then were stable over the 7-day period, whereas 7-methylguanine levels continued to decrease. OMG and 7-methylguanine levels increased threefold after a second iv injection of 7 mg MNU. A comparison with previous studies indicated that the OMG level in the mammary gland following the second dose of MNU was equal to that in the bladder and brain after four doses of MNU. In three different tissues after repeated doses of MNU, the OMG level was nearly the same, 40-50 moles/10⁶ moles of guanine residues. The amount of OMG that persists may be a carcinogenic level. (13 refs)

- 79-3683** Effect of Simultaneous Administration of Saccharin or Cyclamate and a Nitrosamine (MNU) on Bladder Epithelium and the Dominant Lethal Test. (Eng) Aeschbacher, H. U. (Nestle Products Technical Assistance Co., Ltd., Biological Experimentation Services, CH-1350, Orbe/VD, Switzerland); Bexter, A.; Wurzner, H. P.; Luginbuhl, H. *Toxicol Lett* 3(5): 273-278; 1979.

The possible comutagenic effect of sodium saccharin or sodium cyclamate and N-methylnitrosourea was studied in Swiss mice. Three groups of male mice (150-160 animals/group) received 300 mg/kg methylurea + 15 mg/kg sodium nitrite daily, by gavage, plus (Group 2) 3.8 g/kg saccharin or (Group 3) 1.9 g/kg cyclamate in the diet. The treatment was given for 1 wk followed by 3 wk on a normal diet. Each male was mated 1 and 3 wk after treatment. Females were killed 13 days after mating and scored for dead and living implants. After mating, the males were returned to the same test regimen for 3 mo and then examined histologically. No hyperplastic or neoplastic changes were observed in the bladders or livers after 3 mo of treatment. Focal nonneoplastic proliferation of lymphoreticular cells was observed in the bladder mucosa and submucosa, but it was attributed to the action of MU + nitrite, since there was no significant difference between the test groups. The incidence of focal nodular proliferation of alveolar epithelium in the lung was increased significantly in all groups that received MU + nitrite. A significant induction of dominant lethals was observed in the groups that received MU + nitrite alone or in combination with sodium saccharin, but the number of dominant lethals was only slightly increased with cyclamate. It is concluded that a very weak induction of dominant lethals occurred in all three groups and that it was exclusively due to MU + sodium nitrite. (17 refs)

- 79-3684** Carcinogenicity Test of Six Nitrosamides and a Nitrosocyanamide Administered Orally to Rats. (Eng) Bulay, O. (Ankara Universitesi Tip Fakultesi Patoloji Kursusu, Morfoloji, Ankara, Turkey); Mirvish, S. S.; Garcia, H.; Pelfrene, A. F.; Gold, B.; Eagen, M. *J Natl Cancer Inst* 62(6): 1523-1528; 1979.

The carcinogenicity of ethylnitrosourea (ENU), 2-hydroxyethylnitrosourea (HENU), carboxymethylnitrosourea (CMMU), 1-nitroso-5,6-dihydrouacil (NDHU), 1-nitrosohydantoin (NHYD), N-methyl-N-nitrosobenzamide (MNB), and ethylnitrosocyanamide (ENC) was studied. The compounds were administered in the sodium citrate-buffered drinking water of MRC Wistar rats. ENU induced tumors of the reticuloendothelial system (RES, 50% incidence), mammary glands, and large intestine; it also significantly increased the incidence of forestomach tumors in male rats. HENU caused tumors of the small intestine, RES, and bone. CMNU increased the incidences of gastrointestinal (GI), adrenal, and epidermal tumors.

NDHU was associated with a 96% incidence of live tumors and an increased incidence of tumors at the injection site following ip administration. NHYD produced GI and RES tumors and lymphocytic lymphomas. Forestomach tumors were significantly increased after MNB, and tumors of the nasal cavity, esophagus, and forestomach were produced by ENC. The upper GI tumors produced by CMNU, NHYD, MNB, and ENC were attributed to local action related to ingestion of the compounds. (19 refs)

- 79-3685** Modification of Renal Tumorigenesis by Gonadal Ablation in Rats Treated with N-Butylnitrosourea or Dimethylnitrosamine. (Eng) Takizawa, S. (Dept. Cancer Res., Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Hiroshima, Japan); Hirose, F. *Hiroshima J Med Sci* 27(4): 247-252; 1978.

Hormonal modification of renal tumorigenesis was studied in rats using N-nitrosobutylurea (BNU), a multipotent but weak renal carcinogen, and dimethylnitrosamine (DMN), a procarcinogen acting selectively on the kidney. BNU (5 mg/15 ml) was administered in the drinking water of 8-wk-old W/Fu rats. Seven groups were used: intact females, ovariectomized females, ovariectomized and ovary-regrafted females, intact males, orchiectomized males, orchiectomized and ovary-grafted males, and orchiectomized males injected with 0.4 mg progesterone and 0.01 mg estradiol benzoate 2x/wk for 7 mo. Kidney tumor incidence was 18.5% in intact females and 24.0% in intact males. In castrated rats, however, the incidence dropped to 4.7% and 0% in females and males, respectively. Kidney tumor induction was significantly restored in the castrated rats by ovarian grafting or hormone injections. DMN was given po (6 doses of 0.8 mg/0.5 ml saline/100 g body wt at 2- to 3-day intervals) to 8-wk-old JCL:SD rats and sc (2 doses of 0.1 mg/ml) to neonatal JCL:SD rats at birth and at age 1 wk. Castrated and sham-castrated rats of both sexes were studied. Castration did not affect DMN renal carcinogenesis in adult or neonatal rats of either sex. This discrepancy is explained by the difference in carcinogenic potency between the two agents: DMN-induced preneoplastic and neoplastic changes were more numerous and readily escaped the influence of sex hormones. The results also suggest that both genetic factors and hormonal environment play a role in determining the histologic type of kidney tumors induced by BNU but not by DMN. (20 refs)

- 79-3686** Carcinogenicity Tests in Rats of Two Nitrosamines of High Molecular Weight, Nitrosododecamethyleneimine and Nitrosodi-n-octylamine. (Eng) Lijinsky, W. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, P.O. Box B, Frederick,

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MD 21701); Taylor, H. W. *Ecotoxicol Environ Saf* 2(3/4): 407-411; 1978.

Nitrosododecamethyleneimine (NDMI) and nitrosodioctylamine (NDOA), the largest molecules in their respective nitrosamine classes, were tested for carcinogenicity in Sprague-Dawley rats. The animals were administered 0.2 ml of an olive oil soln (120 mg/ml for NDMI and 150 mg/ml for NDOA) of either nitrosamine by gavage twice a week. Treatment lasted 50 wk, so that each rat received a total of 2.4 g NDMI and 3 g NDOA. The cyclic nitrosamine NDMI was definitely carcinogenic, although not very potent, giving rise to a 30% incidence of hepatocellular carcinomas after administration of 2.4 g per rat (12 g/kg). The aliphatic nitrosamine NDOA did not induce a significant incidence of tumors in rats after administration of 3 g per rat (15 g/kg). It is concluded that NDMI does not exceed the limit in size of cyclic nitrosamines that are carcinogenic, but NDOA is at or beyond the limiting size of symmetrical aliphatic nitrosamines consistent with carcinogenic activity. (15 refs)

79-3687 Mutagenicity and Mutagenic Specificity of Metronidazole and Niridazole in *Neurospora crassa*. (Eng) Ong, T. (Natl. Inst. Occupational Safety and Health, ALOSH, Morgantown, WV 26505); Slade, B.; de Serres, F. J. *J Environ Pathol Toxicol* 2(4): 1109-1118; 1979.

The mutagenicity and mutagenic specificity of niridazole (NA) and metronidazole, (MNA) two agents used in the treatment of human parasitic diseases, were studied with the adenine-3 test system of *Neurospora crassa*. The results showed that 0.2 mg/ml NA for 4 hr or 8.8 mg/ml MNA for 24 hr was not mutagenic in resting conidia. In growing vegetative cells, however, both compounds were mutagenic in *Neurospora crassa*. Genetic analysis of the mutants indicated that NA induced predominantly base-pair substitution mutations. None of the ND-induced mutants resulted from multilocus deletions. The spectra of genetic alterations induced by MNA were similar to those induced by the monofunctional alkylating agents ethyleneimine, ethyl methanesulfonate, and ICR-177. It is suggested, therefore, that the mechanism of mutation induction by MNA in *Neurospora* is similar to that of monofunctional alkylating agents. (27 refs)

79-3688 The Anaerobic Metabolism of Metronidazole Forms N-(2-Hydroxyethyl)-oxamic Acid. (Eng) Koch, R. L. (Dept. Pharmacology, Harvard Medical Sch., Beth Israel Hosp., 330 Brookline Ave., Boston, MA 02215); Goldman, P. *J Pharmacol Exp Ther* 208(3): 406-410; 1979.

The formation of a previously undetected metabolite of

metronidazole by rat cecal contents and in male Sprague-Dawley rats following metronidazole administration (200 mg/kg by gavage) is reported. When incubated with rat cecal contents, metronidazole yielded four metabolites. Compound A was identified as N-(2-hydroxyethyl)oxamic acid (HEOA); the other three compounds were not identified. When compound A was incubated with the cecal contents, it was recovered completely 6 hr later; thus, there is no evidence that the unidentified metabolites are derived from compound A. After administration of radiolabeled metronidazole to rats in vivo, between 0.1% and 3.0% of the administered radioactivity was found in the urine as HEOA. The amount of HEOA was increased somewhat after treatment with β -glucuronidase. Neither free nor conjugated HEOA was found in the urine of germfree rats or in the feces of conventional or germfree rats. The HEOA appeared to form in the conventional rats as a result of the activity of the intestinal microflora. (22 refs)

79-3689 Alkylating Activity in the Extract and Pyrolyzate of Tobacco Leaves Varying in Genotype and Chemical Treatment. (Eng) Crosthwaite, L. (Dept. Plant Pathology, Univ. Kentucky, Lexington, KY); Sheen, S. J.; Burton, H. R. *Tob Int* 181(7): 110-112; 1979.

The results of spectrophotometric studies of the alkylating activity in the extract and pyrolyzate of air-cured leaves of tobacco cultivars and lines suggested that aromatic nitrogenous compounds are good precursors of alkylating compounds in the pyrolyzate. Genetic control of alkylating activity in tobacco leaf is feasible but much less effective than leaf modification by solvent treatment. (18 refs)

79-3690 Acute Myeloid Leukemia as a Late Complication of Hodgkin's Disease. (Ger) Ho, A. D. (Medizinische Poliklinik der Universität, Hospitalstrasse 3, D-6900 Heidelberg 1, W. Germany); Hunstein, W.; Uhl, N. *Munch Med Wochenschr* 121(15): 521-522; 1979.

A 45-yr-old man treated by radiotherapy, vinblastine, and cyclophosphamide for Hodgkin's disease developed acute myeloid leukemia (AML) 3 yr later. A 54-yr-old man with Hodgkin's disease who received Trenimon (triaziquone) developed AML about 10 yr later. It is suggested that the drugs were involved in the development of the AML. (45 refs)

79-3691 Therapy-Linked Leukemia: A Case Report. (Eng) Shetty, M. R. (Northwest Community Hosp., 800 W. Central Road, Arlington Heights, IL 60005); Freel, R. *Gynecol Oncol* 7(2): 264-266; 1979.

A 53-yr-old woman developed acute myelomonocytic

leukemia following melphalan and radiation therapy for ovarian cancer. The ovarian tumor was a moderately to poorly differentiated papillary serous cystadenocarcinoma that was discovered approx 2 yr previously. After surgery, the patient received whole-abdomen radiation (3,800 rads) plus a pelvic boost (5,820 rads). A bone marrow biopsy revealed evolving myelomonocytic leukemia after 15 mo of melphalan chemotherapy (total, 310 mg). The main features of the marrow were a marked left shift, including a significant increase in granulocytic blast forms, many of which had a somewhat monocytoid appearance, together with marked hyperplasia of the erythroid series, which showed a megaloblastoid appearance. The peripheral blood showed an occasional blast form. This case implicates melphalan as the leukemogenic agent; whether the radiation therapy contributed to leukemogenesis is difficult to establish. It is concluded that there is need for caution in developing chemotherapy protocols involving alkylating agents in patients who are candidates for adjuvant chemotherapy. (22 refs)

- 79-3692** Direct Urinary Assay Method for N¹-Methylnicotinamide by Soap Chromatography. (Eng) Shaikh, B. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701); Pontzer, N. J. *J Chromatogr* 162(4): 596-600; 1979.

A rapid method for N¹-methylnicotinamide quantitation in rat and human urine is described. The method involves the direct injection of urine onto a reversed-phase high-performance liquid chromatography column operating in the soap chromatography mode. The system employs a reversed-phase packing in combination with a hydrophilic eluent containing methanol as an organic modifier and a small concentration of a detergent that forms an ion pair with an ionized form of the solute. (7 refs)

- 79-3693** "Low-Risk" Cigarettes: The Debate Continues (2 Letters to Editor). (Eng) Gart, J. J. (3406 Kenilworth Drive, Chevy Chase, MD 20015); Schneiderman, M. A.; Gori, G. B. *Science* 204(4394): 688-692; 1979.

The statistical methodology used in two recent articles on "low-risk" cigarettes is criticized. Some of the methodologic errors made in the handling of data relating the relative risks of cancer of the lung and bronchus to the number of cigarettes smoked per day are detailed. In reply, the author of the original articles states that the articles presented a realistic procedure for gradually reducing the levels of hazard and addiction for smokers who persist in their habit despite its known health consequences. Although this would not eliminate the risk to the smoker, it has the potential of reducing the current epidemic of diseases associated with smoking. (6 refs)

- 79-3694** The First Published Chemical Analyses of Smoke from South African Cigarettes. (Eng) Seftel, H. C. (Dept. Medicine, Johannesburg General Hosp., Witwatersrand, Johannesburg, South Africa). *S Afr Med J* 55(19): 743-748; 1979.

The smoke from 69 brands of cigarettes sold in South Africa in March 1978 was analyzed for tar, nicotine, carbon monoxide, and carbon dioxide levels. The av amount of tar delivered per cigarette varied from 12.3 to 38.5 mg (mean, 28.7 mg), and the nicotine yield per cigarette varied between 0.52 and 2.40 mg (mean, 1.65 mg). In general, the nicotine yield paralleled that of tar, but in 15 brands, the nicotine:tar ratio was 0.05 or less. The yield of CO varied between 12.4 and 57.7 mg/cigarette (mean, 23.1 mg), and that of CO₂ varied from 46.4 to 95.3 mg/cigarette (mean, 70.1 mg). The yield of CO correlated well with that of CO₂, but the yield of these gases did not correlate well with that of tar. Compared with cigarette brands made and sold in the US, the tar and nicotine yields of the same brands sold in South Africa were higher. Similar results were found when the South African cigarettes were compared with those sold in England, Australia, and West Germany. It is recommended that: the State Health Department arrange for the regular chemical analysis of all tobacco products in South Africa; the results and their meaning be widely publicized; the cigarette manufacturers be required to make public the results of all previous chemical analysis; and the yields of tar, nicotine, and CO be clearly stated on every pack of cigarettes and in every cigarette advertisement. (14 refs)

- 79-3695** Nicotine and Cotinine in Breast Fluid. (Eng) Hill, P. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595); Wynder, E. L. *Cancer Lett* 6(4): 251-254; 1979.

Breast fluid aspirated from the nipples of 13 healthy, menstruating women (5 smokers and 8 nonsmokers) was assayed for nicotine and its major metabolite cotinine. With the smokers, a blood sample was taken and breast fluid aspirated 30 min after smoking. Neither nicotine nor cotinine could be detected in the plasma or breast fluid of nonsmokers, but in smokers both were detected in the breast fluid 30 min after smoking. The similarity of the nicotine levels in the breast fluid and plasma suggests that nicotine passes rapidly into the fluid but cotinine passes less readily. Further analysis of the breast fluid in various female populations may provide leads as to whether components are present that may be related to breast carcinogenesis. Breast fluid may contain many or all factors that contribute to the induction of breast cancer. (9 refs)

- 79-3696** Analysis of Food for Mycotoxins. (Fre) Fremy, J. M. (Laboratoire Central d'Hygiene

Alimentaire, Ministère de l'Agriculture, 43, rue de Dantzig, 75015 Paris, France); Corbion, B. *Ann Falsif Expert Chim* 72(772): 53-64; 1979.

Methods for the determination of mycotoxins in food are reviewed. The samples are extracted with acetonitrile, chloroform/water, or chloroform/methanol/water and then purified with hexane, isooctane, or diethyl ether or by precipitation with lead acetate. Thin-layer chromatography or gas-liquid chromatography is used for separation, and the mycotoxins are determined by visual comparison, fluorodensitometry, or UV spectrophotometry. (16 refs)

- 79-3697 **Comparative Mutagenicity of Palmotoxin Bo and Aflatoxins B₁ and M₁.** (Eng) Uwaifo, A. O. (Dept. Biochemistry, Univ. Ibadan, Ibadan, Nigeria); Emerole, G. O.; Bababunmi, E. A.; Bassir, O. *J Environ Pathol Toxicol* 2(4): 1099-1107; 1979.

The mutagenicities of palmotoxin Bo (PBo), aflatoxin M₁ (AFM), and aflatoxin B₁ (AFB) for *Salmonella typhimurium* strains TA98 and TA100 (containing R-factor plasmids), TA1535 (for base-pair substitution), and TA1537 and TA1538 (for frameshift mutation) were studied. PBo was isolated from chloroform extracts of cultures of *Aspergillus flavus* grown on palm sap, an alcoholic beverage regularly consumed in quantities of approx 2.5 liters/day by adult male Nigerians. Recent studies suggested that the structures of palmotoxins are similar to those of aflatoxins. At 1 µg, PBo increased the number of revertants in TA98, TA100, and TA1535. AFB and AFM increased the number of revertants in TA98, TA100, and TA1538. All mutagens required metabolic activation with S-9 rat liver microsomal mix. Over the concentration range 0.1-2.0 µg, the mutagenicity of AFB for TA100 was about 6 fold greater than that of PBo and about 10-fold greater than that of AFM. It is concluded that the mutagenic potential of a metabolite of a carcinogen may not necessarily be related to its carcinogenic potential, even when the dogma holds for the parent carcinogen. (27 refs)

- 79-3698 **Studies of Mycotoxins in Foods. XI. Analysis of Aflatoxins in Commercial Foods.** (Jpn) Naoi, Y. (Tokyo Metropolitan Res. Lab., Public Health, 24-1 Hyakunincho 3-chome, Shinjuku-ku, Tokyo, Japan); Nishijima, M.; Ogawa, H.; Saito, K.; Kan, K.; Kamimura, H.; Ibe, A.; Ochiai, S. *J Food Hyg Soc Jpn* 20(1): 54-58; 1979.

An analytical procedure involving column chromatography, thin-layer chromatography, fluorescence analysis using 365-nanometer UV light, and fluorodensitometry detected up to 465.0 ppb aflatoxin B₁ in Virginia peanuts shipped to Japan from the US. The formation of

molds during transport is thought to be the source of the aflatoxins. (5 refs)

- 79-3699 **Methods of Sorting Peanuts to Attain a Standard Quality.** (Ger) Hanssen, E. (H. Bahlsens Keksfabrik, Hannover, W. Germany); Birn, K. *Dtsch Lebensm Rundsch* 75(2): 43-49; 1979.

An electronic sorter that separates dark-colored (usually brown) peanuts from light ones and thus separates those with the higher aflatoxin (AF) levels is described. The device can sort approx 170 kg peanuts/hr. Tests showed that the AF level (sum of aflatoxins B₁, B₂, G₁ and G₂) of the sorted acceptable peanuts was reduced by 90% compared with unsorted peanuts. (4 refs)

- 79-3700 **Studies of Mycotoxins in Foods. IX. Analysis of the Natural Occurrence of Aflatoxins in Dairy Products.** (Jpn) Saito, K. (Tokyo Metropolitan Res. Lab. Public Health, 24-1, Hyakunincho 3-chome, Shinjuku-ku, Tokyo, Japan); Nishijima, M.; Kamimura, H.; Ibe, A.; Ochiai, S.; Naoi, Y. *J Food Hyg Soc Jpn* 20(1): 27-32; 1979.

An analytical procedure comprising column chromatography, thin-layer chromatography, and fluorodensitometry was used to determine aflatoxin levels in various dairy products. In spiked samples, av recoveries were 86% aflatoxin B₁, 99% B₂, 69% G₁, 89% G₂, and 102% M₁. Analysis of commercial samples of modified powdered milk (113 samples), powdered skim milk (16), raw cream (13), condensed milk (56), and cheese (83) revealed that all were negative for aflatoxins except for 17 cheese samples, which contained 0.2-1.3 ppb aflatoxin M₁. (16 refs)

- 79-3701 **gamma-Glutamyl Transferase as Oncofetal Marker of Experimental Hepatocarcinogenesis in the Rat.** (Eng) Kalengayi, M. M. (Dept. Pathology, Faculty Medicine, Natl. Univ. Zaire, Kinshasa, Zaire); Desmet, V. J. *Scand J Immunol [Suppl]* 8(8): 547-556; 1978.

The histochemical patterns of γ-glutamyltransferase (γ-GTase) in the rat liver during aflatoxin B₁ (AFB)-induced hepatocarcinogenesis were analyzed, along with features of this enzyme in rat and mouse transplantable hepatocarcinomas. γ-GTase is abundant in the fetal and neonatal rat liver, but it almost completely disappears in the normal adult rat liver. The AFB-induced hepatocarcinogenesis was divided into the early, preneoplastic, and neoplastic phases, during which combined histological-histochemical analyses were performed. Five types of

hepatocyte populations were encountered: (1) original hepatocytes; (2) small newly formed hepatocytes and transitional cells; (3) early (first wave) proliferating hepatocytes; (4) late (second wave) proliferating hepatocytes with the formation of altered foci and neoplastic nodules; and (5) malignant hepatocytes. Marked γ -GTase activity reappeared reversibly in populations 2 and 3, but it reappeared irreversibly in population 4 and in every hepatocellular carcinoma. The liver parenchyma (original hepatocytes) surrounding the other four populations showed no γ -GTase activity, similar to normal adult rat liver. The reemergence of γ -GTase in populations 2-5 indicates that the development of hepatocellular carcinoma involves the reacquisition of some embryonal biochemical characteristics. Furthermore, it illustrates the similar immature state of specific and focal preneoplastic and neoplastic liver cell lesions. In the transplantable hepatocarcinomas, γ -GTase activity was prominent in the carcinogen-induced tumor but absent in the spontaneous tumors. Histochemically γ -GTase is a sensitive marker of hepatocarcinogenesis. (29 refs)

- 79-3702 DNA Repair in Primary Monkey Kidney Cells after Exposure to Aflatoxin B₁ and Sterigmatocystin.** (Eng) Seegers, J. C. (Dept. Physiology, Medical Sch., Univ. Pretoria, Pretoria, S. Africa); Pitout, M. J.; Kempff, P. G. *S Afr J Sci* 75(1): 21-24; 1979.

The effects of aflatoxin B₁ (AFB) and sterigmatocystin (SC) on DNA repair in primary monkey kidney cells (PMKC) and on cell nuclear endonuclease activity were studied. PMKC exposed to AFB or SC showed a decrease in the numbers of nuclei that became heavily labeled with ³H-thymidine and an increase in the number of lightly labeled nuclei. It appeared that only cells exposed to toxin concentrations of ≤ 0.1 mg/liter for 24 hr or 0.5 mg/liter for 2 hr were in the process of DNA repair. PMKC exposed to 0.1 mg/liter SC for 2 hr, but not 0.01 mg/liter for 20 hr, showed marked inhibition of acid DNase activity; at 0.01 mg/liter for 2 or 20 hr, there was a 30% increase in alkaline DNase activity. With AFB, 10 mg/liter for 2 or 24 hr, but not 0.1 mg/liter, markedly decreased acid DNase activity; however, 0.1 mg/liter for 2 or 24 hr significantly increased alkaline DNase activity. At 0.5 mg/liter AFB, no DNA strand breaks were observed after 10 min, whereas a broad spectrum of nucleotides was detected after 2 or 24 hr. In cells exposed to toxin for 2 hr followed by a 24-hr recovery period, no strand breaks were noticed. It is concluded that PMKC enter the mitotic cycle before the toxin-induced DNA damage is repaired completely. The data also indicate that alkaline DNase may be associated with the DNA repair mechanism. (22 refs)

- 79-3703 Gastrointestinal Carcinogenesis in Germ-free Rats Given M-Methyl-N'-nitro-N-nitrosoguanidine in Drinking Water.** (Eng) Sumi, Y. (Lab.

Germfree Life Res., Nagoya Univ. Sch. Medicine, Tsumurai-cho, Showa-ku, Nagoya 466, Japan); Miyakawa, M. *Cancer Res* 39(7): 2733-2736; 1979.

To clarify the role of gut microflora in tumorigenesis, the carcinogenic action of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG, 100 μ g/ml in the drinking water) was studied in male germ-free (GF) and conventional Wistar (CW) rats. Gastrointestinal (GI) tumors developed in 17% of the GF rats and 91% of the CW rats. Adenocarcinomas occurred in 1/30 GF rats and 14/33 CW rats; more anaplastic cells and mitoses were found in the tumors of the CW rats than in those of the GF rats. Leiomyosarcomas were found in 1/30 GF rats and 11/33 CW rats; the tumor cells of the CW rats demonstrated more nuclear and cytoplasmic polymorphism than those of the GF rats. Hemangioendotheliomas developed in 2/30 GF rats and 4/33 CW rats; there were no differences in tumor morphology between the two groups of rats. A carcinosarcoma developed in one CW rat, and adenomas were found in one GF rat and six CW rats. In addition, one hepatocellular carcinoma and two hepatocellular adenomas were observed in CW rats. Multiple tumors were observed in 8/33 CW rats and 0/30 GF rats. The data indicate that the gut microflora might have exercised a promoting effect on GI tumorigenesis in the MNNG-treated animals. (15 refs)

- 79-3704 Mutagenesis of Chinese Hamster Cells Is Facilitated by Thymidine and Deoxycytidine (Letter to Editor).** (Eng) Saffhill, R. (Paterson Labs., Christie Hosp. and Holt Radium Inst., Manchester, England); Abbott, P. J. *Nature* 278(5704): 581; 1979.

Evidence that modification of the mutagenic potential of N-methyl-N'-nitro-N-nitrosoguanidine methylation by the presence of thymidine and/or deoxycytidine in Chinese hamster cell cultures may be partly due to competitive miscoding by O⁶-methylguanine is presented. This competitive miscoding leads to the possibility of an alteration in the miscoding level by factors that influence the size of the deoxynucleoside 5'-triphosphate DNA-precursor pools in the cell. Thus, agents that alter the size of these pools may enhance or reduce the mutagenic potential of alkylating agents. (4 refs)

- 79-3705 The Oncogenic Effect of Immunosuppressive (Cytotoxic) Agents in (NZB X NZW) Mice. II. Emergence of Tumors in Young Animals Treated with Azathioprine and Ifosfamide, Including a Histologic Assessment of the Neoplasms.** (Eng) Mitrou, P. S. (Abteilung für Hamatologie, Zentrum der Inneren Medizin, Johann-Wolfgang-Goethe-Universität, Theodor-

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Stern-Kai 7, 6,000 Frankfurt am Main 70, W. Germany); Fischer, M.; Mitrou, G.; Rottger, P. *Arzneim Forsch* 29, 1(4): 662-667; 1979.

The possible correlation between age at the start of treatment with cytotoxic agents in (NZB X NZW) mice and tumor development was investigated. Azathioprine (ATP) or isophosphamide (IP) (0.2 mg sc 5x/wk for 7 or 8 mo) were administered to mice aged 6, 7, 8, or 12 wk. In the ATP-treated groups, 10/39 animals developed tumors. Tumors occurred among each of the four age groups. Only one death due to malignant lymphoma was associated with IP treatment; no additional tumors were discovered at the end of the study. The incidence of tumors in the IP-treated groups did not differ significantly from that in controls. The lesions observed in the tumor-bearing animals were mainly lymphomas, and they could be classified according to two histologic types: Type I, in which cells varied somewhat in size but displayed a uniform morphology, and Type II, which contained pleomorphic cells with round, oval, or irregularly shaped pale nuclei and prominent nucleoli. The lymphoma in the IP group could not be distinguished histologically from those in the ATP groups. These results indicate that there is no correlation between age at the start of treatment and tumor incidence. It is also suggested that immunosuppression alone cannot explain the high rate of malignant lymphomas observed in mice treated with cytotoxic agents. (17 refs)

79-3706 Morphological Studies of Persistent Nucleoli in Cultured Cells. (Jpn) Hattanmaru, K. (First Dept. Surgery, Kagoshima Univ. Sch. Medicine, Kagoshima, Japan). *Igaku Kenkyu* 48(8): 639-651; 1978.

The effect of 4-nitroquinoline 1-oxide (NQO: 10^{-8} M for 24-96 hr) and several of its carcinogenic and non-carcinogenic derivatives on the number and morphology of persistent nucleoli (PN) in a Yoshida ascites sarcoma clonal cell line was investigated. PN (cytoplasmic remnants of nucleoli that do not disintegrate during mitosis) occur naturally in 3%-14% of cultured Yoshida cells. NQO treatment increased the number of cells with PN to a max of 70.5% at 24 hr; this was followed by a decrease to 63% at 30 hr, 50% at 36 hr, 38% at 48 hr, and control levels at 72 hr. This treatment also increased the size of the nucleoli. The PN were seen least often during anaphase and metaphase. The percentage of cells with PN was higher with the carcinogenic NQO derivatives than with the non-carcinogenic derivatives, the latter producing values in the control range. Electron microscopically, the PN in the NQO-treated cells were shown to be of two types: a free body in the cytoplasm with a pars amorphous structure or a body attached to the chromosomes with the structure of a nucleolonema, suggesting possible ribosomal RNA synthesis. (28 refs)

79-3707 Metabolic Activation of Nitrofurantoin--Possible Implications for Carcinogenesis.

(Eng) Boyd, M. R. (Clinical Pharmacology Branch, NCI, NIH, Bethesda, MD 20014); Stiko, A. W.; Sasame, H. A. *Biochem Pharmacol* 28(5): 601-606; 1979.

The present investigation was undertaken to determine whether nitrofurantoin (NF), like other furans and nitrofurans, is enzymatically converted either aerobically or anaerobically to metabolites sufficiently reactive to alkylate tissue macromolecules. Microsomal and soluble fractions from HLA-SD rat liver and lung mediated the covalent binding of [14 C]NF to tissue macromolecules in vitro. Oxygen strongly inhibited the binding in both the microsomal and soluble fractions, and carbon monoxide failed to inhibit binding in microsomal preparations, indicating that NF was activated in both systems by nitroreduction (NR) and not by oxidation of the furan ring. An antibody against NADPH-cytochrome c reductase inhibited the microsomal NR and covalent binding of NF, but the addition of a flavin (FAD) markedly enhanced covalent binding. Max rates of covalent binding were obtained in soluble fractions in the presence of NADH or hypoxanthine; covalent binding was inhibited in these fractions by allopurinol, an inhibitor of xanthine oxidase. The NR of NF was enhanced but covalent binding was decreased in liver microsomes from phenobarbital-pretreated rats. Phenobarbital did not alter the NR or covalent binding of NF in lung microsomes or in soluble fractions from lung or liver. Reduced glutathione markedly decreased the covalent binding of NF in both the microsomal and the soluble fractions from liver and lung, but it did not alter the rate of NR in any of the fractions. Radioactivity was covalently bound in several organs of rats given [14 C]NF in vivo. Although past investigations have indicated that NF is non-carcinogenic, the present studies demonstrate several features of the metabolism of the drug that are similar to those of other nitrofurans that are known carcinogens. (45 refs)

79-3708 Synthesis and Chemical Carcinogen Inhibitory Activity of 2-tert-Butyl-4-hydroxyanisole.

(Eng) Lam, L. K. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455); Pai, R. P.; Wattenberg, L. W. *J Med Chem* 22(5): 569-571; 1979.

2-tert-Butyl-4-hydroxyanisole (isomer 1), an isomer of butylated hydroxyanisole (BHA), was selectively synthesized in pure isomeric form by means of the hydroxyl-protecting reagent dimethyl-tert-butylchlorosilane. Both isomer 1 and 3-tert-butyl-4-hydroxyanisole (isomer 2), the other isomer found in commercially available BHA, were tested for their inhibitory effects toward benzo(a)pyrene (BP)-induced neoplasia in the forestomach of ICR/Ha mice. BP (1 mg/0.2 ml cottonseed oil) was administered by po intubation 2x/wk for a total of eight doses. Addition of isomers 1 and 2 to the diet (5 mg/g starting 1 wk prior to the first BP dose and ending 2 days after the last BP dose) reduced the number of mice with tumors (30% and 10%

decrease, respectively) and the number of tumors per mouse (70% and 50% decrease, respectively). Isomer 1, which has a less-hindered free hydroxyl group, showed a higher inhibitory effect. The protection afforded by the BHA isomers is most likely due to an alteration of the enzyme systems responsible for the biotransformation of BP. (14 refs)

- 79-3709 Studies on Metabolism and Toxicity of Styrene. II. Mutagenesis in *Salmonella typhimurium* by Metabolic Activation of Styrene with 3-Methylcholanthrene Pretreated Rat Liver. (Eng) Watabe, T. (Lab. Drug Metabolism and Toxicology, Dept. Hygienic Chemistry, Tokyo Coll. Pharmacy, Horinouchi, Hachiojishi, Tokyo 192-03, Japan); Isobe, M.; Yoshikawa, K.; Takabatake, E. *J Pharmacobio Dyn* 1(5): 301-309; 1978.

The mutagenicity of styrene to *Salmonella typhimurium* in the presence of 9,000 x g supernatant fractions (S9) obtained from Wistar rats pretreated with various inducers was evaluated. Styrene showed a weak mutagenic activity toward *S. typhimurium* strain TA100 after being activated by the S9 fraction obtained from rats pretreated with 3-methylcholanthrene (3-MC) or phenobarbital (PB) and fortified with an NADPH-generating system, in the presence of the epoxide hydratase (EH) inhibitor 3,3,3-trichloropropene oxide (TCPO). The 3-MC-pretreated rat liver S9 mix activated styrene more effectively than the PB-pretreated one. No mutagenic activity was observed when TCPO or the S9 mix was omitted. Phenylloxirane, an intermediate in the major metabolic pathway of styrene by hepatic microsomes, induced mutations in TA100 and TA1535 cells in the absence of both the S9 mix and TCPO. Analytical data for phenylloxirane and its hydrolytic product 1-phenyl-1,2-ethanediol indicated that the inducers used for the hepatic drug-metabolizing enzymes enhanced both microsomal monooxygenase and EH activities. Relative ratios of the enhanced monooxygenase to hydratase activities were twice as high with S9 mix from 3-MC-pretreated rats as with control preparations and 1.8 and 1.2 times in S9 mixes from PC- and PCB-pretreated rats, respectively, although the last S9 mix enhanced both activities to the highest extent. Accumulation of phenylloxirane in the presence of TCPO in the styrene-activating system was most significant when 3-MC-pretreated rat liver S9 mix was used. However, the mutagenicity due to the metabolic activation of styrene could not be explained only by phenylloxirane, since sums of both metabolites were smaller than the amount of epoxide required to induce mutations in *Salmonella*. There may be at least one more unknown mutagenic metabolite with an epoxide structure. (28 refs)

- 79-3710 Induction of Unscheduled DNA Synthesis in Suspensions of Rat Hepatocytes by an Environmental Toxicant, 3,3'-4,4'-Tetrachloroazobenzene.

(Eng) Hsia, M. T. (Dept. Entomology, 237 Russell Labs., Univ. Wisconsin, 1630 Linden Drive, Madison, WI 53706); Kreamer, B. L. *Cancer Lett* 6(4): 207-212; 1979.

A rapid method to measure DNA repair in suspended rat hepatocytes is described. Suspensions of freshly isolated hepatocytes from 2-mo-old male Sprague-Dawley rats were exposed to a soln of tetrachloroazobenzene (TCAB), benzo(a)pyrene (BP), or methyl methanesulfonate (MMS) in dimethyl sulfoxide for 1 hr. The induction of unscheduled DNA synthesis (UDS) was measured by the incorporation of ³H-thymidine in the presence of hydroxyurea, as determined by scintillation counting. The extent of UDS in the hepatocyte suspensions was compared following three methods of DNA isolation. When the intact hepatocytes were treated directly with trichloroacetic acid (TCA) after incubation with ³H-thymidine, only 19% of UDS above control values was seen with BP. When hepatocellular DNA was isolated by TCA precipitation after lysis of nuclei, UDS increased to 62% of control values for the same concentration of BP (10⁻⁵). When chloroform-isoamyl alcohol-phenol extraction of nuclear lysates was used, UDS rose to 213% of control values. A dose-dependent response of UDS was observed with the three compounds tested. It is concluded that this DNA extraction/scintillation counting technique represents a rapid and sensitive screen for chemical carcinogens. The results also indicate that TCAB is a potential carcinogen. (12 refs)

- 79-3711 Singlet Oxygen Reacts with Inhibitors of Ultraviolet Mediated Damage to Skin: p-Aminobenzoic Acid and Its Derivatives. (Eng) Bodaness, R. S. (Dept. Biochemistry, State Univ. New York, Downstate Medical Center, Brooklyn, NY 11203); Chan, P. C. *Biochem Biophys Res Commun* 87(4): 1116-1123; 1979.

The reaction of p-aminobenzoate (PABA) and some of its derivatives with singlet oxygen (¹O₂) was studied. The absorption spectrum of p-dimethylaminobenzoate (dimethyl-PABA) in a singlet oxygen-generating system changed steadily with increasing length of UV irradiation, indicating a hematoporphyrin (HP)-catalyzed photoreaction of dimethyl-PABA. At first, the loss of dimethyl-PABA was accompanied by the appearance of product(s); however, after prolonged irradiation (28 min), absorbance measurements indicated that the product(s) may also have been destroyed by ¹O₂. Methionine, a scavenger of ¹O₂, effectively inhibited the HP-catalyzed photooxidation of PABA and its derivatives. The hydroxyl radical scavengers mannitol, benzoate, formate, and isopropanol had no significant effects on the photooxidation. The results are consistent with a single oxygen-mediated oxidation of PABA and its derivatives in this system. (24 refs)

- 79-3712 Correlation of Calculated Electronic Parameters of Fifteen Aniline Derivatives with

Their Mutagenic Potencies. (Eng) Loew, G. H. (Dept. Genetics, Stanford Univ. Medical Center, Stanford, CA 94035); Sudhindra, B. S.; Walker, J. M.; Sigman, C. C.; Johnson, H. L. *J Environ Pathol Toxicol* 2(4): 1069-1078; 1979.

Electronic parameters for a series of 15 amino-, chloro-, and nitro-substituted aniline compounds relative to their potential for activation to hydroxylamines, arylnitrenium ions, and ring epoxides and to their potential deactivation to phenols were calculated using semiempirical molecular orbital methods. The relative mutagenicities of aminoanilines could be explained by parameters reflecting potential for N-hydroxylation, as indicated by N-superdelocalizabilities (N-SDL's), and the stability of the arylnitrenium ions. Based on calculations of the N-SDL's of the amine nitrogens on the three chloroaniline isomers compared with those of aniline, little or no activity due to N-hydroxylation would be expected. In addition, although each isomer has a ring bond that would be reactive toward epoxidation, rearrangement to a phenol due to skewed SDL's of the C atoms about the reactive ring bond, and, thus, potential deactivation would be predicted. All of the nitro-substituted compounds would be expected to be inactive if their mechanism of action proceeded through N-hydroxylation, since the amine N-SDL's of these compounds are lower than that of aniline. In addition, all of the nitro compounds would be expected to undergo deactivation by ring metabolism. Although each compound has one or more ring bonds that would be moderately susceptible to epoxidation, the epoxide would be expected to rearrange to a phenol, since the S of the C atoms about the reactive ring bond are moderately to highly skewed. Thus, no active products from cytochrome P-450 would be predicted for chloro- and nitro-substituted anilines. This result is consistent with the lack of mutagenic activity observed for chloro derivatives, but it does not account for the activity of the nitro derivatives, which is presumed to be due to transformation of the nitro group itself into an active mutagenic species by other enzyme systems. (12 refs)

79-3713 A Role for Liver Glutathione in the Hepatobiliary Fate of N,N-Dimethyl-4-aminoazobenzene. (Eng) Levine, W. G. (Dept. Molecular Pharmacology, Albert Einstein Coll. Medicine, Yeshiva Univ., Bronx, NY 10461); Finkelstein, T. T. *J Pharmacol Exp Ther* 208(3): 399-405; 1979.

The metabolic control of the biliary excretion of N,N-dimethyl-4-aminoazobenzene (DAB) by male Wistar rats was studied. Following iv injection of 1 or 10 mg/kg DAB, glucuronide and sulfate conjugates of the products of N-demethylation, 4'-hydroxylation, and N-acetylation were rapidly excreted in the bile. After ip injection of doses of diethyl maleate (DEM) or methyl iodide (MeI) that depleted liver glutathione (GSH) levels by >90%, the rate of biliary excretion of DAB metabolites was inhibited

following administration of DAB but not administration of its metabolites. Concurrent administration of cysteine reversed the depletion of GSH and inhibition of biliary excretion. Quantitative examination of the DAB metabolites excreted in the bile as well as those formed in vitro in the presence of a 10,000 x g supernatant fraction of liver showed that depletion of liver GSH was associated with depression of N-demethylation but not of 4'-hydroxylation of DAB. When the hydroxylation step was bypassed through injection of [¹⁴C]4'-OH-DAB, inhibition of biliary excretion was still seen in GSH-depleted animals. However, when N-demethylation was bypassed by injection of [¹⁴C]aminoazobenzene, the rate of biliary excretion of metabolites was not affected by GSH depletion. (40 refs)

79-3714 In Vitro Effect of L-Tryptophan and Its Metabolites on Dimethylaminoazobenzene Reductase Activity of Rat Liver. (Eng) Mostafa, M. H. (Medical Res. Inst., Univ. Alexandria, Alexandria, Egypt); Evarts, R. P.; Weisburger, E. K. *Biochem Pharmacol* 28(6): 815-819; 1979.

The effects of o-aminophenol (AP) and of L-tryptophan (TP) and its metabolites L-kynurenine, anthranilic acid, kynurenic acid, quinaldic acid, 3-hydroxy-DL-kynurenine, 3-hydroxyanthranilic acid (HAA), xanthurenic acid, quinolinic acid, N-methylnicotinamide, and N'-methylnicotinamide on rat liver azoreductase activity were determined, using the hepatocarcinogen 4-dimethylaminoazobenzene (DAB) as substrate. Only HAA, 3-hydroxykynurenine, and AP decreased enzyme activity. The inhibition was greater if the buffered solns (pH 7.4) of these three compounds were kept overnight before use, but the effect was prevented if these compounds were prepared in solns of L-ascorbic acid and/or L-cysteine HCl. This observation indicates that autoxidation products were probably responsible for inhibition of the enzyme. Further study of the oxidation products, including the phenylquinoneimine formed from the oxidation of HAA in air, cinnabaric acid, xanthommatin, 2-amino-3H-isophenoxazin-3-one, 1,9-dimethyl-2-amino-3H-phenoxazin-3-one, and actinomycin D, showed that all these compounds inhibited enzyme activity. A noncompetitive type of inhibition was observed in the presence of cinnabaric acid and xanthommatin. Cinnabaric acid, xanthommatin, 2-amino-3H-isophenoxazin-3-one, 1,9-dimethyl-2-amino-3H-phenoxazin-3-one, and actinomycin D all have the same phenoxazinone ring system, suggesting that the driving factor in azoreductase inhibition is the presence of the phenoxazinone chromophore. The chemical resemblance of these phenoxazinones to the coenzyme riboflavine further supports this supposition. (34 refs)

79-3715 Levels of Polychlorinated Biphenyls (PCBs) and Organochlorine Pesticides in Human Milk

and Blood Collected in Osaka Prefecture from 1972 to 1977. (Eng) Yakushiji, T. (Osaka Prefectural Inst. Public Health, 3-69 1-Chome, Nakamichi, Higashinari-ku, Osaka 537, Japan); Watanabe, I.; Kuwabara, K.; Yoshida, S.; Koyama, K.; Kunita, N. *Int Arch Occup Environ Health* 43(1): 1-15; 1979.

Levels of polychlorinated biphenyls (PCB's) and other organochlorine pesticides were determined in the milk and blood of lactating women in Osaka Prefecture between 1972 and 1977. The number of milk samples analyzed ranged from 100 to 141/yr, the number of blood samples from 100 to 129/yr. The av PCB levels in milk were 0.03-0.04 ppm, 10 times higher than those in the blood. However, the PCB levels in 15 milk samples exceeded 0.1 ppm, the highest level being 0.24 ppm. PCB levels were higher in the milk and blood of mothers with only one child at the time of the study than in those who had given birth one or more times before the study (for 1974, $p < 0.01$; for 1976, $p < 0.001$). The milk levels of the other pesticides studied were proportional to the PCB levels, suggesting that these compounds are ingested in the same food. Analysis of the residual PCB components and their chlorobiphenyl contents in milk samples showed that there were large amounts of tri- and tetrachlorobiphenyls. None of the lactating women or their babies showed any symptoms related to PCB toxicity. However, further studies of PCB toxicity in infants should be carried out. (38 refs)

79-3716 Determination of Benzidine and Its Acetylated Metabolites in Urine by Liquid Chromatography. (Eng) Rice, J. R. (Bioanalytical Lab., Dept. Chemistry, Purdue Univ., West Lafayette, IN 47907); Kissinger, P. T. *J Anal Toxicol* 3(2): 64-66; 1979.

A method for the analysis of benzidine (BD; 4,4'-diaminobiphenyl) and its acetylated metabolites in urine involves extraction of the compound(s) followed by quantitation via liquid chromatography with electrochemical detection. BD gave a linear calibration curve over the range 25 nanograms (ng)/ml to 5 μ g/ml ($r = 0.990$), with a precision of $\pm 0.06\%$ relative standard deviation at 250 ng/ml. (14 refs)

79-3717 Carcinogenicity Testing of Herbicide 2,4,5-Trichlorophenoxyethanol Containing Dioxin and of Pure Dioxin in Swiss Mice. (Eng) Toth, K. (Res. Inst. Oncopathology, Rath Gyorgy 7-9, H-1122 Budapest, Hungary); Somfai-Relle, S.; Sugar, J.; Bence, J. *Nature* 278(5704): 548-549; 1979.

The carcinogenic effects of 2,4,5-trichlorophenoxyethanol (TCPE) contaminated with dioxin (DO) and of DO alone (both compounds administered weekly by gastric tube for 1 yr) on male and female Swiss mice were studied. The LD₅₀ of an acute po dose of TCPE containing 0.1 ppm DO was

1,320 mg/kg, and the max tolerated dose was 70 mg/kg. The only difference in tumor incidence between the treatment groups and controls was in that of liver tumors in male mice. Liver tumor incidence in Groups 1 (67 mg/kg TCPE + 0.112 μ g/kg DO), 2 (70 mg/kg TCPE + 0.007 μ g/kg DO), and 10 (0.7 μ g/kg DO) was twice that in the respective control groups (vehicle alone). Liver tumor incidence in Group 5 (7 mg/kg TCPE + 0.0007 μ g/kg DO) and Group 6 (0.7 mg/kg TCPE + 0.00007 μ g/kg DO) did not differ from that in the respective control groups. There were no significant differences in the frequency of liver tumors between Groups 1 and 2 or Groups 4 (7 mg/kg TCPE + 0.07 μ g/kg DO) and 5. There was also no significant difference in liver tumor incidence between Groups 2 and 11 (0.007 μ g/kg DO), although they received the same amount of DO. The incidence in Group 10 was twice that in control Group 12. Spontaneous and induced liver tumors were not histologically different, and the ratio of benign hepatomas to hepatocellular carcinomas was not affected by treatment. Av life-span was decreased considerably in Group 9, which received the highest dose of DO (7 μ g/kg). DO contamination and exposure to TCPE during its production and use should be controlled and reduced. (22 refs)

79-3718 Mutagenicity Test with Saccharin in the Male Mouse. (Eng) Leonard, A. (Mammalian Genetics Lab., Dept. Radiobiology, C.E.N.-S.C.K., B-2400 MOL, Belgium); Leonard, E. D. *J Environ Pathol Toxicol* 2(4): 1047-1053; 1979.

The ability of saccharin to induce chromosome aberrations in male C57BL mice was tested after acute (1, 2, or 4 g/kg ip) or chronic (20 g/liter in drinking water for 100 days) treatment. All in vivo tests on mice of both treatment groups were negative. The incidence of chromosome aberrations in the bone marrow cells was not increased, $<4\%$ RBC with micronuclei were found in treated and control mice, and there were no demonstrable effects on somatic or germ cells. In the acute study, 44% of females mated with control males and 27% of those mated with saccharin-treated males became pregnant during the first week ($p < 0.001$); this difference was attributed to stress resulting from saccharin injection. The total number of dead embryos per pregnant female was significantly lower in the second- and third-week mating groups treated with saccharin than in the controls; the reason for this is not known. In the chronic experiment, the incidence of pregnancy among females mated to treated males was significantly greater ($p < 0.01$) than that among the females mated to controls. It is concluded that the positive findings reported in the literature were probably due to the mutagenic activity of saccharin impurities. (17 refs)

79-3719 Activation and Induction of Rat Liver Microsomal UDP-Glucuronyltransferase with

3-Hydroxybenzo(a)pyrene and N-Hydroxy-2-Naphthylamine as Substrates. (Eng) Bock, K. W. (Dept. Pharmacology and Toxicology, The University, D-3400 Gottingen, W. Germany); Lilienblum, W. *Biochem Pharmacol* 28(5): 695-700; 1979.

To evaluate the importance of conjugation in eliminating phenols of polycyclic hydrocarbons and N-hydroxyarylamines, the glucuronidation of 3-hydroxybenzo(a)pyrene (BP) and N-hydroxy-2-naphthylamine (NA) by rat liver microsomes was studied. Glucuronidation of the two toxic intermediates was markedly activated by uridine diphosphate-N-acetylglucosamine, which probably acts as a physiological activator of glucuronyltransferase (GT). Full activation was achieved by addition of a nonionic detergent. GT activity toward the two reactive intermediates was markedly enhanced by pretreatment of the rats with 3-methylcholanthrene (3-MC: 40 mg/kg, ip). It is likely that BP and NA are conjugated by the 3-MC-inducible form of GT. (20 refs)

79-3720 A Carcinogenicity Study of the Pesticide Dieldrin in Hamsters. (Eng) Cabral, J. R. (Toxicology Unit, Medical Res. Council Labs., Woodmansterne Road, Carshalton, Surrey, England); Hall, R. K.; Bronczyk, S. A.; Shubik, P. *Cancer Lett* 6(4): 241-246; 1979.

Groups of Syrian hamsters were fed 0, 20, 60, or 180 ppm dieldrin for life, and the carcinogenic effects of this treatment were evaluated. The experiment lasted for 120 wk, when the last survivor was killed. The first tumors (thyroid adenocarcinoma and vertebral osteochondroma) were observed in one moribund female (180-ppm group) killed at 20 wk of age. In females, 5/39 controls developed tumors, vs 1/32, 5/34, and 5/38 animals that received 20, 60, and 180 ppm dieldrin, respectively. Three of 40 control males developed tumors, vs 5/32, 5/32, and 10/40 males that received 20, 60, and 180 ppm. Although the percentage of tumor-bearing animals was not significantly different between the treated and control groups, more treated animals had more than one tumor. There were 17 tumors, 11 adenomas and 6 carcinomas. The carcinomas appeared only in the treated groups. A hepatoma was detected in one female and in one male, each fed 180 ppm dieldrin; none was detected in controls. These results show that the hamster is resistant to the carcinogenic effects of dieldrin at doses considerably higher than those producing a high incidence of liver tumors in mice. (20 refs)

79-3721 Substituent Effects in Chemical Carcinogenesis: Methyl Derivatives of the Benzacridines. (Eng) Smith, I. A. (Dept. Chemistry and Biological Chemistry, Wright State Univ., Dayton, OH

45435); Seybold, P. G. *J Heterocycl Chem* 16(3): 421-425; 1979.

An extensive theoretical examination of the putative activation steps for the methyl derivatives of angular benzacridines was undertaken using reactivity indices taken from molecular orbit theory. All the known strong carcinogens showed values of I-R, a measure of the tendency for epoxidation to occur at the K region, of >2.10 and all weak and noncarcinogens showed values <2.10 . The weak and inactive compounds were not distinguished by I-K. The index I-A, which measures the tendency for epoxidation to occur at the A region, separated the known carcinogens ($IA \geq 1.841$) from the noncarcinogens ($IA \leq 1.840$), but it did not distinguish between strong and weak carcinogens. The correlation between the reactivity index $I=B'$, a measure of the tendency of the A-region dihydrodiols to undergo epoxidation at their B regions, and carcinogenicity was about that same as for I-K. The correlation of two carbonium ion indices, net charge density and superdelocalizability, carcinogenic potency also appeared to be rather good. The results suggest that benzacridines are activated to carcinogenic end products in a manner similar to that for benz(a)anthracene and other aromatic hydrocarbons and that epoxidation of the A-region dihydrodiols and the stability of the final carbonium ion may be important determinants of carcinogenicity. (23 refs)

79-3722 Dimethyl-10,12-benz(a)acridine: Evidence for Differential Effects on the Synthesis of RNA of Mammalian or Avian Fibroblasts and Some RNA Viruses. (Eng) Gamulin, V. (Dept. Organic Chemistry and Biochemistry, Rudjer Boskovic Inst., Ilica 197, 41000 Zagreb, Yugoslavia); La Regina Rodrigues, M. A.; Brdar, B. *Biochim Biophys Acta* 562(1): 139-148; 1979.

The effects of dimethyl-10,12-benz(a)acridine (DMBAcr) on the synthesis of the RNA of cultured chick or mouse fibroblasts and that of some RNA-containing viruses such as Rous sarcoma virus (RSV) and Mengo virus (MV) were compared. Even at low concentrations ($5 \mu\text{g/ml}$), DMBAcr blocked the growth of both normal and RSV-transformed chicken fibroblasts, the effect being greater in the transformed cells. This effect was reversible after short incubation periods. DMBAcr depressed cellular DNA and RNA synthesis in parallel, and the inhibition was 80%-90% at $20 \mu\text{g/ml}$. Protein synthesis was inhibited only about 40%-50%. The inhibitory effect on cellular RNA synthesis was due mostly to a block in the formation of 28S and 18S ribosomal RNA; in contrast, the synthesis of 45S ribosomal RNA precursor proceeded at almost the control rate. Heterogeneous nuclear RNA synthesis was not blocked by DMBAcr. The rate of RSV production was unaffected by incubation with $10 \mu\text{g/m}$ DMBAcr. The synthesis of MV RNA was strongly suppressed by 10 or $50 \mu\text{g/ml}$ DMBAcr. The results suggest that the synthesis of RSV RNA and that of messenger and heterogeneous nuclear

RNA share a similar specific resistance to DMBAcr. (19 refs)

- 79-3723 Rank Order of Sarcoma Susceptibility among Mouse Strains Reverses with Low Concentrations of Carcinogen.** (Eng) Prehn, L. M. (Jackson Lab., Bar Harbor, ME 04609); Lawler, E. M. *Science* 204(4390): 309-310; 1979.

Ten mouse strains in which aryl hydrocarbon hydroxylase can be induced or F¹ hybrids of these strains were ranked according to their sarcoma susceptibility when exposed to 5% 3-methylcholanthrene (3-MC). The most susceptible strain was C3H/HeJ and the least susceptible C57BL/6J. This rank order was reversed when the 3-MC concentration was reduced to 0.05%. (7 refs)

- 79-3724 Effects of Microsomal Enzyme Inducers on Carrier-mediated Transport Systems in Isolated Rat Hepatocytes.** (Eng) Eaton, D. L. (Dept. Pharmacology, Univ. Kansas Medical Center, Kansas City, KS 66103); Klaassen, C. D. *J Pharmacol Exp Ther* 208(3): 381-385; 1979.

Studies of the effects of phenobarbital, 3-methylcholanthrene, and pregnenolone-16 α -carbonitrile carbonitrile pretreatment on ouabain, procaine amide ethobromide, and taurocholate uptake by rat hepatocytes suggest that the effects of microsomal enzyme inducers on hepatic transport systems do not correlate with their ability to increase cytochrome P-450 or microsomal protein synthesis. Thus, isolated hepatocytes, which are not affected by alterations in blood and bile flow, may be used to study alterations in hepatic clearance mechanisms. (26 refs)

- 79-3725 The Early Effects of Chemical Carcinogens on Adult Rat Hepatocytes in Primary Culture: I. Quantitative Changes in Intracellular Enzyme Activities Following a Single Dose of Carcinogen.** (Eng) Lowing, R. K. (Health and Safety Executive, Cricklewood, London, England); Fry, J. R.; Jones, C. A.; Wiebkin, P.; King, L. J.; Bridges, J. W. *Chem Biol Interact* 24(2): 121-131; 1979.

The effects of exposure of adult Wistar rat hepatocytes to chemical carcinogens (3-methylcholanthrene, 2-acetylaminofluorene, 6-aminochrysene, or 3-methyldimethylaminoazobenzene, 0.1-100 μ M for 3 hr) were studied using a short-term maintenance culture system. Scanning microdensitometry was used to quantitate the observed changes in enzyme activity. The dose-response curves showed a biphasic response for all four en-

zymes studied (glucose-6-phosphate dehydrogenase, succinate dehydrogenase, NADPH oxidase, and γ -glutamyl transpeptidase), there being decreased enzyme activities at the higher dose levels used, possibly due to cytotoxicity. The enhancement of enzyme activity at low dose levels was due to generalized increases occurring in every cell, rather than to selection of a cell species particularly high in enzyme activity. A culture period of 24 hr was necessary for the complete adaptation of the cells to the culture environment, as evidenced by the response of intracellular glucose-6-phosphate dehydrogenase activity to carcinogen treatment. (35 refs)

- 79-3726 Parameters Influencing Quantitation of 3-Methylcholanthrene-induced Aryl Hydrocarbon Hydroxylase Activity in Cultured Human Lymphocytes.** (Eng) Kouri, R. E. (Dept. Biochemical Oncology, Microbiological Associates, Bethesda, MD 20016); Imblum, R. L.; Sosnowski, R. G.; Slomiany, D. J.; McKinney, C. E. *J Environ Pathol Toxicol* 2(4): 1079-1098; 1979.

Several parameters affecting the reproducible measurement of aryl hydrocarbon hydroxylase (AHH) activity in mitogen-activated human lymphocytes were determined. The most important factors controlling reproducibility were conditions that affected the degree of mitogen activation. Alterations in conditions such as initial lymphocyte concentration and type and/or lot of serum supplement resulted in large variations in control and 3-methylcholanthrene (3-MC)-treated AHH levels and variations in the time of occurrence of peak AHH activity. Suggested improvements of the assay procedure include (1) standardization of the mitogen activation step by initiation of cultures at a known cell concentration (eg, 10⁶ cells/ml), (2) cultivation of lymphocytes in medium supplemented with human AB serum instead of fetal calf serum, (3) induction of AHH activity using at least a 48 hr exposure to 3-MC, (4) assay for AHH activity at two different times to insure detection of peak activity, (5) measurement of the extent of mitogen activation by ³H-thymidine incorporation, (6) comparison of this specific activity in cultures in terms of their 3-MC-treated specific AHH activity, and (7) presentation of this specific activity in terms of units AHH/units NADH-dependent cytochrome c reductase (cyt c) activity. These changes resulted in an assay in which induced AHH levels among nine individuals were determined reproducibly, and significant differences in AHH levels were observed when no differences in degree of mitogen activation occurred. The changes should lead toward the refinement of a standardized AHH assay employing human lymphocytes, that may permit observation of genetic differences between individuals with respect to their ability to metabolize certain chemical carcinogens and, hence, their susceptibility to certain known environmental carcinogens. (29 refs)

79-3727 Initiation-Promotion Skin Carcinogenesis: Inhibition by Cyclic and Non-cyclic Nucleotides.

(Eng) Curtis, G. L. (Dept. Biochemistry, Univ. Nebraska Medical Center, 3018 South Lab. Bldg., Omaha, NE 68105); Stenback, F.; Ryan, W. L. *Cancer Lett* 6(4): 291-300; 1979.

The effect of nucleotides on initiation-promotion skin carcinogenesis was investigated in Swiss mice. Four groups of 7-wk-old female mice (30 mice each) received a single dose of 100 μ g 7,12-dimethylbenz(a)anthracene (DMBA) followed 2 wk later by application of 20 μ l of a 2.5% croton oil soln in acetone 2x/wk. Group 1 received only the DMBA + croton oil. Group 2 received 200 μ g cyclic AMP (cAMP) ip 1x/day for 5 days before the DMBA croton oil treatment. Group 3 received 200 μ g cAMP ip for 5 days starting the day after DMBA application. Group 4 received 200 μ g cAMP ip with each croton oil treatment, 2x/wk. Most of the skin tumors formed were papillomas, and there was a very low incidence of malignant tumors. The greatest reduction in total tumor formation was seen in Group 4. When cAMP-related compounds were tested during promotion, 5'-AMP also reduced tumor formation significantly, but adenosine and dibutyl-cAMP did not. Cyclic GMP inhibited tumor formation when injected at the same time as application of the promoting agent 12-O-tetradecanoylphorbol-13-acetate, but 5'-GMP was ineffective. The reduction in tumor formation may be due to the inhibition of DNA synthesis by compounds that raise intracellular cAMP levels. (23 refs)

79-3728 The Importance of the "Bay Region" Diol-Epoxide in 7,12-Dimethylbenz(a)anthracene Skin Tumor Initiation and Mutagenesis. (Eng) Slaga, T. J. (Biology Div., Oak Ridge Natl. Lab., P.O. Box Y, Oak Ridge, TN 37830); Huberman, E.; DiGiovanni, J.; Gleason, G.; Harvey, R. G. *Cancer Lett* 6(4): 213-220; 1979.

The ability of various derivatives of 7,12-dimethylbenz(a)anthracene (DMBA) to induce skin tumors in mice and mutagenesis in V79 cells was investigated to determine the cellular metabolite(s) responsible for the carcinogenicity and/or mutagenicity of DMBA. Groups of female CD1 mice received a single 200-nanomole topical application of the test compound, followed 1 wk later by twice weekly applications of 12-O-tetradecanoylphorbol-13-acetate (TPA). The frequency of ouabain-resistant mutants was determined by V79 cells that had been treated with DMBA or its derivatives 5 hr after seeding and incubated for 2 days. The 1-, 2-, 3-, 4-, and 5-OH-DMBA derivatives did not have skin-tumor-initiating activity, whereas 9- and 10-OH-DMBA had weak activity. The 5,6- and 8,9-diols were also inactive, and the (\pm)DMBA 8 β ,9 α -diol-10 α ,11 α -epoxide had only weak skin-tumor-initiating activity. None of these derivatives was mutagenic in the cell-mediated or direct V79 mutagenesis assays at

concentrations up to 4 μ M. Addition of a methyl or fluoro group to the 1, 2, or 5 positions almost completely blocked the skin-tumor-initiating and V79 mutagenic activities of DMBA, whereas addition of a fluoro group to position 11 did not. These results indicate that a bay-region diol-epoxide of DMBA may represent its carcinogenic and mutagenic forms. (37 refs)

79-3729 Estrogenic Properties of 3,9-Dihydroxy-7,12-dimethylbenz(a)anthracene in Rats. (Eng)

Morreale, C. E. (Dept. Breast Surgery, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263); Schneider, S. L.; Sinha, D. K.; Bronstein, R. E. *J Natl Cancer Inst* 62(6): 1585-1588; 1979.

The estrogenic properties of 3,9-dihydroxy-7,12-dimethylbenz(a)anthracene (DDMBA) were studied using Sprague-Dawley rats. Competitive binding studies of DDMBA with 17 β -estradiol in the uterine cytosol of immature rats gave a K_a of 1.7×10^8 M $^{-1}$. DDMBA inhibited 17 β -estradiol binding to the 8S binding protein in the rat uterus at a concentration (10^{-5} M) equal to that of nafoxidine HCl required to inhibit 17 β -estradiol specific binding. At a concentration of 10^{-8} M, DDMBA inhibited total 17 β -estradiol binding by approx 75%. DDMBA at 500 μ g increased uterine wts to the same extent as 0.112 μ g of 17 β -estradiol; thus, DDMBA had 1/4,464 the estrogenic potency of 17 β -estradiol. (20 refs)

79-3730 Comparative Metabolism and DNA Binding of 7,12-Dimethylbenz(a)anthracene and Its Weakly Carcinogenic 5-Fluoro Analog. (Eng) Daniel, F. B. (Dept. Radiology and Pharmacology, Coll. Medicine, Ohio State Univ., Columbus, OH 43210); Cazer, F. D.; D'Ambrosio, S. M.; Hart, R. W.; Kim, W. H.; Witiak, D. T. *Cancer Lett* 6(4): 263-272; 1979.

The metabolism and DNA binding of 7,12-dimethylbenz(a)anthracene (DMBA) and its weakly carcinogenic analog 5-fluoro-7,12-dimethylbenz(a)anthracene (5F-DMBA) were compared in cultured Syrian hamster embryo (SHE) cells and activated liver microsomes. The liver microsomes were incubated for 60 min with 50-200 μ Ci 3 H-DMBA or 10-40 μ Ci 14 C-5F-DMBA (100 nanomoles) in 0.10 ml acetone. SHE cells were treated with 50 μ l of 3 H-DMBA or (14 C)5F-DMBA acetone soln to give a final hydrocarbon concentration of 0.5 μ M. DMBA and 5F-DMBA were converted to water-soluble metabolites by SHE cells and rat liver microsomes at equal rates. 8,9-Dihydro-8,9-dihydroxy-7,12-dimethylbenz(a)anthracene was the major metabolite produced when both compounds were oxidized by the liver microsomes. Both compounds were also readily metabolized at the 7-methyl position. 5F-DMBA appeared to undergo less metabolism at the 5,6-bond (K region). The Sephadex LH20 chromatographic

mobilities of the DMBA-deoxynucleoside adducts were compared with those of analogous products generated by treatment with 5F-DMBA. In both the microsomal and SHE cell systems, DMBA bonded to DNA 2.5-3.0 times more extensively than did 5F-DMBA. Since insertion of a 5F substituent into DMBA greatly reduces the carcinogenicity of the parent compound, blocks metabolism in the K region, and reduces DNA binding, this position is probably involved in carcinogenesis. (26 refs)

- 79-3731 DNA Repair in Syrian Hamster Embryo Cells Treated with 7,12-Dimethylbenz(a)anthracene and Its Weakly Carcinogenic 5-Fluoro Analog.** (Eng) D'Ambrosio, S. M. (N-212 Univ. Hosp., 410 W. 10th Ave., Columbus, OH 43210); Daniel, F. B.; Hart, R. W.; Cazer, F. D.; Witiak, D. T. *Cancer Lett* 6(4): 255-261; 1979.

The postreplication repair potential of Syrian hamster embryo cells treated in culture with 7,12-dimethylbenz(a)anthracene (DMBA) and its weakly carcinogenic analog 5-fluoro-7,12-dimethylbenz(a)anthracene (5F-DMBA) were compared. Cells were incubated with ^{14}C -thymidine for 36 hr to label the parent DNA. DMBA or 5F-DMBA dissolved in dimethyl sulfoxide (0.1% final concentration) was added to the cultures for 6 hr. The cells were pulse-labeled for 30 min with ^3H -thymidine to label daughter DNA and chased for 60 min. The size of the newly synthesized DNA was determined by sedimentation in alkaline sucrose. The daughter DNA from DMBA-treated cells was smaller than the daughter DNA from cells treated with 5F-DMBA at the same concentration (5 $\mu\text{g}/\text{ml}$) or from untreated cells. The size of daughter DNA from 5F-DMBA was the same as that from untreated cells. At 5 and 1 $\mu\text{g}/\text{ml}$ DMBA, less of the daughter DNA sedimented as high-mol-wt DNA than after the same concentrations of 5F-DMBA. Although both DMBA and 5F-DMBA damage DNA, it appears that the differences are probably not related to the total number of adducts formed but rather to the levels of specific types of adducts entering DNA replication. (21 refs)

- 79-3732 Comparison of Mutagenesis and Malignant Transformation by Dihydrodiols from Benz[a]anthracene and 7,12-Dimethylbenz[a]anthracene.** (Eng) Marquardt, H. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Baker, S.; Tierney, B.; Grover, P. L.; Sims, P. *Br J Cancer* 39(5): 540-547; 1979.

Five dihydrodiols derived from benz(a)anthracene (BA) and four derived from 7,12-dimethylbenz(a)anthracene (DMBA) were tested for their abilities to induce mutagens in V79 Chinese hamster cells and malignant transformation in M2 mouse fibroblasts. The non-K-region 1,2- and 3,4-dihydrodiols of BA induced mutation to 8-azaguanine

resistance in V79 cells, whereas the K-region 5,6- and non-K-region 8,9-dihydrodiols were inactive. The non-K-region 8,9-dihydrodiol of DMBA and the corresponding 3,4-dihydrodiol were active in this system, whereas the K-region 5,6-dihydrodiol and non-K-region 10,11-dihydrodiol were inactive. Both of the parent hydrocarbons were inactive, but both, especially DMBA, showed some activity in transforming the M2 cells. None of the BA dihydrodiols showed significant activity in the induction of malignant transformation, whereas at 1 $\mu\text{g}/\text{ml}$, the 3,4- and 8,9-dihydrodiols of DMBA appeared to be more active than the parent hydrocarbon. M2 cells transformed by the 3,4-dihydrodiol of DMBA induced malignant fibrosarcomas in C3H/HeJ mice. The anti-8,9-dihydrodiol-10,11-epoxide of BA was more active as a mutagen for V79 cells than was the syn-isomer, and the anti-isomer induced malignant transformation in M2 cells but the syn-isomer did not. (46 refs)

- 79-3733 Carcinogenicity and Polarographic Behavior of Dibenz(a,h)anthracene, Dibenz(a,h)acridine, and Dibenz(a,h)phenazine.** (Eng) Bahna, L. (Cancer Res. Inst., Slovak Acad. Sci., 880 32 Bratislava, Czechoslovakia); Podany, V.; Benesova, M.; Godal, A.; Vachalkova, A. *Neoplasma* 25(6): 641-645; 1978.

The carcinogenic activities of dibenz(a,h)anthracene, dibenz(a,h)acridine, and dibenz(a,h)phenazine in rats were compared to the values of their polarographic reduction and oxidation half-wave potentials. The compounds (10 mg) were incorporated in paraffin disks and implanted sc in female Wistar rats (30 rats/compound). The number of sarcomas induced was 20, 5, and 3 for dibenz(a,h)anthracene, dibenz(a,h)acridine, and dibenz(a,h)phenazine, respectively. The tumorigenicity of the compounds was proportional to their electron-donating ability and inversely proportional to their electron-accepting ability, as defined by oxidation and reduction polarography. Previous studies have shown that the oxidation half-wave potentials of polycyclic carcinogens are in the range of several hundred millivolts. However, the half-wave potentials of some inactive polycyclic compounds also fall within this range, so that polarography cannot have predictive significance in the search for new polycyclic carcinogens. Polarography, however, might be used as a screen to exclude carcinogenicity if the oxidation half-wave potentials fall outside the range characteristic of polycyclic carcinogens. (13 refs)

- 79-3734 Inhibition of DNA Synthesis In Vitro by Binding of Benzo(a)pyrene Metabolite Diol-epoxide I to DNA.** (Eng) Mizusawa, H. (Lab. Molecular Carcinogenesis, NCI, NIH, Bethesda, MD 20014); Kakefuda, T. *Nature* 279(5708): 75-78; 1979.

CHEMICAL CARCINOGENESIS

The effect of benzo(a)pyrene (BP) binding to DNA on the replication of double-stranded circular pBR322 DNA (an artificial plasmid DNA derived from ColE1 and pBR313 in *Escherichia coli*) in vitro was studied. Incubation of unmodified pBR322 DNA in an extract from *E. coli* cells resulted in a linear increase in the incorporation of ^{32}P -TMP into the acid-precipitable fraction. When DNA covalently bound to the BP metabolite (\pm)r-7,t-8-dihydroxy-t-9, 10-oxy-7,8,9,10-tetrahydrobenzo(a)pyrene [diol epoxide I (DEI)] was used, ^{32}P -TMP incorporation was reduced about 30%, with the reduction rate being proportional to the number of DEI molecules bound to one pBR322 DNA molecule. The size classes of DNA synthesized during the first 30 min of incubation were generally unrelated to the number of DEI molecules bound. The only exception was that the formation of initial 6S fragments was slightly inhibited by DEI binding. Many intermediate-sized newly synthesized DNA fragments with lengths less than the unit size of pBR322 DNA were accumulated when DEI-modified DNA was subjected to replication. The effect of covalently bound DEI was, therefore, to inhibit chain elongation with little effect on the initiation of DNA replication. The DEI binding site was found to be heat/alkali-labile. Parent strands that had completed a round of replication no longer possessed DEI binding sites that were present before replication (16 refs)

- 79-3735 Metabolism of Benzo[a]pyrene by the Cytochrome P-450/P-448 of *Saccharomyces cerevisiae*. (Eng) Woods, L. F. (Dept. Biochemistry, Univ. Surrey, Guildford, Surrey GU2 5XH, England); Wiseman, A. *Biochem Soc Trans* 7(1): 124-127; 1979.

The metabolism of benzo(a)pyrene (BP) by the cytochrome P-450/P-448 aryl hydrocarbon hydroxylase system of *Saccharomyces cerevisiae* was studied. The pH optimum of the yeast enzyme was 7.0, compared with 7.4 for the liver enzyme, and the K_m for BP was 0.52 mM with untreated yeast. Growth of yeast in the presence of 5 $\mu\text{g}/\text{ml}$ BP decreased the K_m to 0.16 mM. Pretreatment of yeast with phenobarbital had no effect on the K_m . Cumene hydroperoxide (CM: 2.4 mM) reduced the K_m for BP from 2.7 mM to 0.36 mM when yeast was pretreated with BP; phenobarbital did not alter it. Solubilization of the yeast enzyme with sodium cholate yielded a preparation that could metabolize BP with CH, but not with an NADPH-regenerating system, as cofactor. Addition of phosphatidylcholine did not restore the ability to use NADPH. BP metabolites formed by incubation with yeast enzyme were 3-hydroxy-BP, 9-hydroxy-BP, and the 7,8-dihydrodiol. The yeast enzyme produced type-I spectral changes with BP, although there was an additional peak in the spectrum at 360 nanometers. Untreated rat liver microsomal fraction produced the same spectrum. The binding constant for the yeast microsomal fraction was 36 μM , and that for liver microsomal fraction was 9 μM . The bin-

ding constant for the solubilized yeast cytochrome P-450/P-448 system was 53 μM . (23 refs)

- 79-3736 Induction of Benzo(a)pyrene Monooxygenase in Fish and the Salmonella Test as a Tool for Detecting Mutagenic/Carcinogenic Xenobiotics in the Aquatic Environment. (Eng) Kurelec, B. (Lab. Marine Molecular Biology, Center Marine Res., Rudjer Boskovic Inst., P.O. Box 1016, 41001 Zagreb, Yugoslavia); Matijasevic, Z.; Rijavec, M.; Alacevic, M.; Britvic, S.; Muller, W. E.; Zahn, R. K. *Bull Environ Contam Toxicol* 21(6): 799-807; 1979.

The induction of benzo(a)pyrene monooxygenase (BPMO) following the ip injection of young carp (*Mugil cephalus*) with polluted water extracts and the testing of these extracts in the *Salmonella*/microsome mutagenicity test using liver homogenates of pollution-induced fish were evaluated as an assay for detecting xenobiotics in aquatic environments. Injection of the following substances induced BPMO activity in young carp within 1-2 days: benzo(a)pyrene (BP), 25 and 50 μg ; aflatoxin, 25 μg ; crude oil, 50 μl ; hexane extract of charcoal-filtered seawater (HESW), 50 μl , + crude oil, 50 μl ; and HESW 50, collected from the mixing zone 50 m off a cannery outlet, 50 μl . Carp caught in the mixing zone had extremely high BPMO activity (1,600 units), but three specimens caught in clean areas had activities of only 65, 71, and 52 units. This indicates that the BPMO in the specimens caught in the mixing zone was induced by substances in the fish cannery effluent. In the mutagenicity assay, neither HESW, aflatoxin, nor BP was mutagenic in the absence of a liver postmitochondrial fraction. HESW 50, but not HESW 500 (collected 500 m from the cannery outlet), induced BPMO in carp and was mutagenic for *Salmonella* when the liver fraction from HESW 50-treated carp was added. The max mutagenic effect of HESW was seen with the liver fraction from carp from the mixing zone, but this liver fraction also produced revertants without a substrate. It is concluded that the ip injection of polluted water extracts into carp followed by determination of BPMO induction plus the *Salmonella* assay can be used to detect mutagenic and/or carcinogenic xenobiotics in the aquatic environment. (23 refs)

- 79-3737 Hepatic Microsomal Mixed-Function Oxidase Activities in Several Marine Species Common to Coastal Florida. (Eng) James, M. O. (Lab. Pharmacology, Natl. Inst. Environmental Health Sciences, C. V. Whitney Marine Lab., St. Augustine, FL 32084); Khan, M. A.; Bend, J. R. *Comp Biochem Physiol [C]* 62(2): 155-164; 1979.

NADPH-dependent hepatic microsomal mixed-function oxidase activities were measured in several marine

vertebrate and invertebrate species common to the Florida Atlantic coast using benzo(a)pyrene, 7-ethoxycoumarin, benzphetamine, and aniline as substrates. Cytochrome P-450 and NADPH-cytochrome c reductase activity was found in the hepatic and hepatopancreatic microsomes from all fish and crustaceans studied. Mixed-function oxidase activity was easily detected in hepatic microsomes from teleost and elasmobranch fish, but it was low or undetectable in hepatopancreatic microsomes from crustaceans. Mixed-function oxidase activity in microsomes prepared from extrahepatic organs was usually much lower than that found in hepatic microsomes. However, for two teleost species (sheepshead and black drum), benzphetamine N-demethylase activity of the gill microsomes approached that of the hepatic microsomes. The temperature optimum for in vitro mixed-function oxidase activity was higher in the Florida fish tested than has been reported for cold-water-acclimated marine or freshwater fish. (32 refs)

- 79-3738 Benzo(a)pyrene Metabolism in Neonatal Rat Liver Nuclei.** (Eng) Bresnick, E. (Dept. Biochemistry, Univ. Vermont Coll. Medicine, Burlington, VT 05401); Chuang, A. H.; Bornstein, W. A. *Chem Biol Interact* 24(1): 111-115; 1979.

The effects of the mixed-function oxidase inducer 3-methylcholanthrene (3-MC: 25 mg/kg, ip 24 hr before sacrifice) on the formation of various benzo(a)pyrene metabolites (BP) in the liver of young adult and newborn (1-2-day-old) male Sprague-Dawley rats were studied. Liver nuclei from 100-g rats formed the three known BP transdiols, 4,5-, 7,8-, and 9,10-dihydrodiol, and three quinones, 1,6-, 3,6-, and 6,12-quinone, and the two phenols, 3- and 9-phenol; the 4,5-oxide of BP may also have been formed. The nuclei from newborn rats formed all metabolites but the 9,10-dihydrodiol. The major metabolites in both cases were the quinones and phenols. In the newborns, phenol I (the 9-hydroxy-BP derivative) had approx 4-fold greater activity than phenol II (the 3-hydroxy-BP derivative), whereas the amounts of these derivatives were approx equal in the young adults. After 3-MC treatment, the specific activities of the phenols and 4,5-dihydrodiols were elevated four- to fivefold in the newborn rats, and the activities of phenol II and the 4,5-dihydrodiol were increased eight- and sixfold, respectively, in the young adults. The proximate carcinogen, the 7,8-dihydrodiol, was elevated three- to fourfold in both systems after 3-MC treatment. (28 refs)

- 79-3739 In Vitro Benzo(a)pyrene Metabolism from Lindane-treated Rat Liver: Effect of Oral and Acute Administration, and Comparison with Phenobarbital and Methylcholanthrene Pretreatment.** (Eng) Mikol, Y. B. (Carcinogen Metabolism and Toxicology Branch,

NCI, Bethesda, MD 20014); Decloitre, F. *Toxicol Appl Pharmacol* 47(3): 461-467; 1979.

The effects of po and acute administration of lindane (LD: γ -hexachlorocyclohexane) on the rat liver metabolism of benzo(a)pyrene (BP) were studied by determining aryl hydrocarbon hydroxylase (AHH) activity and binding of 14 C-BP metabolites to DNA and by chromatographic analysis of BP metabolites. Male Sprague-Dawley rats (4 wk old) were fed 24, 120 or 240 ppm LD in their basal diet for 4 wk. Acute intoxication was obtained by ip injection of LD (20 mg/kg/day) or phenobarbital (PB: 80 mg/kg/day) for 3 consecutive days. The spectrofluorometric measurement of AHH activity did not show any induction after po or acute LD or PB pretreatment, but quantitative determination of BP metabolites by thin-layer chromatography indicated an increase in the amount of 3-hydroxy-benzo(a)pyrene (3-OH-BP). The amount of metabolized BP was higher in PB-treated rats (60%) than in LD-treated rats (41%), which suggests that LD is a moderate inducer of BP hydroxylase and is less efficient than PB. The lower po dose of LD produced an 80% increase in metabolites bound to DNA. The increase was dose-dependent up to 120 ppm, but a plateau was observed at 120-240 ppm. Acute intoxication by LD increased BP metabolite binding to DNA to levels similar to those obtained with 120 ppm po. The large increase in 4,5-BP dihydrodiol (8.9%) induced by LD was related to a twofold increase in epoxide-hydratase activity. These results suggest that LD could be considered a PB-like inducer. (26 refs)

- 79-3740 Metabolic Activation of Benzo(a)pyrene and 9-Hydroxybenzo(a)pyrene by Tissue Fractions from Rat Liver and Lung.** (Eng) Prough, R. A. (Dept. Biochemistry, Univ. Texas Health Science Center, 5323 Harry Hines Boulevard, Dallas, TX 75235); Capdevila, J.; Lubet, R. A. *Biochem Soc Trans* 7(1): 122-124; 1979.

The conversion of 9-hydroxybenzo(a)pyrene (HBP) and benzo(a)pyrene (BP) to mutagenic and alkylating moieties by liver and lung tissue from male Sprague-Dawley CD rats was studied. The rats were pretreated with 5,6-benzoflavone (80 mg/kg ip for 4 days). When activated by rat liver microsomes, HBP was 1.6-fold more active than BP as a premutagen for *Salmonella typhimurium* strain TA100, and it was 6-fold more active as an alkylating agent. After activation by the postmitochondrial supernatant of alveolar tissue, HBP was nearly as active as a premutagen as 7,8-dihydrobenzo(a)pyrene-7,8-diol and two to three times more active than BP. (9 refs)

- 79-3741 The DNA Binding of Benzo(a)pyrene Metabolites Catalysed by Rat Lung Microsomes In Vitro and in Isolated Perfused Rat Lung.** (Eng) Vahakangas, K. (Dept. Pharmacology, Univ. Oulu,

SF-90220 Oulu 22, Finland); Nebert, D. W.; Pelkonen, O. *Chem Biol Interact* 24(2): 167-176; 1979.

When [^3H]benzo(a)pyrene (BP) was incubated with DNA, NADPH, and rat lung microsomes, covalent binding of BP metabolites to the DNA occurred. These metabolite-nucleoside complexes could be resolved into several distinct peaks by elution of a Sephadex LH-20 column with a water-methanol gradient. 3-Methylcholanthrene (3-MC) pretreatment of animals induced the total covalent binding in vitro severalfold and increased the amounts of at least five metabolite-nucleoside complexes associated with the 7,8-diol-9,10-epoxides, the 7,8-oxide or quinones oxygenated further, and the 4,5-oxide and phenols oxygenated further. These increases corresponded well with increases in the production of both non-K-region and K-region BP metabolites by lung microsomes, as determined by high-pressure liquid chromatography. When [^3H]BP was metabolized in isolated perfused rat lung, only the peak representing the 7,8-diol-9,10-epoxide bound to nucleoside(s) was readily detectable, and then only in lungs from 3-MC-treated animals. The extent of binding of BP metabolites to lung DNA was very low, about 0.0004% of the total dose applied to the perfusion medium; >60% of this could be accounted for by the binding of the 7,8-diol-9,10-epoxides to nucleoside(s). It is suggested that the further metabolism leading to metabolites not available to covalent binding (eg conjugation) of primary BP metabolites in the intact tissue is responsible for differences in the metabolite-nucleoside patterns observed in vivo, compared with microsomal metabolism in vitro. (44 refs)

79-3742 A Comparison of the Activities of Aryl Hydrocarbon Monooxygenase in Liver Microsomes from Mice of Different Strains During Prolonged 3,4-Benzo(a)Pyrene Administration. (Eng) Tsyrlav, I. B. (Dept. Biochemistry, Inst. Experimental and Clinical Medicine, Acad. Medical Sciences of USSR, Siberian Branch, Novosibirsk 630091, USSR); Lyakhovich, V. V. *Biochim Biophys Acta* 584(1): 11-20; 1979.

Aryl hydrocarbon monooxygenase (AHM) activity was measured in the liver microsomes of C57BL/6, DBA/2, and F₁ hybrid mice during prolonged treatment with benzo(a)pyrene (BP: 40 mg/kg/day ip for 4 days, then 20 mg/kg ip 3x/wk). Animals were sacrificed at 5 days and every 2 wk after the start of treatment. The content and activity of the components of the liver microsomal AHM system changed biphasically during treatment in the C57BL/6 and F₁ hybrid mice. The first activity peak (4-14 days) was associated with the induction of AHM by BP; the second peak (70-84 days) was related to a noninductive mechanism. In the DBA/2 mice, the second peak was absent, but a slight increase in AHM activity on days 14-28 indicated the aberrant inductive capacity of BP after prolonged administration. It is suggested that the weak sensitivity to BP-induced carcinogenesis in C57BL/6 and F₁

mice was due to the high level of liver AHM activity at the time of tumor appearance (70-84 days after BP administration). (29 refs)

79-3743 The Binding to Mouse Skin DNA of Benzo(a)pyrene, Its 7,8-Diol and 7,8-Diol-9,10-epoxides in Relation to the Tumorigenicity of These Compounds. (Eng) Brookes, P. (Chemical Carcinogenesis Div., Inst. Cancer Res., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, England). *Cancer Lett* 6(4): 285-289; 1979.

The binding to mouse skin DNA OF ^{14}C - and ^3H -labeled benzo(a)pyrene (BP), (\pm)trans-7,8-dihydroxy-7,8-dihydrobenzo(a)pyrene (BP-7,8-diol), (\pm -7 α ,8 β -dihydroxy-9 β ,10 β -epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (anti-BPDE), and (\pm -7 α ,8 β -dihydroxy-9 α ,10 α -epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (syn-BPDE) was compared. The test compounds were applied topically in equimolar doses in 0.3 ml tetrahydrofuran soln to 7- to 8-wk-old CBA female mice. After 24 hr, the DNA was isolated from the treated skin and the extent of hydrocarbon binding determined by liquid scintillation counting. The binding to DNA of BP, BP-7,8-diol, and anti-BPDE was of the same order, but for syn-BPDE it was significantly lower (about one-third). These results were consistent with the similar tumor-initiating activity of BP and BP-7,8-diol and with the greater activity of anti-BPDE compared with syn-BPDE. The lower activity of anti-BPDE compared with BP is not consistent with their approx equal binding to DNA. This may indicate that metabolites other than anti-BPDE are involved in skin tumor initiation or that an in vivo metabolically generated diol epoxide reacts more specifically with DNA than does topically applied anti-BPDE. (19 refs)

79-3744 The In Vitro and In Vivo Reaction at the N⁷-Position of Guanine of the Ultimate Carcinogen Derived from Benzo(a)pyrene. (Eng) King, H. W. (Chemical Carcinogenesis Div., Inst. Cancer Res., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, England); Osborne, M. R.; Brookes, P. *Chem Biol Interact* 24(3): 345-353; 1979.

The reaction occurring at the N²- and N⁷- positions of guanine following the addition of 7 α ,8 β -dihydroxy-9 β ,10 β -epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (anti-BPDE) to an aqueous soln of DNA was studied in detail. The extent of the reaction and the relative yields of N²- and N⁷-products were measured over the pH range 4-7. The depurination following reaction at the N⁷-position of guanine was found to have a half-life of 3 hr that varied little over the pH range studied. Reaction of the isomeric 7 α ,8 β -dihydroxy-9 α ,10 α -epoxy-7,8,9,10-tetrahydro-

benzo(a)pyrene (syn-BPDE) with DNA gave the expected N²-but no N⁷-guanine product. When benzo(a)pyrene or anti-BPDE was added to mouse embryo or Chinese hamster V79 cells, respectively, a major N²-guanine product and a very minor adenine product were isolated from the DNA, but no N⁷-guanine product was detected. (15 refs)

- 79-3745 Toxicologic and Pathologic Data on Polycyclic Aromatic Hydrocarbons and Automobile Exhaust Condensate.** (Eng) Mohr, U. (Abteilung für Experimentelle Pathologie, Medizinische Hochschule Hannover, Karl-Wiechert Allee 9, 3000 Hannover 61, W. Germany). *Ecotoxicol and Environ Saf* 2(3/4): 267-276; 1978.

The carcinogenicity of polycyclic aromatic hydrocarbons (PAH) and automobile exhaust condensate (AEC) in the respiratory tract was tested in Syrian golden hamsters by instillation, implantation, and inhalation. Intratracheal instillation of AEC (2.5 or 5 mg/animal at 2-wk intervals for 30-60 wk) induced multiple pulmonary tumors in 100% of treated hamsters. Implantation of beeswax pellets containing 0.05, 0.1 or 1.0 mg benzo(a)pyrene (BP) in the hamster lung induced sarcomas that were highly malignant. In two inhalation experiments in which hamsters were exposed to 2, 10, 40, or 50 mg BP/m³ air for 4.5 hr/day, no neoplastic alterations were detected after as long as 14 mo. Of the three methods, instillation is the most tested and is relatively simple to perform. Tumor induction is high but it is not suitable for establishing a dose-response relationship. Implantation is highly effective in inducing neoplasms at very small doses of BP and in establishing a dose dependence. Its disadvantages are the high temperature at the time of implantation and the introduction of a foreign body. All three methods should be correlated for a final estimation of the carcinogenic hazards of PAH and AEC to humans. (22 refs)

- 79-3746 Investigations on the Carcinogenic Burden by Air Pollution in Man. Intratracheal Instillation Studies with Benzo(a)pyrene in a Mixture of Tris Buffer and Saline in Syrian Golden Hamsters.** (Eng) Ketkar, M. (Abteilung für Experimentelle Pathologie, Medizinische Hochschule Hannover, Karl-Wiechert-Allee 9, 3000 Hannover 61, W. Germany); Green, U.; Schnieder, P.; Mohr, U. *Cancer Lett* 6(4): 279-284; 1979.

The effect of low doses of benzo(a)pyrene (BP) in a mixture of Tris buffer and physiological saline was studied in Syrian golden hamsters. Four groups of hamsters (30 males each) were given weekly intratracheal instillations of 0.125, 0.25, 0.50, or 1.00 mg BP for life. Animals were observed for life or sacrificed when moribund, and complete autopsies were performed. With increasing dose levels >0.25 mg BP, survival times, av body wt, and tumor incidence decreased. No respiratory tract tumors were observed in

untreated animals or in those given the vehicle only. Nine of 29 animals given 0.125 mg BP developed respiratory tract tumors. The corresponding figures for the other groups were 24/29 animals given 0.25 mg, 19/29 given 0.50 mg, and 9/29 given 1.0 mg BP. At similar cumulative BP doses in groups given 0.25, 0.50, and 1.0 mg BP, wt loss and shortened survival times correlated as did tumor distribution, indicating that treatment regimen is more important than total BP dose. Papillary polyps, squamous cell papillomas, and squamous cell carcinomas developed in the larynx and trachea. Bronchogenic adenomas, adenocarcinomas, and squamous cell carcinomas were induced in the lung. The results indicate that doses >0.25 mg, when given chronically, are toxic rather than carcinogenic. (8 refs)

- 79-3747 Experimental Hepatocarcinogenesis in Rat and Cellular Detection of alpha₁-Fetoprotein by Use of Peroxidase Conjugates.** (Eng) Kuhlmann, W. D. (Immunocytochemistry SFB 136, Institut für Nuklearmedizin, D.K.F.Z., Im Neuenheimer Feld 280, D-69 Heidelberg, W. Germany). *Scand J Immunol [Suppl]* 8(8): 407-416; 1978.

To provide more information on the cellular basis of α_1 -fetoprotein (AFP) synthesis during hepatocarcinogenesis, intracellular AFP was stained with immunoperoxidase at various stages of hepatoma induction in male BD X rats. Hepatomas were induced by feeding the rats low (6 mg/kg) or high (20 mg/kg) doses of N-nitrosomorpholine (NNM) for 12 and 6 wk, respectively. Necrosis of adult hepatocytes and proliferation of oval-shaped cells were seen only in rats fed the high dose of NNM. These changes occurred in parallel, with the early reappearance of AFP in the sera. At this time, AFP was detected in the oval-shaped cells by the immunoperoxidase staining technique. Both low and high doses of NNM resulted in the development of hepatomas. At this stage of hepatocarcinogenesis, AFP-staining-nodules were seen concomitantly with non-AFP-staining nodules in the same animal. AFP-staining cells consisted of distinct neoplastic hepatocytes displaying a certain degree of dedifferentiation. Pulse-chase experiments showed the highly proliferative character of the carcinoma cells, among which the AFP-staining population was the most proliferative. (39 refs)

- 79-3748 Altered Lipid Microviscosity in Lymphoblastoid Cells Treated with 12-O-Tetradecanoyl Phorbol 13-Acetate, a Tumor Promoter.** (Eng) Castagna, M. (Institut de Recherche Scientifique sur le Cancer, 7 rue Guy Mocquet, 94800 Villejuif, France); Rochette-Egly, C.; Rosenfeld, C.; Mishal, Z. *FEBS Lett* 100(1): 62-66; 1979.

The effect of 12-O-tetradecanoylphorbol-13-acetate (TPA) on lipid microviscosity was studied in several lym-

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phoblastoid cell lines. Murine (L1210) and human lymphoblastoid cell lines of normal (LHN 13) and malignant (Namalwa, Raji, Reh 6) origin were treated with TPA or 4-O-methylphorbol-12,13-didecanoate (MePDD) 12 hr after plating. T and B lymphocytes and leukemic cells were also treated. TPA decreased the lipid microviscosity the B lymphocytes and 3/5 cell lines rapidly and in a dose-dependent manner, as demonstrated by an increase in fluorescence polarization. The effect was detectable at a concentration of 1 nanogram/ml. The cells were about 100-fold less sensitive to MePDD, the inactive derivative of TPA. The TPA-treated cells also exhibited altered adhesive properties. The unresponsive lymphoblastoid cell lines (Raji or L1210) neither attached to the culture dish nor showed a decrease of membrane microviscosity, regardless of TPA concentration and time of the fluorescence polarization measurements. Phospholipid and cholesterol synthesis was markedly stimulated at 6 hr after the start of TPA treatment. These results favor the possibility that TPA specifically acts upon lipid metabolism, and they suggest that the TPA-mediated alterations of lipid metabolism and lipid membrane fluidity may be relevant to tumor promotion. (31 refs)

79-3749 Tumor Promoters Induce Membrane Changes Detected by Fluorescence Polarization. (Eng)

Fisher, P. B. (Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, 701 W. 168th St., New York, NY 10032); Flamm, M.; Schachter, D.; Weinstein, I. B. *Biochem Biophys Res Commun* 86(4): 1063-1068; 1979.

Fluorescence polarization of the probe 1,6-diphenyl-1,3,5-hexatriene (DPH) was used to determine if tumor-promoting agents alter cell membranes. The potent tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) decreased the fluorescence polarization of DPH in rat embryo cells at a concentration as low as 0.1 nanogram (ng)/ml; a max decrease was induced by 25 mg/ml TPA. A time-course (1-5 hr) study showed that TPA (200 ng/ml) caused a significant decrease in polarization, compared with control values, within 1 hr; the effect was max at 3-4 hr and partially reversed at 5 hr. The addition of actinomycin D (10 µg/ml) or cycloheximide (100 µg/ml) failed to block the TPA effect, suggesting that the change in DPH fluorescence polarization does not require new RNA or protein synthesis. Two compounds inactive in tumor promotion, phorbol and 4 α -phorbol-12,13-didecanoate (100 ng/ml), failed to alter the fluorescence polarization significantly, but the active tumor promoters TPA, phorbol-12,13-didecanoate, and phorbol-12,13-dibenzoate decreased DPH fluorescence polarization by 15.7% ($p < 0.001$), 11.3% ($p < 0.001$), and 4.0% ($p < 0.05$), respectively. The results provide further evidence that TPA and related tumor promoters affect the dynamic organization of cell membranes. (27 refs)

79-3750 Effect of Tumor Promoters, Protease Inhibitors, and Repair Processes on X-ray-

induced Sister Chromatid Exchanges in Mouse Cells. (Eng) Nagasawa, H. (Dept. Physiology, Harvard Univ. Sch. Public Health, 665 Huntington Ave., Boston, MA 02115); Little, J. B. *Proc Natl Acad Sci USA* 76(4): 1943-1947; 1979.

The induction of sister chromatid exchanges (SCE) in the second postirradiation mitosis was studied in mouse 10T1/2 cells irradiated with 400 rads and maintained in stationary growth for several hr after x-ray exposure (similar to liquid holding recovery experiments in bacterial cells). X-irradiation with no recovery period induced few SCE. With short recovery intervals, however, the SCE frequency rose in parallel with the increase in survival, reaching a max twofold increase after 4 hr; SCE declined with longer recovery intervals. The influence of postirradiation incubation with the tumor promoter 12-O-tetradecanoylphorbol 13-acetate (TPA) and with the protease inhibitors antipain and leupeptin was studied on spontaneous, x-ray-induced (no recovery), and recovery-induced (4 hr) SCE. TPA (0.1 µg/ml and 1.0 µg/ml) increased the frequency of both spontaneous and direct x-ray-induced SCE, but not of recovery-induced SCE. Incubation with the protease inhibitors suppressed both TPA- and recovery-induced SCE, but it had no effect on direct x-ray-induced SCE. The increase in SCE frequency induced by irradiation with a short recovery period, and the decrease with the longer period, corresponds with the results of a previous experiment in which 400 rads induced a threefold increase in transformation frequency with recovery intervals of 3-4 hr, while transformation frequency declined with longer intervals; this suggests that a relationship exists between SCE induction and malignant transformation. (39 refs)

79-3751 The Tumor Promoter 12-O-Tetradecanoylphorbol-13-acetate Elevates Serum Progesterone Levels. (Eng) Sharma, O. K. (Dept. Basic Oncology, AMC Cancer Res. Center, 6401 West Colfax Ave., Lakewood, CO 80214); Kerr, S. J. *Biochem Biophys Res Commun* 87(4): 1039-1043; 1979.

The induction of ovalbumin in estrogen-treated immature Rhode Island Red chicks by the phorbol ester 12-O-tetradecanoyl-phorbol-13-acetate (TPA: 200 µg ip) was studied. TPA caused a five- to sevenfold elevation in serum progesterone levels and induced ovalbumin synthesis within 3-4 days. In some experiments, ovalbumin synthesis could be induced by 40 µg TPA. 4- α -Phorbol-12,13-didecanoate, which is inactive as a mouse skin tumor promoter, did not increase progesterone levels or induce ovalbumin synthesis. Ovalbumin synthesis was not induced by TPA in chicks that had not received primary stimulation with estradiol benzoate. (26 refs)

79-3752 Maintenance of Human Skin on Nude Mice for Studies of Chemical Carcinogenesis. (Eng)

Yuspa, S. H. (In Vitro Pathogenesis Section, Lab. Experimental Pathology, Carcinogenesis Res. Program, NCI,

Bethesda, MD 20014); Viguera, C.; Nims, R. *Cancer Lett* 6(4): 301-310; 1979.

Pieces of neonatal human foreskin were grafted to the right and left dorsal thoraces of nude mice in an attempt to develop a model of carcinogenesis for human skin. Of the 180 double-sided grafts that were attempted, 88 were sufficiently successful (either one or both sides) to undergo treatment. By the end of the experiment (22-27 wk), only 38 animals had grossly visible grafts. The mice were given a single injection of urethan (1 mg/g ip), and topical administration of 12-O-tetradecanoylphorbol-13-acetate (TPA: 2.5 µg in 0.05 ml acetone 2x/wk) was started 7 days later. In mice with two grafts, one side was treated with TPA and the other with acetone. In groups given urethan and human grafts, the incidence of mouse skin papillomas increased. TPA treatment was required, since all of the tumors were in the TPA-treated areas. No carcinomas were observed. TPA induced hyperplasia in the grafts relative to acetone-treated controls. There were two benign squamous papillary lesions associated with human skin. One appeared to be of human origin, but the origin of the second lesion could not be determined. It is concluded that this model provides an in vivo system for studying mechanisms of carcinogenesis in human tissue. As techniques for prolonged survivals of nude mice and grafts improve, such a model should be applicable to bioassays as well. (16 refs)

79-3753 Lymphocyte Activation by the Tumor-promoting Agent 12-O-Tetradecanoylphorbol-13-acetate (TPA). (Eng) Abb, J. (Max von Pettenkofer Inst. of Hygiene and Medical Microbiology, Pettenkoferstrasse 9a, D-8000 Munich 2, W. Germany); Bayliss, G. J.; Deinhardt, F. *J Immunol* 122(5): 1639-1642; 1979.

The ability of 12-O-tetradecanoylphorbol-13-acetate (TPA) to stimulate DNA synthesis in human marmoset, baboon, rhesus monkey, chimpanzee, and dog peripheral blood lymphocytes (PBL) and in spleen cells from guinea pigs, rats, and mice was studied. TPA in concentrations from 2.5 to 750 nanograms (ng)/ml stimulated human lymphocytes, with optimal stimulation occurring 96 hr after the addition of 7.5 ng/ml. Dimethyl sulfoxide had no such effect, and it did not significantly affect the lymphocyte response to plant lectins. Both T- and B-enriched lymphocyte populations responded to TPA. Similar responses to TPA were obtained with cord blood lymphocytes and with lymphocytes from Epstein-Barr virus-negative and -positive adult donors. Incorporation of ³H-thymidine by marmoset, baboon, rhesus monkey, and chimpanzee PBL lymphocytes was significantly increased by TPA, whereas canine PBL and spleen cells from guinea pigs, rats, and mice were not stimulated by TPA. The data suggest that TPA-induced lymphocyte blastogenesis may be useful for studies of lymphocyte activation and of the molecular mechanisms of action of tumor-promoting phorbol esters. (27 refs)

79-3754 Tumor-promoting Phorbol Diester Induces Substrate-Adhesion and Growth Inhibition in Lymphoblastoid Cells. (Eng) Castagna, M. (Institut de Cancerologie et d'Immunogenetique, 16 Avenue Paul Vaillant Couturier, 94800 Villejuif, France); Rochette-Egly, C.; Rosenfeld, C. *Cancer Lett* 6(4): 227-234; 1979.

The responses of normal or leukemic human lymphoblastoid cells to 12-O-tetradecanoylphorbol-13-acetate (TPA) were studied. The cells were subcultured every 2-3 days, treated with TPA [100 nanograms(ng)/ml] 12 hr after subculturing, and examined for adhesion. TPA altered the smooth spherical shape of normal lymphoblastoid LHN 13 cells, which appeared ruffled and formed clusters. The leukemic Reh 6 cells did not aggregate significantly after treatment. Both cell lines adhered to plastic and glass dishes at similar rates and to the same extent. After 24 hr exposure to TPA, 80% of the LHN 13 line was adherent. The response of the Reh 6 line was similar but less intense. At 24 hr, TPA affected adhesion and growth inhibition at a concentration as low as 1 ng/ml, with the max response occurring at 10-100 ng/ml. 4-O-Methylphorbol-12,13-didecanoate which has no tumor-promoting activity, was much less effective than TPA in altering cell adhesion and cell growth. Some tumor cell lines exhibited altered substrate-adhesion properties, but others did not. There was no relationship between growth and adhesion effects, since all the TPA-treated lines but one were growth-inhibited at an early treatment stage, even when adhesion was unaffected. These results suggest that the tumor promoter alters the dynamic properties of the membrane, properties believed to play a major role in cellular control mechanisms. (19 refs)

79-3755 A Possible Pathogenic Mechanism for the Induction of Rotenone Tumours. (Eng) Gosalvez, M. (Clinica Puerta de Hierro, San Martin de Porres 4, Madrid-35, Spain); Diaz-Gil, J.; Alcaniz, J.; Borrell, J. *Biochem Soc Trans* 7(1): 113-115; 1979.

Injection of rotenone (0.1 mg/day ip) in female Wistar rats produced a marked elevation of growth hormone (GH) levels on day 2, a diminution of prolactin levels, and transient elevations in estrogen, progesterone, and corticosterone levels, suggesting hypophyseal stimulation. Increases in cerebral glutamate and hypothalamus norepinephrine levels on days 3 and 4, respectively, suggested a mechanism for pituitary gland stimulation. The physiopathogenic sequence elicited by rotenone administration is suggested to be an increase in glutamate, noradrenaline, GH, and somatomedins, in that order. (16 refs)

79-3756 Vitamin A and Lung Cancer. (Eng) Mettlin, C. (Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263); Graham, S.; Swanson, M. *J Natl Cancer Inst* 62(6): 1435-1438; 1979.

The interaction of dietary vitamin A and smoking in the etiology of lung cancer was studied retrospectively by interview of 292 white male patients with lung cancer and 801 controls with nonrespiratory, nonneoplastic diseases. An ascending relative risk (RR) of lung cancer was associated with descending levels of vitamin A in the diet. The same pattern was observed for cigarette smoking, the association being greatest for older heavy smokers. An increased RR associated with heavy and light cigarette smoking was observed among men who consumed milk infrequently, and an increased RR associated with heavy smoking was observed among those who ingested carrots infrequently. Cancer of the respiratory tract was not associated with intake of fat, protein, carbohydrate, or vitamin C or with rural vs urban living. (28 refs)

- 79-3757 Retinyl Acetate Prophylaxis in Cancer of the Urinary Bladder.** (Eng) Dawson, W. D. (Dept. Biology, Northeast Louisiana Univ., Monroe, LA 71209); Miller, W. W.; Liles, W. B. *Invest Urol* 16(5): 376-377; 1979.

The prophylactic effects of four previously untested low levels of natural vitamin A, as retinyl acetate (RA), on N-4-(5-nitro-2-furyl)-2-thiazolylformamide (FANFT)-induced bladder cancer were determined in 275 virgin female C3H/He mice. The mice were divided into five groups and fed a vitamin A-deficient diet containing 300 IU RA/kg feed with no FANFT (Group 1, controls) or a vitamin A-deficient diet containing 300, 600, 1,200, or 2,400 IU RA/kg feed + 0.1% FANFT (Groups 2-5, respectively). After 45 wk, the bladders were removed and inspected for neoplasms. The incidence of bladder tumors was 0%, 75%, 42%, 72%, and 73% in Groups 1-5, respectively. RA had significant prophylactic effects against transitional cell and squamous cell tumors in Group 3 mice. In addition, squamous cell tumors were inhibited at all RA levels. There was a tendency toward more invasive cancer in mice receiving >600 IU RA/kg; however, the differences among the groups with regard to tumor stage were not significant. The results reveal that RA has a prophylactic effect against transitional cell and squamous cell tumors of the urinary bladder at a dose level lower than those used previously but that there is no increased prophylaxis at high RA levels. (12 refs)

- 79-3758 The Binding of Metabolites Formed from Aminostilbene Derivatives to Nucleic Acids in the Liver of Rats.** (Eng) Gaugler, B. J. (Institut für Pharmakologie und Toxikologie, Universität Würzburg, Versbacher Landstrasse 9, 8700 Würzburg, W. Germany); Neumann, H. G. *Chem Biol Interact* 24(3): 355-372; 1979.

The carcinogens trans-4-dimethylaminostilbene (trans-DAS) and trans-4-acetylaminostilbene (trans-AAS), as well as the inactive compounds cis-DAS and 4-

dimethylaminobiphenyl (DABB), were highly and specifically labeled with tritium and administered po to female Wistar rats. Except for DABB, which was given at a dose of 22 micromoles (μmol)/kg, the dose given was 25 μmol /kg. Covalent binding to liver ribosomal RNA (rRNA) and DNA was measured and found to be higher for the carcinogens. Digests from these nucleic acids were chromatographed on Sephadex LH-20, and 16 different nucleoside adducts were characterized by their retention volumes. Labeled trans-DAS was administered in doses ranging from 0.025-250 μmol /kg. Binding to nucleic acids was directly proportional to the dose at low doses (0.025-2.5 μmol /kg) and less than proportional at higher doses (25-250 μmol /kg). The pattern of nucleoside adducts remained practically constant over the wide dose range. A pharmacokinetically determined threshold of metabolic activation thus could not be demonstrated for this compound. A modified procedure to simultaneously isolate pure liver rRNA and DNA from nonfasted rats in high yields is described. (52 refs)

- 79-3759 Endometrial Adenocarcinoma: In Estrogen, Oral Contraceptive and Nonhormone Users.** (Eng) Blythe, J. G. (Dept. Obstetrics and Gynecology, Section Gynecologic Oncology, St. John's Mercy Medical Center, 615 S. New Ballas Road, Creve Coeur, MO 63141); Ali, Z. *Gynecol Oncol* 7(2): 199-205; 1979.

The possible role of hormone use in endometrial carcinoma was investigated in 40 patients younger than age 50 with a diagnosis of endometrial adenocarcinoma (EAC). The average age of the patients was 44.2 yr. Seven patients had consumed oral contraceptives, 14 had consumed other estrogen-containing hormones, and 19 patients had no history of receiving estrogen-containing hormones. Sixty-percent of the 40 patients had a Class I pap smear. Twenty-nine patients (72.5%) had a well-differentiated malignancy. There were few differences among the groups as to average age, presenting symptoms, uterine size, stage of the disease, and relation of EAC to metabolic abnormality. (15 refs)

- 79-3760 Reasonable Surgical Treatment for Tumors of the Liver Associated with the Use of Oral Contraceptives.** (Eng) Catalano, P. W. (Dept. Surgery, Ohio State Univ. Hosps., Columbus, OH); Martin, E. W.; Ellison, C.; Carey, L. C. *Surg Gynecol Obstet* 148(5): 759-763; 1979.

The occurrence of benign liver tumors in nine Caucasian women (mean age, 30 yr) who had been taking oral contraceptives is reported. Routine laboratory tests did not aid in the diagnosis, since only two patients were diagnosed correctly prior to surgery. Only the liver scan proved of significant diagnostic value. All patients had lesions in the right hepatic lobe, and three patients also had lesions in the left lobe. The lesions were diagnosed as focal nodular hyperplasia in six patients and as hepatic adenomas in

three. Prodromal symptoms were vague--gnawing pain in the epigastric region and right upper quadrant unrelated to meals, position, or activity. One patient died following an emergency right total lobectomy; the remaining eight patients are alive and asymptomatic. No specific oral contraceptive could be implicated in the etiology of the tumors. (3 refs)

- 79-3761 Computer-assisted Structure-Activity Studies of Chemical Carcinogens. A Heterogeneous Data Set.** (Eng) Jurs, P. C. (Dept. Chemistry, Pennsylvania State Univ., University Park, PA 16802); Chou, J. T.; Yuan, M. *J Med Chem* 22(5): 476-483; 1979.

The structure-activity relationships of a series of heterogeneous organic compounds were studied to develop predictive ability for carcinogenic potential. The compounds were from more than 12 structural classes and they consisted of 130 carcinogens and 79 noncarcinogens. A set of 28 calculated molecular structure descriptors was identified that supported a linear discriminant function able to classify 192 compounds as carcinogens or noncarcinogens. A predictive ability of 90% for carcinogens and 78% for noncarcinogens was obtained in randomized testing. The results demonstrate that pattern-recognition methods can be used to analyze a diverse set of compounds, each of which is represented by calculated molecular structure descriptors for a common biological activity. Thus, pattern-recognition methods may be useful for predicting the carcinogenicity of chemical compounds. (36 refs)

- 79-3762 Biochemical and Ultrastructural Changes in Teleost Liver Following Subacute Exposure to PCB.** (Eng) Klaunig, J. E. (Comparative and Environmental Pathobiology Program, Dept. Pathology, Univ. Maryland Sch. Medicine, 31 South Green St., Baltimore, MD 21201); Lipsky, M. M.; Trump, B. F.; Hinton, D. E. *J Environ Pathol Toxicol* 2(4): 953-963; 1979.

The response of the channel catfish liver to subacute exposure to polychlorinated biphenyls (PCB's; Aroclor 1254: 1,000 mg/kg via gastric intubation) was evaluated electron microscopically and biochemically. Twenty-one days after exposure to PCB, the ratio of liver wt to body wt and the microsomal protein content did not differ from control values. However, cytochrome P-450 was elevated threefold, cytochrome b₅ twofold, aminopyrine demethylase activity threefold, and NADPH-cytochrome c reductase activity was increased by 50%-55% over that in control livers. The hepatocyte cytoplasm of the PCB-treated fish revealed changes primarily in the endoplasmic reticulum (ER). Structural alterations included an increase in tubular smooth ER and the formation of parallel stacks of smooth ER showing continuity with rough ER and membranous whorls. Based on the biochemical and morphological findings, it is concluded that the hepatic mixed-function oxidase system of the catfish was induced by subacute exposure to PCB. (35 refs)

- 79-3763 Studies on Metabolic Activation of Vinyl Chloride in *Drosophila melanogaster* after Pretreatment with Phenobarbital and Polychlorinated Biphenyls.** (Eng) Magnusson, J. (Environmental Toxicology Unit, Wallenberg Lab., Univ. Stockholm, S-106 91 Stockholm, Sweden); Hallstrom, I.; Ramel, C. *Chem Biol Interact* 24(3): 287-298; 1979.

The metabolic activation of vinyl chloride (VC) by the hepatic mixed-function oxygenase system was studied in *Drosophila melanogaster* by measuring the uptake of ¹⁴C from labeled VC in five different strains with and without pretreatment with phenobarbital or a polychlorinated biphenyl (PCB: Clophen A50). The latter compounds are well-known inducers of cytochrome P-450. In accordance with previous data on VC-induced sex-linked recessive lethals, pretreatment with inducers increased the uptake of labeled compound up to 10 times. There was, however, a marked difference in response among the five strains. In particular, the Hikone strain, known to be resistant to insecticides, had a comparatively higher initial uptake of VC than any of the other strains tested. However, this uptake was unaffected by phenobarbital, even at doses 10 times higher than those used with the other strains, and it was induced to only a very small degree by PCB. Crosses between Hikone and an inducible strain indicated essentially a dominance for the Hikone genotype. Tests of inducible strains showed the same response to phenobarbital by 2 hr old larvae and adult males and females. The use of dimethyl sulfoxide as a solvent decreased both the initial uptake of ¹⁴C and, particularly, the induction by PCB. The use of Tween 80 as an emulsifier did not have such an effect. The interstrain variation in metabolic activation and inducibility has to be considered for optimization of the use of *Drosophila* in mutagenicity testing. This variation also opens up new possibilities of analyzing the mixed-function oxygenase system biochemically and genetically. (26 refs)

See also:

- * (Rev.): 79-3601, 79-3602, 79-3603, 79-3604, 79-3605, 79-3606, 79-3607, 79-3608, 79-3609, 79-3610, 79-3611, 79-3612, 79-3613, 79-3614, 79-3615, 79-3616, 79-3617, 79-3618, 79-3619, 79-3620, 79-3621, 79-3622, 79-3623, 79-3641, 79-3642, 79-3652.
 * (Phys.): 79-3774.
 * (Viral): 79-3777, 79-3793, 79-3799, 79-3813, 79-3843, 79-3865, 79-3910, 79-3923, 79-3935, 79-3948, 79-3951, 79-3976.
 * (Immun.): 79-3986, 79-3988, 79-3989, 79-3990, 79-3997, 79-4006, 79-4009, 79-4010, 79-4014, 79-4101.
 * (Epid.-Biom.): 79-4124, 79-4132, 79-4134, 79-4136, 79-4137, 79-4139, 79-4140, 79-4141, 79-4145, 79-4146, 79-4149, 79-4151, 79-4154, 79-4156, 79-4162, 79-4163, 79-4165, 79-4166, 79-4167, 79-4169, 79-4171, 79-4172, 79-4177, 79-4180, 79-4182.

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- 79-3764 The Determination of ^{210}Po and ^{210}Bi in Human Urine by Direct Extraction on Nickel. (Eng) Helmkamp, R. W. (Dept. Radiation Biology and Biophysics, Univ. Rochester Sch. Medicine and Dentistry, Rochester, NY 14642); Bale, W. F.; Hrynyszyn, V. *Int J Appl Radiat Isot* 30(4): 237-246; 1979.

Recoveries of ^{210}Po and ^{210}Bi were determined after extraction from large volumes of 'spiked' hydrochloric acid or acidified urine on nickel foils at room (RT) and steam-bath (90-100 C) temperatures. Recoveries from HCl at 90-100 C and RT and from urine at 90-100 C were quantitative. The av recovery from urine at RT was 92.7%. Extraction of added ^{210}Po from HCl at RT and from acidified urine at 90-100 C and RT was less efficient on silver foils than on nickel foils. Recovery of ^{210}Bi on nickel from spiked HCl was quantitative at 90-100 C and at RT. Recovery from spiked urine at 90-100 C was 94.5%. Assuming that the detection efficiency of deposited ^{210}Po and ^{210}Bi was 50%, the av ^{210}Po and ^{210}Bi contents of three different pools of normal human urine at the time of excretion were 0.03 and 0.89 picocurie/liter, respectively. (23 refs)

- 79-3765 The Fate of U.V.-induced Lesions Affecting SCEs, Chromosome Aberrations and Survival of CHO Cells Arrested by Deprivation of Arginine. (Eng) MacRae, W. D. (Environmental Carcinogenesis Unit, B.C. Cancer Res. Center, Vancouver, B.C., Canada); MacKinnon, E. A.; Stich, H. F. *Chromosoma* 72(1): 15-22; 1979.

The fate of UV-induced lesions, as indicated by the induction of sister chromatid exchanges (SCE's) and chromosome aberrations, was examined in Chinese hamster ovary (CHO) cells in which the interval between the time of UV exposure and the onset of the S phase was varied. CHO cells were seeded at low density in arginine-deficient medium (ADM), allowed to undergo proliferative arrest, and then exposed to UV radiation (0, 20, 40, or 60 ergs/mm²). Some cultures were released by the addition of complete medium 24 and 48 hr after irradiation. UV irradiation 48 hr prior to release of the CHO cells from ADM induced fewer SCE's than exposure to UV 24 to 0 hr prior to release. The reduction in SCE levels with time in ADM after irradiation was consistent for each dose. At two UV doses that induced significant increases in the incidence of chromosome aberrations in ADM-arrested CHO cells (2 and 5 ergs/mm²), there was no significant difference in the chromosome aberration levels at 10 sampling times in ADM-arrested CHO cells that were irradiated either immediately or 48 hr prior to ADM release. A 48-hr postirradiation incubation in ADM did not increase cell survival.

These results indicate that a large proportion of UV-induced lesions responsible for the formation of SCE's in ADM-arrested CHO cells are modified or repaired during postirradiation incubation in ADM, but lesions resulting in chromosome aberrations are not. (25 refs)

- 79-3766 Increased Near-Ultraviolet Induced DNA Fragmentation in Xeroderma Pigmentosum Variants. (Eng) Netrawali, M. S. (Biochemistry and Food Technology Div., Bhabha Atomic Res. Centre, Trombay, Bombay 400085, India); Cerutti, P. A. *Biochem Biophys Res Commun* 87(3): 802-810; 1979.

The formation of strand breaks in parental DNA was studied in normal, excision-deficient xeroderma pigmentosum (XP), and XP variant (XPV) skin fibroblasts following exposure to near-UV at 313 nanometers. Irradiated cell suspensions were applied to polyvinyl chloride filters for cell lysis and alkaline elution. The fraction of DNA eluted after irradiation with 2.25 kilojoules/m² at 0 C was comparable for all cell groups. Only small increments were observed in the fraction eluted from the normal and XP cells when irradiation was applied at 37 C. However, large increases were detected for the XPV cells when the temperature was raised to 37 C. The time course of the rejoining of the strand breaks following irradiation at 37 C was studied. A return to the elution values of unirradiated control cultures was fastest for XP cells and was achieved within 30 min. It was slowest for XPV cells, for which elution rates were still significantly above those of controls 5 hr postirradiation. Three alternative interpretations of the results are given: (1) a late step in excision repair may be deficient in XPV cells; (2) a nuclease function involved in the removal of radiation lesions could be more active in XPV cells; or (3) a glycosylase function could be more active. Regardless of which interpretation is correct, the results indicate that the abnormality in the metabolism of damaged DNA in XPV cells affects parental DNA. (21 refs)

- 79-3767 The Mutagenic Potential of Unexcised Pyrimidine Dimers in *Saccharomyces cerevisiae*, *rad1-1*. Evidence from Photoreactivation and Pedigree Analysis. (Eng) Kilbey, B. J. (Div. Biological Sciences, Natl. Res. Council Canada, Sussex Drive, Ottawa, Canada); James, A. P. *Mutat Res* 60(2): 163-171; 1979.

The mutagenic potential of persistent pyrimidine dimers in the *Saccharomyces cerevisiae* excision-defective strain

rad1-1 was studied by photoreactivation (PR) and pedigree analysis. PR was applied at the conclusion of the first postirradiation cell division, and its effect on the frequency of first-generation, second-generation, and mixed-clone mutants was followed. UV induced few, if any, zero-generation mutants in *rad1-1*. The frequency of first-generation mutations per pedigree was increased by UV to 0.35 without PR and to 0.39 with PR. Second-generation mutations, however, were reduced to one-third of their frequency after UV if photoreactivating light was given just before the second cell division following UV. The frequency of mutations occurring at the third and subsequent generations was also lowered by PR. This indicates that the dimers responsible for them must have persisted for at least two generations. The frequency of mixed clones per sister-clone pair dropped from 0.50 after UV alone to 0.20 when UV was followed by PR. This drop is highly significant and indicates that the delayed mutants are traceable to persistent dimers. The majority of second-generation mutations, whether photoreactivable or not, seem to derive from photoproducts that initiated them at or after the second DNA replication to follow UV. The corollary is that mismatch repair in the first generation must be extremely efficient. The results also show that, at the UV dose used (3.2 joules/m²), the dimers in the DNA failed to impair mismatch correction. The obvious efficiency of mismatch repair may mean that errors are introduced not opposite but to one side of the noncoding lesion. (12 refs)

- 79-3768 Effect of Thymic Hormone on Radiation-induced Leukemia in the Mouse.** (Fre) Comsa, J. (Otorhinolaryngological Clinic, Saar Univ., D-6650 Homburg, Saarland, W. Germany); Baumann, B.; Zeppezauer, M.; Leonhardt, H.; Weber, N. *C R Acad Sci D (Paris)* 288(1): 185-187; 1979.

The effects of two different thymic extracts on leukemia induction by x-ray radiation (dose rate 16.5 rads/min, total dose 165 rads in 4 fractions at 8-day intervals, beginning at age 36-40 days) were studied in normal and thymectomized (Th-x) male and female C57BL mice. The animals were Th-x at age 28-33 days. A partially purified thymic extract (obtained by acid extraction, ammonium sulfate precipitation, and isoelectric precipitation) was administered sc at a dose of 100 guinea pig units/day starting the day after the last irradiation. Purified thymic hormone, obtained from the partially purified preparation by chromatography on Sephadex and hydroxyapatite, was administered under the same conditions in doses of 20-100 µg/day (activity: 1,000 guinea pig units/mg). The partially purified extract increased leukemia mortality from 0/25 in Th-x, irradiated mice that did not receive the extract to 10/25. Leukemia mortality in untreated and treated intact, irradiated mice was 23/25 and 24/25, respectively. The purified hormone caused a dose-dependent inhibition of leukemia induction: leukemia mortality was 23/25 in intact, irradiated mice treated with 20 µg/day, 3/25 after treatment with 60

µg/day, and 1/25 after treatment with 100 µg/day. The findings indicate that the partially purified thymic extract contains one or more components that facilitate leukemia induction by radiation and that these components are absent in the purified hormone. (12 refs)

- 79-3769 Hyperparathyroidism Induced by Radiation Therapy.** (Fre) Saint-Hillier, Y. (Service de Medecine V, Nephrologie, CHU Saint-Jacques, 2, place Saint-Jacques, 25030 Besancon, France); Hory, B.; Colomb, H.; Berthelay, S.; Dumoulin, G.; Pageaut, G.; Buet, L.; Ferganne, B.; Etievent, J. P.; Perol, C. *Sem Hop Paris* 55(7/8): 410-412; 1979.

Primary hyperparathyroidism due to a parathyroid adenoma was diagnosed in a 59-yr-old woman who had undergone neck irradiation (30 sessions) for Basedow's disease 38 yr earlier. The cause-effect relationship seems to be established in the light of animal experiments and similar literature data. (18 refs)

- 79-3770 Diagnostic Ultrasound: Effects on the DNA and Growth Patterns of Animal Cells.** (Eng) Liebeskind, D. (Dept. Radiology, Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY 10461); Bases, R.; Elequin, F.; Neubort, S.; Leifer, R.; Goldberg, R.; Koenigsberg, M. *Radiology* 131(1): 177-184; 1979.

In the DNA of HeLa cells, diagnostic levels of ultrasound at the G1 stage increased immunoreactivity to antinucleoside antibodies, strongly suggesting an unwinding of the helix or single-strand break induction. In addition, ultrasound produced low levels of nonsemiconservative synthesis in logarithmically growing cells treated with hydroxyurea, indicating repair synthesis. In the C3H mouse cell line 10T-1/2, Cl8, loss of contact inhibition with a criss-crossed growth pattern was seen. In one experiment, tumors developed in syngeneic mice at the site of injection of ultrasonically treated cells. (38 refs)

- 79-3771 Second Tumors in Childhood: A Contribution to Tumor Induction by Tumor Therapy.** (Ger) Hofmann, V. (Klinik für Kinderchirurgie, St. Barbara-Krankenhaus, Barbarastrasse 3-5, DDR-4020 Halle/Saale, E. Germany). *Z Kinderchir* 26(1): 1-15; 1979.

Data on the occurrence of second tumors in children irradiated for malignant tumors are reviewed, and six new cases are presented. The six children were subjected to radiotherapy (1,000-3,000 R, 10,000 R in 1 case) for malignant synovialoma, hemangioma (3 cases), Hodgkin's disease, and reticulum cell sarcoma at ages ranging from 6 to 113 mo. The second tumors (chondrosarcoma after

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synovialoma, adenocarcinoma of the parotid gland after 1 hemangioma, osteochondromas after hemangiomas in 2 cases and after Hodgkin's disease in 1, and leukemia after reticulum cell sarcoma) developed at intervals of 30-144 mo. (44 refs)

- 79-3772 Parathyroid Adenomas Following Irradiation.** (Eng) Russ, J. E. (Dept. Surgery, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., 6723 Bertner, Houston, TX 77030); Scanlon, E. F.; Sener, S. F. *Cancer* 43(3): 1078-1083; 1979.

A retrospective analysis was conducted to determine the incidence of previous head and neck irradiation in a consecutive group of 83 patients operated for parathyroid adenoma from 1971 through 1976. Of the 74 patients who were contacted, 19 (14 women, 5 men) had a history of previous irradiation of the neck. Only 8/101 individuals in the control group gave a positive history of irradiation. Age at exposure ranged from birth to 44 yr, and the mean latent period was 30 yr. Thyroid abnormalities, including four carcinomas and nine adenomas, were present in 13/19 irradiated patients. Sixteen of the 19 irradiated patients developed 22 other tumors within the radiation field, including tumors of the skin, breast, and parotid gland. Forty-seven percent of the irradiated group had malignant neoplasms within the irradiation field. The histopathology of the radiation-associated parathyroid adenomas was similar to that seen experimentally. Oxyphil cells were present and comprised almost half the adenoma in two instances. These results suggest that there is a strong positive correlation between radiotherapy to the neck and the subsequent development of parathyroid tumors. Whether the long-term effect of radiation is a direct one or a manifestation of systemic immunosuppression is not clear. (33 refs)

- 79-3773 Phagocytosis of Asbestos Fibers by Human Pulmonary Alveolar Macrophages.** (Eng) McLemore, T. (Dept. Biology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Corson, M.; Mace, M.; Arnott, M.; Jenkins, T.; Snodgrass, D.; Martin, R.; Wray, N.; Brinkley, B. R. *Cancer Lett* 6(4): 183-192; 1979.

Human pulmonary alveolar macrophages (PAM's) from eight healthy nonsmoking volunteers (19-29 yr old) were cultured for 24-72 hr with 1-300 $\mu\text{g}/\text{ml}$ amosite asbestos (AS: 5-100 μm in length), and phagocytosis of AS, cytotoxicity, and changes in cell-surface morphology were studied by light and scanning electron microscopy (SEM). At 100 $\mu\text{g}/\text{ml}$ AS, no significant cytotoxicity occurred during the first 24 hr of culture, but cell viability decreased significantly between 48 and 72 hr of culture. At higher concentrations, PAM viability decreased proportionately and significant cytotoxicity was observed after the initial 24 hr. SEM demonstrated that the cells were very heavily laden with AS fibers, and some fibers, too large to be completely phagocytized, were seen protruding through the PAM cytoplasm into the surrounding medium. Morphological changes observed in the PAM cytoplasmic membranes during phagocytosis of AS included an increase in cytoplasmic blebbing (zeiosis) and the appearance of a fiberlike material on the surface of AS fibers in the area of initial contact with the membrane. The biochemical structure of this fibrous material is not known. Continued investigation of the biological interactions between AS and human cells might provide information regarding the etiology of AS-related lung diseases. (13 refs)

- 79-3774 Increased Effectiveness of Hyperthermia in the Presence of Local Anesthetics (Meeting Abstract).** (Eng) Yatvin, M. B. (Dept. Human Oncology, Wisconsin Clinical Cancer Center, Univ. Wisconsin Medical Sch., Madison, WI 53792); Clifton, K. H.; Dennis, W. H. *Proc Am Assoc Cancer Res* 20: 43; 1979 (no refs)

See also:

- *(Rev.): 79-3609, 79-3623, 79-3624, 79-3625, 79-3626, 79-3652.
*(Chem.): 79-3668, 79-3670, 79-3691, 79-3711, 79-3750, 79-3948, 79-3951.
*(Immun.): 79-3984.
*(Epid.-Biom.): 79-4108, 79-4110, 79-4111, 79-4112, 79-4113, 79-4114, 79-4115, 79-4118, 79-4123, 79-4125, 79-4132, 79-4133, 79-4135, 79-4142, 79-4151, 79-4155, 79-4160, 79-4161, 79-4173, 79-4174, 79-4176, 79-4177.

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- 79-3775** Initiation of DNA Synthesis by the Avian Retrovirus Reverse Transcriptase In Vitro: Nature and Location of the Oligodeoxycytidylic Acid Primer Binding Site. (Eng) Collett, M. S. (Dept. Pathology, Univ. Colorado Medical Sch., Denver, CO 80262); Perdue, M. L.; Faras, A. J. *J Virol* 30(1): 319-326; 1979.

The use of oligodeoxycytidylic acid [oligo(dC)] as a primer for the initiation of DNA synthesis by the avian retrovirus reverse transcriptase in vitro was investigated employing the viral RNA genome as template. The addition of oligo(dC)12-18 to viral 35S RNA resulted in a stimulation of DNA synthesis by the viral RNA-directed DNA polymerase comparable to that observed when oligo(dT) was employed as a primer. Under similar conditions, neither oligo(dA)12-18 nor oligo(dG)12-18 was active as a primer for transcription of the avian retrovirus genome. Several different approaches were used to localize the oligo(dC)12-18 binding site on the viral genome, including isolation of poly(A)-containing fragments, competition hybridization, and RNase H hydrolysis. These analyses indicated that oligo(dC)12-18 binds to a site approx 2,000 to 3,000 nucleotides from the 3' terminus of the genome of transforming strains of avian sarcoma viruses and approx 700 to 1,000 nucleotides from the 3' terminus of non-transforming avian retroviruses. Therefore, the major site of initiation of DNA synthesis by oligo(dC)12-18 appears to be in the vicinity of the 3' end of the *env* gene and the 5' end of the *src* gene, although the presence of minor initiation sites located elsewhere on the viral genome cannot be excluded by these data. Characterization of oligonucleotides after pancreatic RNase hydrolysis and poly(C)-Sepharose chromatography of viral RNA directly demonstrated the presence of oligoguanilyc acid residues in the avian sarcoma virus genome. DNA sequences transcribed from the oligo(dC) primer appeared to be conserved in all of the avian leukosis-sarcoma viruses tested. Oligo(dC) may be useful in the production of specific complementary DNA probes. (27 refs)

- 79-3776** Biochemical Characterization of the Type C Retrovirus Associated with Lymphoproliferative Disease of Turkeys. (Eng) Yaniv, A. (Dept. Human Microbiology, Sackler Sch. Medicine, Tel-Aviv Univ., Tel-Aviv, Israel); Gazit, A.; Ianculescu, M.; Perk, K.; Aizenberg, B.; Zimber, A. *J Virol* 30(1): 351-357; 1979.

The first report of the biochemical features of the C-type virus associated with lymphoproliferative disease (LPD) of turkeys is presented. Turkeys inoculated with spleen ex-

tracts from LPD-affected birds developed viremia and then typical LPD lesions. Electron microscopy and biochemical characterization established that the virus present in the blood of infected turkeys is a C-type retrovirus. The viral particles possessed a buoyant density of 1.17 g/ml in sucrose gradients. They contained high-mol-wt RNA and an RNA-directed DNA polymerase with efficient exogenous and endogenous activity. The LPD virus polymerase was preferentially activated by magnesium ions. Cross nucleic acid hybridization assays revealed no sequence homology between the viral genome of LPD and avian myeloblastosis virus or reticuloendotheliosis virus, indicating that the LPD virus belongs to a distinct group unrelated to the avian leukosis-sarcoma virus complex or to the reticuloendotheliosis virus group. (20 refs)

- 79-3777** Chemical Activation and Regulation of a C-Type Virus from Ring-Necked Pheasant Cells. (Eng) Tereba, A. (Div. Virology, St. Jude Children's Res. Hosp., 332 N. Lauderdale, P.O. Box 318, Memphis, TN 38101). *Virology* 93(2): 340-347; 1979.

To better understand the mechanisms involved in transcriptional regulation and internal cell restrictions against virus infection, the growth properties of a chemically induced, endogenous C-type virus from ring-necked pheasant (RNP) fibroblasts were studied. 20-Methylcholanthrene was used to induce the permanent expression of nondefective C-type virus in the fibroblasts. This chemical induction demonstrated that non-virus-producing RNP cells are unable to produce virus because of a regulatory mechanism and not because they lack a complete virus genome. The induced virus infected and grew to moderate titers in untreated RNP fibroblasts, demonstrating that it had permanently overcome the growth restriction mechanism. The virus also grew efficiently in several lines of chicken fibroblasts that restrict A, B, D, and E subgroup oncoviruses. However, it did not grow efficiently, if at all, in fibroblasts from a variety of other avian species that lack an endogenous Rous-associated virus type O-like virus. The restricted growth in these heterologous avian species is not due to a membrane host range restriction, because pseudotypes of a Rous sarcoma virus with a deleted glycoprotein gene and the induced RNP virus can infect the fibroblasts of all avian species tested equally well. This selectivity thus suggests that there may be a species-specific intracellular restriction mechanism against infecting endogenous virions derived from evolutionarily divergent hosts. (19 refs)

- 79-3778** Construction and Characterization of a Plasmid Containing a Nearly Full-Size DNA

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Copy of Bacteriophage MS2 RNA. (Eng) Devos, R. (Lab. Molecular Biology, State Univ. Ghent, Ledeganckstraat, 35, B-9000 Ghent, Belgium); van Emmelo, J.; Contreras, R.; Fiers, W. *J Mol Biol* 128(4): 595-619; 1979.

An apparently full-length complementary DNA (cDNA) copy of in vitro-polyadenylated MS2 RNA was synthesized with avian myeloblastosis virus RNA-dependent DNA polymerase. After the MS2 RNA template was removed from the cDNA strand with T₁ and pancreatic RNase digestion, the cDNA was a good template for the synthesis of double-stranded MS2 DNA with *Escherichia coli* DNA polymerase I. Molecular chimeras were constructed by inserting the double-stranded MS2 DNA into the *Pst*I restriction endonuclease cleavage site of the *E. coli* plasmid pBR322 by means of the poly(dA)-poly(dT) tailing procedure. MS2 RNA was compared with that of pMS2-7, an *E. coli* transformant carrying a plasmid with a nearly full-length MS2 DNA insertion. The complete 3'-terminal sequence derived from the viral genome was present in pMS2-7; the transformant lacked only 14 nucleotides at the extreme 5' end of MS2 RNA. The approx length of the dA.dT sequence at the 5' end of MS2 DNA in pMS2-7 was about 110 base pairs, which is shorter than the length of homopolymer linker (150 base pairs) connecting the 3' end of the MS2 DNA to the plasmid DNA. An extra DNA insertion of about 800 base pairs in this region was identified as the translocatable element insertion sequence 1. There was no evidence for any misincorporation during the reverse transcriptase copying processes. (36 refs)

79-3779 Mutagenesis In Vitro by DNA Polymerase from an RNA Tumour Virus. (Eng)

Gopinathan, K. P. (Microbiology and Cell Biology Lab., Indian Inst. Science, Bangalore 560 012, India); Weymouth, L. A.; Kunkel, T. A.; Loeb, L. A. *Nature* 278(5707): 857-859; 1979.

The in vitro mutagenicity of an avian myeloblastosis virus (AMV) DNA polymerase that has a high frequency of misincorporation with synthetic polynucleotides was studied with the use of single-stranded circular DNA from bacteriophage Φ X174 as template. This DNA contains one nucleotide substitution mutation (*am3*) located in the overlapping genes *D* and *E*. Φ X *am3* DNA was copied by homogeneous AMV DNA polymerase and then used to infect *Escherichia coli* spheroplasts. The infected spheroplasts were plated onto indicator bacteria permissive or nonpermissive for *am3*. The results indicated that copying by AMV DNA polymerase led to a significantly increased frequency of back mutations (revertants) on the Φ X DNA. The calculated error rates of DNA synthesis past the *am3* mutation by AMV DNA polymerase averaged 1 in 904 in six determinations. In view of the fact that some molecules of the template remain uncopied, the error rates could have been underestimated by as much as 30%. The error rates were similar to those observed with

homopolymer templates. The results support the hypothesis that DNA polymerases from oncogenic viruses copy host DNA inaccurately, causing mutations and initiating tumorigenesis. (21 refs)

79-3780 Detection of Reverse Transcriptase Activity in Human Cells. (Eng) Kiessling, A. A. (Dept. Medicine, Sch. Medicine, Univ. California, San Diego, La Jolla, CA 92093); Gouljian, M. *Cancer Res* 39(6, part 1): 2062-2069; 1979.

Leukemic cells from seven patients with acute myelomonocytic leukemia and acute or chronic myelogenous leukemia were examined for DNA polymerase activity corresponding to RNA tumor virus reverse transcriptase. Control cells used for comparison with the leukemic cells were HeLa cells, a cultured lymphocyte cell line, and normal placenta and spleen cells. Animal cells infected with avian myeloblastosis virus or murine leukemia virus (MuLV) provided the basis for cell fraction procedures, and reconstituted MuLV particles added to human cells established a threshold of virus detection by enzyme assay at 1-10 particles/cell. DNA polymerase activity with some properties similar to a reverse transcriptase was detected in some of the human leukemic cells. However, parallel analyses of nonmalignant cells showed sufficient similarities to raise serious questions about the specificity of the criteria. Reverse transcriptase activity has been reported to be present in WBC from a proportion of leukemia cases. However, it is concluded that the usual enzymatic criteria based on the use of synthetic template primers are not sufficient to identify a DNA polymerase activity as viral reverse transcriptase. (48 refs)

79-3781 Biological Techniques for Avian Sarcoma Viruses. (Eng) Hunter, E. (Dept. Microbiology, Medical Center, Univ. Alabama in Birmingham, Birmingham, AL 35294). *Methods Enzymol* 48: 379-393; 1979.

A quantitative and reproducible Rous sarcoma virus (RSV) assay, the focus assay, is described together with methods for obtaining cloned RSV preparations through both the focus and soft-agar-colony assays. The assays are carried out with the use of secondary chick embryo cultures. (21 refs)

79-3782 Large-Scale Growth of Rous Sarcoma Virus. (Eng) Smith, R. E. (Dept. Microbiology, Duke Univ. Medical Center, Durham, NC 27710). *Methods Enzymol* 48: 393-403; 1979.

Techniques for the growth of transformed cells are described, and procedures for preparing large quantities of

purified Rous sarcoma virus are outlined. Virus-producing chick embryo fibroblasts were cultured in roller culture bottles. (13 refs)

- 79-3783** Product of In Vitro Translation of the Rous Sarcoma Virus *src* Gene Has Protein Kinase Activity. (Eng) Sefton, B. M. (Tumor Virology Lab., The Salk Inst., San Diego, CA 92112); Hunter, T.; Beemon, K. *J Virol* 30(1): 311-318; 1979.

An attempt was made to exclude the possibility that the protein kinase (PK) activity associated with immunoprecipitated p60src, the 60,000-dalton polypeptide product of the Rous sarcoma virus (RSV) *src* gene, is due to an adventitiously bound cellular enzyme present in transformed cells. Immunoprecipitates that contain p60src synthesized in vitro in a rabbit reticulocyte lysate were prepared. In vitro translation of RSV virion RNA in the nuclease-treated reticulocyte lysate resulted in the synthesis of a PK that, when immunoprecipitated with antitumor serum, phosphorylated the immunoglobulin heavy chain. Even though in vitro translation of virion RNA resulted in the synthesis of several polypeptides that were recognized by the antitumor serum, control experiments demonstrated that an immunoprecipitable PK activity was found only when an immunoprecipitable p60src was synthesized. A PK with similar properties was therefore intimately associated with the p60src synthesized in vitro in the reticulocyte lysate, just as it is with the p60src obtained from transformed chick and mammalian cells. Because the immunoprecipitation of p60src synthesized in the rabbit reticulocyte lysate or in transformed chick and mammalian cells results in the precipitation of PK's having apparently identical properties, it is highly unlikely that this association is artifactual. In vitro translation of the RNA of ts NY68 (a temperature-sensitive RSV mutant able to transform cells at 36 but not 41 C) at 30 C resulted in efficient synthesis of immunoprecipitable p60src, but very inefficient synthesis of an immunoprecipitable PK. The p60src obtained by in vitro translation of wild-type virion RNA was >20-fold more active as a PK than the p60src obtained from ts NY68 RNA. The correlation in the case of ts NY68 of a deficiency in PK activity with an inability to transform cells at high temperature suggests that the PK activity associated with p60src is critical to cellular transformation. (14 refs)

- 79-3784** Regulation of DNA Replication by Serum and the Transforming Function in Cultured Rat Fibroblasts Transformed by Rous Sarcoma Virus. (Eng) Magun, B. E. (Dept. Anatomy, Univ. Arizona Health Sciences Center, Tucson, AZ 85724); Thompson, R. L.; Gerner, E. W. *J Cell Physiol* 99(2): 207-216; 1979.

The onset and rate of semiconservative DNA replication

were measured in stimulated cultured rat fibroblasts (Rat-1) and in derivatives transformed by wild-type [Rat-1 (wt/RSV)] and temperature-sensitive [Rat-1 (tsLA24/RSV)] Rous sarcoma virus (RSV) after a period of serum deprivation. After 54 hr of serum deprivation, all cultures had the general characteristics of a nondividing state, although the Rat-1 (wt/RSV) cells showed more mitotic activity during deprivation than the other cultures. The Rat-1 (tsLA24/RSV) cells initiated DNA synthesis following a shift to the permissive temperature or upon addition of serum at the nonpermissive temperature. Their rate of DNA replication was unaffected by the presence of serum at the permissive temperature, although there was a serum requirement at the nonpermissive temperature. The transition probability was less at the permissive temperature than in the presence of serum at the nonpermissive temperature. The amount of DNA induced to replicate by the addition of serum at the nonpermissive temperature or by a shift to the permissive temperature was similar. The rate of entry into the S phase was always lower in Rat-1 (wt/RSV) cells than in Rat-1 cells at both 39 and 35 C. The data suggest that the transforming function initiates a process that acts at the level of commitment to DNA replication, which may render normal serum-related control mechanisms ineffective in the regulation of growth. (30 refs)

- 79-3785** Transformation-defective Mutants of Rous Sarcoma Virus with Longer Sizes of Genome RNA and Their Highly Frequent Occurrences. (Eng) Yoshida, M. (Dept. Viral Oncology, Cancer Inst., Kami-Ikebukuro, Toshima-ku, Tokyo 170, Japan); Yamashita, M.; Nomoto, A. *J Virol* 30(2): 453-461; 1979.

Transformation-defective (td) mutants with different sizes of genomic RNA were isolated from the Prague strain of Rous sarcoma virus, subgroup C (PR-C). Six mutants isolated from one stock of UV-irradiated PR-C, TYPR-C, all belonged to subgroup C and were designated tdTY4-9. All were deletion mutants and had an RNA that was slightly larger than the class b RNA of most td avian sarcoma virus mutants. Two td mutants isolated from another stock of PR-C, LAPR-C, contained class b RNA's that were shorter than those of tdPR-C. Oligonucleotide fingerprint analysis indicated that tdTY9 and tdLAPR-C were deletion mutants derived from the respective parental strains and that the RNA sequences of the two parental strains were different. The td mutants isolated from another clone of TYPR-C had RNA of the same size as that of tdTY9 and fingerprints identical to those of tdLAPR-C. The results suggest that differences in the nucleotide sequences of the RNA's of TYPR-C and LAPR-C may be the cause of differences in deletion size. (17 refs)

- 79-3786** A Nonconditional Replication-defective Mutant of the Schmidt-Ruppin Strain of Rous

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Sarcoma Virus. (Eng) Vogt, P. K. (Dept. Microbiology, Univ. Southern California, Sch. Medicine, Los Angeles, CA 90033); Hayman, M.; Hunter, E.; Duesberg, P. H. *Virology* 92(2): 285-290; 1979.

The properties of LA7365, a nonconditional mutant of the Schmidt-Ruppin strain of Rous sarcoma virus (subgroup A) that fails to synthesize infectious progeny virus, are described. Cultures of nonproducing chick embryo fibroblasts transformed by LA7365 contained small amounts (about 1/50 the quantity recovered from wild-type-producing controls) of particles with the hydrodynamic properties of retroviruses. RNA extracted from these noninfectious particles was the same size as the RNA of nondefective sarcoma virus. A series of rescue experiments with LA7365 nonproducer cells demonstrated that the cells have functional viral *src* and *env* genes and that the defect resides in the *gag* and/or *pol* gene. LA7365 could not be complemented by reticuloendotheliosis virus (REV), amphotropic murine leukemia virus, or LA3342, a presumptive temperature-sensitive *gag* mutant. Pulse-chase experiments showed that the proteolytic processing of the *gag* precursor pr76 is strongly inhibited in mutant-infected cells. It is concluded that LA7365 is probably a point mutation and that the genetic lesion interfering with the replication of LA7365 resides in the *gag* gene. It is not clear whether LA7365 carries an additional lesion in the *pol* gene. (27 refs)

79-3787 Avian Sarcoma Virus-transformed Quail Clones Defective in the Production of Focus-forming Virus. (Eng) Mason, W. S. (Inst. Cancer Res., Fox Chase Center, Philadelphia, PA 19111); Hsu, T. W.; Yeater, C.; Sabran, J. L.; Mark, G. E.; Kaji, A.; Taylor, J. M. *J Virol* 30(1): 132-140; 1979.

Japanese quail embryo fibroblasts were infected at low multiplicity with avian sarcoma virus, and transformed cells were selected by their ability to form colonies in agar. Five clones that failed to produce focus-forming virus were examined for (1) intactness of the integrated proviral DNA, (2) intracellular viral RNA production, (3) intracellular viral antigen production, (4) production of virus particles, and (5) rescue of a functional *src* gene and of parental host range determinants by superinfection with Rous-associated virus-60, an avian leukosis virus of subgroup E. In three of the five nonproducer clones, restriction enzyme analysis revealed that the defect is a consequence of a deletion in the viral genome. The mechanism of the formation of these deletions is unknown. In one clone producing focus-forming virus, analysis of the integrated viral DNA revealed an insertion in the region of the genome that codes for *src*. This insertion is sufficient to code for an additional 80 amino acids. A common feature of the defective clones was found to be the maintenance of a large terminal repeat of at least 300 base pairs at each end of the integrated viral DNA. This maintenance is consistent with the proposed

importance of a large terminal repeat in the initiation and termination of transcription of integrated viral DNA into RNA. (39 refs)

79-3788 Polymorphism of Avian Sarcoma Virus *src* Proteins. (Eng) Beemon, K. (Tumor Virology Lab., Salk Inst., San Diego, CA 92112); Hunter, T.; Sef-ton, B. M. *J Virol* 30(1): 190-200; 1979.

The *src* gene products of seven different avian sarcoma viruses (ASV's) were compared. In vitro translation of virion RNA yielded products identified unambiguously as p60*src* in the case of two stocks of the Schmidt-Ruppin strain, three stocks of the Prague strain, the Bryan strain, and the Bratislava 77 strain of ASV. Differences in the electrophoretic mobility of these seven p60*src* proteins in sodium dodecyl sulfate-polyacrylamide gels, corresponding to variations in apparent mol wts ranging from 56,000 to 60,500, were observed. Antigenic variability was also found; only 3/7 viruses tested encoded a p60*src*, which was precipitated by antisera derived from rabbits bearing tumors induced by the Schmidt-Ruppin strain of Rous sarcoma virus. Examination of the methionine-containing tryptic peptides of the seven p60*src* proteins by two-dimensional mapping revealed four common peptides but marked variability in the five to eight other peptides in each protein. Clear differences in the peptide maps of p60*src* were observed, both between different strains of virus and within strains. In the three cases examined, p60*src* synthesized in transformed cells was essentially identical to that synthesized in vitro. It is concluded that there is significant polymorphism in the p60*src* proteins of the ASV's. (27 refs)

79-3789 Avian Oncovirus MH2 is Defective in *Gag*, *Pol*, and *Env*. (Eng) Hu, S. S. (Dept. Microbiology, Univ. Southern California, Sch. Medicine, Los Angeles, CA 90033); Vogt, P. K. *Virology* 92(2): 278-284; 1979.

The defectiveness of MH2, an avian C-type oncovirus, was investigated by genetic experiments and by tryptic peptide mapping. A series of rescue and complementation experiments indicated that MH2 is defective in *env*, *pol*, and *gag* genes. The *gag* and *pol* defects are demonstrated by the failure of MH2 to complement the respective temperature-sensitive mutants of avian sarcoma viruses. These defects are also suggested by the inability of unrelated helper viruses and chick helper factor to rescue MH2. The *env* defect is documented by the lack of complementation between Rous sarcoma virus and MH2, by the absence of interference in MH2 nonproducer cells against challenge infection with avian leukosis viruses of various subgroups, and by the identity of the type-specific envelope properties of MH2 with those of its helper virus. The polyprotein

MH2 p100, which is made in MH2 nonproducer cells, contains the tryptic peptides of *gag* proteins p19 and p27. The replication defects in *gag*, *pol*, and *env* are probably the result of extensive genetic deletions. The 5' end of the genome is apparently retained and translated into the p19 and p27 components of the MH2 p100 protein. (36 refs)

- 79-3790 Uninfected Vertebrate Cells Contain a Protein That Is Closely Related to the Product of the Avian Sarcoma Virus Transforming Gene (*src*).** (Eng) Oppermann, H. (Dept. Microbiology and Immunology, Univ. California, San Francisco, CA 94143); Levinson, A. D.; Varmus, H. E.; Levintow, L.; Bishop, J. M. *Proc Natl Acad Sci USA* 76(4): 1804-1808; 1979.

The neoplastic transformation of cells by avian sarcoma virus is mediated by a single viral gene (*src*) that encodes a phosphoprotein (pp60-*src*) with the enzymatic activity of a protein kinase. The DNA's of vertebrate species contain a highly conserved homolog of *src* that is also represented in the polysomal RNA of uninfected cells and, hence, may specify a normal cellular protein. Rabbit antisera directed against pp60-*src* was used to isolate a closely related phosphoprotein (denoted vertebrate pp60) from uninfected chicken, quail, rat, and human cells. The data indicate that vertebrate pp60 is a homolog of pp60-*src* that is highly conserved antigenically, chemically, and functionally. Moreover, the cellular protein may possess protein kinase activity similar to that associated with pp60-*src*. It is concluded that the product of *src* is a slightly modified analog of a normal cellular protein. (20 refs)

- 79-3791 Genesis of a Virus-Transforming Gene.** (Eng) Bishop, J. M. (Dept. Microbiology, Univ. California, San Francisco, CA 94143); Baker, B.; Fujita, D.; McCombe, P.; Sheiness, D.; Smith, K.; Spector, D. H.; Stehelin, D.; Varmus, H. E. *Natl Cancer Inst Monogr* (48): 219-223; 1978.

Molecular hybridization was used to analyze the origins of the *src* gene of avian sarcoma viruses (ASV). Nucleotide sequences complementary to DNA encoding *src* (cDNA-*src*) were present in the genomes of ASV, but not in those of other avian viruses or adeno-, papova-, or murine viruses. Denatured cellular DNA from avian species formed duplexes with cDNA-*src*, the extent of duplex formation and the thermal stabilities of the duplexes being roughly a function of phylogenetic distance from chickens. More stable duplexes were formed between cDNA-*src* and DNA from rat cells transformed by ASV. RNA from a variety of avian tissues also hybridized with cDNA-*src*, much of the *src* RNA in the normal cells being present in polyribosomes. Among avian DNA's tested, only chicken DNA showed appreciable homology with the *env* gene of the endogenous Rous-associated virus (type O) of normal

chicken cells. Endogenous viral genes and cellular *src* were not coordinately transcribed. The results indicate that, in the chicken, cellular *src* and the endogenous viral genes reside in separate genetic elements. It is further concluded that the viral *src* gene was probably derived from a set of nucleotide sequences that were generated during the emergence of vertebrates and were highly conserved during subsequent evolution. (16 refs)

- 79-3792 Elongation of DNA Complementary to the 5' End of the Avian Sarcoma Virus Genome by the Virion-Associated RNA-Dependent DNA Polymerase.** (Eng) Novak, U. (Max-Planck-Institut für Molekulare Genetik, K-1000 Berlin 33, W. Germany); Friedrich, R.; Moelling, K. *J Virol* 30(2): 438-452; 1979.

The effect of detergent concentration (DC) on the elongation of DNA complementary to the 5' end of the avian sarcoma virus genome (cDNA₁₀₀) by the virion-associated RNA-dependent DNA polymerase was studied. The cDNA₁₀₀ showed continuous growth toward longer DNA at optimal concentrations of Triton X-100; higher DC's retarded the elongation process. Elongation in the presence of the optimal DC was inhibited drastically by actinomycin D, and the resulting double-stranded DNA contained little or no hairpin material. Numerous hairpin structures were found in DNA synthesized at other than optimum DC. The DNA made at the optimal DC had far less chemical complexity than the DNA made at high DC's. The nature of the elongation process was elucidated by analysis of DNA synthesized in a virion-associated reaction in the presence of bacteriophage Q β RNA. At the optimal DC, DNA complementary only to avian sarcoma virus RNA was synthesized. At higher concentrations, DNA was copied from both avian sarcoma virus and Q β RNA. It is concluded that elongation is unspecific at higher than optimal DC's and that factors other than DNA polymerase are involved in the elongation of cDNA₁₀₀. (58 refs)

- 79-3793 Increased Pathogenicity of Avian Sarcoma Virus B77 in Cyclophosphamide Treated Chickens.** (Eng) Smida, J. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia); Smidova, V. *Neoplasma* 25(6): 659-665; 1978.

Sarcoma genesis was studied in cyclophosphamide (CP)-treated and untreated (control) White Leghorn chickens inoculated with avian sarcoma virus B77 (B77V). CP treatment comprised 5 im inoculations of 100 mg/kg/day on 5 consecutive days or on every other day. All birds developed sarcomas after similar latent periods. However, the tumors regressed in 70%-86% of the control birds, whereas all drug-treated birds died of progressively growing sarcomas. The sarcoma mortality rate was significantly higher and the meantime until death significantly shorter in the CP-treated

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chickens. The incidence of visceral metastasis was solely a function of time in all infected birds. The sarcoma incidence and mortality rates were not affected by different treatment schedules, and no cytostatic effect of CP was observed. CP-treated chickens were more sensitive to virus infection following transplantation of B77V-infected rat and mouse cells than infected birds. Progressive tumor growth was observed in all drug-treated chickens. CP treatment improved the rescuability of B77V from the transformed virogenic rodent cells *in vivo*. (20 refs)

- 79-3794 Avian Reticuloendotheliosis Viruses: Evolutionary Linkage with Mammalian Type C Retroviruses.** (Eng) Barbacid, M. (Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014); Hunter, E.; Aaronson, S. A. *J Virol* 30(2): 508-514; 1979.

Radioimmunology was used to study the evolutionary relationship of the reticuloendotheliosis virus (REV) group to retroviruses of mammalian origin. A very high degree of antigenic relatedness was demonstrated among the major structural proteins of the REV group. Type-specific determinants in an REV-A (the nontransforming associated helper virus from REV strain T) low-mol-wt protein made it possible to distinguish REV-A from other members of the REV group. A limited degree of immunologic relatedness was found to exist between the p30 of REV and those of known mammalian C-type retroviruses. Most of the antigenic determinants shared by the REV-A and Rauscher murine leukemia virus p30's were present in all of the mammalian C-type retroviruses tested. However, there was no immunologic cross-reactivity of avian C-type and Rous sarcoma virus or mammalian B- or D-type retroviruses. The other members of the REV group exhibited complete competition curves when tested as competing antigens in the anti-Rauscher MuLV p30-^{125I}-labeled REV p30 assay. Interspecies radioimmunoassays firmly established the major structural proteins of the REV group as mammalian C-type virus in origin. However, it was not possible to assign any of the well-characterized mammalian C-type viral isolates as the direct progenitor of the avian REV group. (43 refs)

- 79-3795 Transcription of the Marek's Disease Virus Genome in Virus-induced Tumors.** (Eng) Silver, S. (Lab. Molecular Virology, Life Sciences, Inc., St. Petersburg, FL 33710); Smith, M.; Nonoyama, M. *J Virol* 30(1): 84-89; 1979.

Transcription of the Marek's disease virus (MDV) genome in tumor tissues from MDV-infected LSI-SPF chickens was studied by analyzing the hybridization kinetics of ³H-labeled MDV DNA with unlabeled RNA extracted from these tissues. Lymphoid tumors of ovary, spleen, liver, and kidney contained MDV genomes, but the virus-specific

RNA sequences were transcribed from <15% of the viral DNA. The virus nonproductive lymphoblastoid cell line MKT-1, established from a kidney lymphoma, contains 15 MDV genomes per cell. In these cells, 12%-14% of the viral DNA was transcribed. Thus, transcription of the MDV genome was restricted both in tumor tissues and in the MKT-1 cells. A hybridization experiment in which RNA extracted from MKT-1 cells and RNA extracted from a spleen tumor were mixed and hybridized to ³H-labeled MDV DNA indicated that the virus-specific RNA's from the two sources were encoded by the same DNA sequences. The polyribosomal fractions of MKT-1 cells and this spleen tumor contained only a portion of the virus-specific RNA sequences found in whole cell extracts, indicating the existence of a posttranscriptional control mechanism that prevents the transfer of certain viral RNA transcripts to the polyribosomes. The data suggest that the repressed expression of the viral genome in lymphoid tumor tissues and MKT-1 cells may be the result of precise controls within the cell at the transcriptional and posttranscriptional levels. (19 refs)

- 79-3796 Morphologic Characterization of Proliferative Cells and Virus Particles in Turkeys with Lymphoproliferative Disease.** (Eng) Perk, K. (Dept. Animal Science, Hebrew Univ. Jerusalem, Rehovot Campus, Rehovot, P.O. 12, Israel); Ianculescu, M.; Yaniv, A.; Zimber, A. *J Natl Cancer Inst* 62(6): 1483-1487; 1979.

Tissue samples from seven dead or moribund turkeys with spontaneous lymphoproliferative disease (LPD) were examined to determine the ultrastructure of the proliferating cells and the viral particles associated with the disease. Histologically, the tumors consisted of pleomorphic cells of the lymphoid series that appeared as cords or nests and may have shown a tendency toward a nodular architecture. Areas with randomly distributed proliferative cells were also seen. The proliferating lymphoid cells were larger than normal turkey lymphocytes and exhibited a much higher cytoplasm:nucleus ratio. They and their nuclei were irregularly shaped, and the cells had pseudopodal extensions. Nuclear bodies, rough endoplasmic reticulum, mitochondria, vacuoles, and lipid droplets were observed. The giant tumor cells had ultrastructural features similar to those of the other proliferating cells. Virus particles that were morphologically similar to murine oncornavirus particles were detected in tissues involved by tumor. Particles resembling intracytoplasmic A particles and herpesvirus particles were found in some macrophages in the proliferating lesions. Tissues from 15 normal specimens showed no C or A particles, but herpesvirus particles were identified in the livers of two normal turkeys. (12 refs)

- 79-3797 Characterization of EV-2, a Virus Isolated from European Eels (*Anguilla anguilla*) with**

Stomatopapilloma. (Eng) Nagabayashi, T. (Kitasato Univ. Sch. Fisheries Sci., Sanriku-cho, Kesen-Gun, Iwate Prefecture, 022-01, Japan); Wolf, K. *J Virol* 30(1): 358-364; 1979.

EV-2 virus, a virus isolated from the external tumor tissue and internal organs of European eels (*Anguilla anguilla*) with stomatopapilloma, was characterized. The virus was found to contain RNA, and it was inactivated by exposure to chloroform, ether, acid pH (3.0), and temperatures >4 C. Concentrated preparations of virus agglutinated chicken and sheep RBC. The addition of actinomycin D to FHM cells during the first 2.75 hr of infection inhibited viral replication. The buoyant density of the virus is 1.19 g/ml in sucrose gradients. EV-2 has a moderately pleomorphic spherical morphology, and its diameter ranges from 80 to 140 nanometers (nm). The virion has narrow, regularly spaced surface projections about 10 nm long. Replication studies in FHM cells at 15 C showed that new infectivity appeared at 10 hr postinfection and reached a plateau at 20 hr. Cytopathic effects consisted of cell fusion, syncytia, and irregularly rounded cell masses. Viral antigen was detected in the cytoplasm of infected cells by specific immunofluorescence. (19 refs)

79-3798 Serological Identification of Viral and Virus-related Antigens on DBA/2 Mouse Leukemia Lymphocytes. (Eng) Steuden, I. (Dept. Tumor Immunology, Inst. Immunology and Experimental Therapy, Polish Acad. Sciences, 53-114 Wroclaw, Poland); Radzikowski, C.; Zak-Nejmark, T. *Arch Immunol Ther Exp (Warsz)* 27(1/2): 187-207; 1979.

Sera active against various antigenic components of leukemic L1210 cells were obtained from various inbred mouse strains, and the cell-surface antigens were identified and their relationships examined. Antisera against mammary leukemia (ML) antigen produced in allogeneic and semisynthetic systems did not react in the cytotoxicity test with Gross-positive indicator cells of spontaneous AKR mouse leukemia and LBN/b-3 leukemia cells. However, these antisera were strongly positive in the indirect immunofluorescence (IMF) test against L1210 cells and spontaneous leukemia cells of AKR mice. The presence of Gross cell-surface antigen (GCSA) was confirmed by a positive surface IMF test using anti-Gross reference sera. Comparison of the activity of the sera in the cytotoxicity and IMF tests and cross absorption experiments demonstrated an independent localization of the ML and GCSA antigens on the surface of the L1210 leukemia cells. The ability of leukemia L1210 cells to absorb activity from anti-ML sera and the reaction between anti-ML sera and isolated B particles of mouse mammary tumor virus (MMTV) upon immunoprecipitation indicated the probable existence of an antigenic component of MMTV within the ML antigen. (47 refs)

79-3799 Eukaryotic Gene Regulation Studies with Mouse Mammary Tumor Virus in Tissue Culture Cells. (Eng) Parks, W. P. (Univ. Miami Medical Sch., Miami, FL 33152); Young, H. A.; Scolnick, E. M. *Natl Cancer Inst Monogr* (48): 215-218; 1978.

The frequency of spontaneous conversions from high-expression murine mammary tumor virus (MuMTV) to low-expression MuMTV in clonal derivatives of C3HMT murine mammary cell lines was studied. Conversion of expression at a rate of approx 6/100 clones was demonstrated, the conversion being largely unidirectional from a high level to a 10-fold lower level. The rate of mutation to 6-thioguanine resistance in the same cells was considerably lower (approx 3/million). The MuMTV phenotype of the parental lines was shown by all somatic cell hybrids between different MuMTV 6-thioguanine-resistant clones and mouse or hamster thymidine kinase-deficient cells. This indicated that MuMTV expression is regulated by at least two levels of positive control, a constitutive level of expression and glucocorticoid stimulation. Dactinomycin, but not inhibitors of DNA or protein synthesis, blocked MuMTV RNA induction. Synthesis of MuMTV RNA increased 10-fold within 10 min of glucocorticoid treatment, indicating that the major glucocorticoid effect on MuMTV expression occurs at the level of transcription and specifically affects the rate of MuMTV synthesis. (23 refs)

79-3800 Endogenous Mammary Tumour Virus DNA Varies Among Wild Mice and Segregates During Inbreeding. (Eng) Cohen, J. C. (Dept. Microbiology and Immunology, Tulane Univ. Sch. Medicine, New Orleans, LA 70112); Varmus, H. E. *Nature* 278(5703): 418-423; 1979.

The restriction endonucleases *Pst*I and *Eco*RI were used to compare endogenous murine mammary tumor virus (MMTV) DNA in 12 wild mice from four geographically distant areas. Following digestion with the endonucleases, hepatic DNA samples were assayed for fragments containing virus-specific sequences by agarose gel electrophoresis and the DNA transfer method. Analysis of the *Pst*I digests of wild mouse DNA showed that all the mice were unique with respect to their MMTV proviruses, although some shared some relatively small fragments that are also observed in DNA digests from inbred mice. In the *Eco*RI digests, few, if any, fragments were common to two or more mice, and the fragment sizes were not identical to those seen in DNA digests from inbred mice. Two of the 12 mice were apparently free of MMTV DNA. The patterns of virus-specific fragments in *Pst*I and *Eco*RI digests of hepatic DNA from inbred Bagg albino x DBA mice revealed the existence of six different MMTV proviruses, which had segregated as stable, independent genetic elements over a 60-yr period. These results support the hypothesis that endogenous MMTV proviruses were established by multiple,

independent infections of germ cells rather than by somatic mutation of ancestral proviruses or of cellular genes. (44 refs)

- 79-3801 Identification of Mouse Mammary Tumor Virus-specific mRNA.** (Eng) Groner, B. (Swiss Inst. Experimental Cancer Res., CH-1066 Epalinges, Switzerland); Hynes, N. E.; Diggelmann, H. *J Virol* 30(1): 417-420; 1979.

A DNA probe complementary (cDNA) to the RNA genome of mouse mammary tumor virus (MMTV) was used to analyze for the presence of virus-specific messenger RNA (mRNA) sequences in polysomes of a mouse mammary tumor-derived cell line (GR cells) and to identify the size of the specific mRNA species. Separation of polysomal mRNA by agarose gel electrophoresis, transfer of the RNA to diazobenzyloxymethyl paper, and hybridization of the polysomal fractions with ³²P-labeled MMTV cDNA revealed three viral RNA size classes of 10, 8.8, and 4.4 kilobases (kb) in length, respectively. Although the gene organization of MMTV is not definitely determined, by analogy with other RNA tumor viruses it may be possible that the gene coding for the viral core proteins is located at the 5' end of the RNA genome and that the glycoprotein gene has an internal location. If this is the case, the 10-kb mRNA most likely codes for the 77,000-dalton precursor to the major core proteins and the 4.4-kb mRNA might code for the 73,000-dalton glycoprotein precursor. The role of the minor 8.8-kb mRNA species remains obscure, particularly since it does not contain the information present at the 3' end of the viral RNA. (16 refs)

- 79-3802 Structural Analysis of the Intracellular RNAs of Murine Mammary Tumor Virus.** (Eng) Robertson, D. L. (Dept. Microbiology and Immunology, Univ. California, San Francisco, CA 94143); Varmus, H. E. *J Virol* 30(2): 576-589; 1979.

Murine mammary tumor virus (MuMTV)-specific RNA was characterized in several cell types in which viral DNA is transcribed into RNA: cultured GR mouse mammary tumor cells; S49 lymphoma cells from BALB/c; cells from lactating mammary glands of C57BL/6 mice; and mink lung cells infected in vitro with MuMTV. Three species of MuMTV-specific RNA (35S, 24S, and 13S) were found in all cell types, regardless of the source of the viral DNA used as template, the presence or absence of glucocorticoid hormones, or the production of extracellular virus. The three RNA's had the characteristics of messenger RNA's in that they were polyadenylated, associated with polyribosomes, and released from the ribosomes by EDTA treatment. Thus, each RNA probably functions within the cell by coding for virus-specific proteins. The 35S and 24S RNA's apparently code for the viral structural proteins, but the

function and genetic content of the 13S RNA is unknown. The 13S species, unlike the 24S species and subgenomic RNA's of other retroviruses, did not contain sequences derived from the 5' terminus of the genomic RNA. (49 refs)

- 79-3803 Regulation of Mouse Mammary Tumor Viral RNA Synthesis in Embryonal Carcinoma Cells and in Teratocarcinoma Derived Myoblasts.** (Eng) Crepin, M. (Dept. Molecular Biology, Pasteur Inst., 28, rue du Docteur Roux, 75015 Paris, France); Gros, F. *Biochem Biophys Res Commun* 87(3): 781-788; 1979.

The level of expression of mouse mammary tumor virus (MMTV) in undifferentiated embryonal carcinoma cells (ECC), in partially differentiated myoblasts derived from ECC, and in fully differentiated myotubes was compared. The sensitivity of MMTV RNA synthesis to glucocorticoid hormones was also examined. Although no appreciable amount of MMTV RNA could be detected in ECC, hybridization with radioactive viral complementary DNA revealed relatively large quantities of tumor virus RNA in the teratocarcinoma-derived myoblasts. The MMTV RNA level was reduced markedly (20-fold) after the myoblasts had differentiated into myotubes. The glucocorticoid hormone dexamethasone which stimulates MMTV RNA synthesis in differentiated mammary cells, did not affect this synthesis in the myoblastic cells. In contrast, the inhibition of MMTV RNA synthesis in the myotubes was almost completely overcome by dexamethasone. (26 refs)

- 79-3804 Identification, Partial Purification and Biochemical Characterization of γ -Glutamyltranspeptidase Present as a Membrane Component in Skimmed Milk and Milk Fat-Globule Membranes, and in Mammary-Tumour Virus from the Milk of Infected Mice.** (Eng) Francois, C. (Laboratoire de Biochimie Generale et Comparee, Universite de Liege, 4000 Liege, Belgium); Calberg-Bacq, C. M.; Gosselin, L.; Kozma, S.; Osterrieth, P. M. *Biochim Biophys Acta* 567(1): 106-115; 1979.

The presence of γ -glutamyltranspeptidase (GGTP) in murine mammary tumor virus (MuMTV) and the milk of infected and uninfected mice was investigated. Enzymic activity was found in milk-fat-globule membranes from MuMTV-infected Swiss mice, viral particles prepared from the milk of infected Swiss and RIII mice, and vesiculated structures from the skimmed milk of uninfected C57BL mice. The enzyme was partially purified from the MuMTV and the milk-fat-globule membranes. It required the presence of detergents to remain soluble, and it appeared to have a mol wt of at least 400,000. The kinetics of the enzyme and its reactions to competitors and specific inhibitors demonstrated that it was identical to kidney

GGTP. Several oncornaviruses, all of them budding from the plasma membrane, also possessed the enzyme. (28 refs)

- 79-3805 Phosphorylation of Murine Mammary Tumor Virus Precursor Polypeptides.** (Eng) Racevskis, J. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Sarkar, N. H. *J Virol* 30(1): 241-247; 1979.

Evidence showing that phosphorylation of murine mammary tumor virus (MuMTV) structural proteins occurs in discrete stages at the precursor level and that the attached phosphate groups are conserved during the processing of these precursors to the mature virion structural proteins is presented. Phosphorylation of MuMTV structural proteins was studied in an MuMTV-infected epithelial cell line derived from a BALB/cf C3H mouse mammary tumor. Immunoprecipitation of ^{32}P -labeled cell extracts with monospecific anti-p27 serum revealed that phosphorylation occurred at the stage of the core-protein polypeptide precursor prp75. Two forms of phosphorylated prp75 were found: one migrating with an apparent mol wt of 80,000 and the other with a mol wt of 76,000. The 80,000-dalton species was found to be the most heavily phosphorylated. In addition, a relatively stable phosphorylated processing intermediate of 34,000 daltons was observed. Tryptic peptide mapping analysis of the ^{32}P -labeled viral proteins indicated a precursor product relationship between the intracellular phosphorylated, high-mol-wt peptides and the mature MuMTV phosphoproteins p23 and p27. Phosphopeptide analysis also suggested that phosphorylation of the viral proteins occurred in discrete steps and that the attached phosphate groups were conserved throughout the processing steps. (21 refs)

- 79-3806 Immunological Characterization of Mouse Mammary Tumor Virus p10 and Its Presence in Mammary Tumors and Sera of Tumor-bearing Mice.** (Eng) Arthur, L. O. (Biological Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Fine, D. L. *J Virol* 30(1): 148-156; 1979.

The purification and immunological properties of another mouse mammary tumor virus (MMTV) structural protein (p10) that can be used to measure MMTV viral gene expression in natural tissues are described. MMTV p10 and glycoprotein gp52 were purified and used as radiolabeled antigens in sensitive radioimmunoassays. These radioimmunoassays were specific for MMTV proteins, since detergent-disrupted MMTV from C3H/HeN, RIIL, and GR/N mice gave complete competition, whereas C3H/HeNf liver extracts and other lysed retroviruses did not. Both gp52 and p10 are coded by the viral genome,

since MMTV grown in a heterologous cell line (feline kidney cells) competed in these assays. Sera from mammary tumor-bearing mice and mammary tumors from C3H/HeN and C3H/HeNf mice competed in both the gp52 and the p10 assays. Although these radioimmunoassays detected predominantly group-specific antigenic determinants in C3H/HeN and C3H/HeNf tumor extracts, type specificity was found with gp52. Absorption of anti-MMTV serum with C3H/HeNf tumor extracts removed all antibodies directed against p10 and decreased the anti-gp52 titer approx 30-fold. When this absorbed antiserum was used at limiting dilution in the gp52 radioimmunoassay, C3H/HeN tumor extracts gave complete competition, whereas no competition was found with C3H/HeNf tumor extracts. (39 refs)

- 79-3807 Mechanisms of Leukemogenesis. I. Generation of Autoreactive Lymphocytes in Response to a Murine Leukemia Virus.** (Eng) Schenk, P. J. (Dept. Microbiology and Immunology, State Univ. New York, Downstate Medical Center, Brooklyn, NY 11203); Howe, M. L. *J Immunol* 122(5): 1874-1880; 1979.

The capacity of 334C murine leukemia virus (MuLV) to stimulate the generation of virus-specific cytotoxic effector cells in C57BL/6 [relatively resistant to Friend, Moloney, and Rauscher (FMR) MuLV-induced leukemia] and BALB/c (relatively susceptible to FMR MuLV-induced leukemia) mice was studied. Lymphocytes from either strain exhibited no significant levels of cytotoxic reactivity immediately after removal from MuLV-injected donors, but they did exhibit cytotoxicity after secondary in vitro culture with syngeneic FMR antigen-bearing tumor cells. The cytotoxicity of C57BL/6 cells was primarily directed against the syngeneic antigen-bearing RBL-5 lymphoma cells used for secondary stimulation, although reactivity was also observed against FMR antigen-bearing LSTRA lymphoma cells and against P815 and PU-5 tumor cells (all H-2d). No significant cytotoxicity was observed against syngeneic EL-4 target cells or against normal, glass-adherent peritoneal cells from H-2b or H-2d donors. The level of reactivity of BALB/c lymphocytes was generally lower than that of C57BL/6 cells, and it peaked after 6 days of culture, compared with 5-9 days of culture for C57BL/6 cells. Development of cytotoxicity in BALB/c cultures proceeded in the absence of stimulating LSTRA tumor cells, whereas generation of C57BL/6 effector cells required the presence of RBL-5 stimulator cells in the secondary culture system. (25 refs)

- 79-3808 Leukemogenesis by an Endogenous Virus Isolated from the CFW Mouse. II. Early Effects of Virus on Thymus Gland and Bone Marrow Cell Populations.** (Eng) Ball, J. K. (Cancer Res. Lab., Univ.

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Western Ontario, London, Ontario, Canada N6A 5B7). *J Natl Cancer Inst* 62(6): 1517-1522; 1979.

The early effects of a highly leukemogenic and rapidly acting murine leukemia virus (DMBALV, isolated from dimethylbenz(a)anthracene-induced thymic lymphomas in CFW/D mice) on the thymuses of CFW/D mice were investigated. DMBALV-injected grafts from newborn mice (<24 hr old) weighed significantly more than control grafts 30 days after injection into syngeneic hosts. All of the dividing cells were derived from the graft, as were cells of the thymic lymphomas resulting from DMBALV infection. Injection of thymic grafts with DMBALV 7 days after grafting did not alter the normal repopulation of the grafts by cells derived from the host, and all thymic lymphomas induced by DMBALV infection of these grafts were composed exclusively of cells derived from the host. Lethally irradiated female mice were injected with bone marrow from male donors and then with thymic grafts from female donors: the grafts were injected with DMBALV 7 days after grafting. The resulting graft tumors were composed of cells derived from the graft and/or the injected bone marrow. Thymocytes cocultured on thymic epithelium reticulum (TER) monolayers prepared from three DMBALV-induced tumors resulted in "leukemia" in normal recipients 37-51 days postinjection. However, the "leukemic" cells had karyotypes identical to those of the DMBALV-induced tumors that served as donors for the TER monolayers. (28 refs)

79-3809 Adsorption, Penetration, and Uncoating of Murine Leukemia Virus Studied by Using Its Reverse Transcriptase. (Eng) Aboud, M. (Microbiology Unit, Faculty Health Sciences, Ben-Gurion Univ. Negev, Beersheva, Israel); Shoor, R.; Salzberg, S. *J Virol* 30(1): 32-37; 1979.

A procedure using virus-associated reverse transcriptase (RT) was developed to follow the kinetics of adsorption, penetration, and uncoating of murine leukemia virus (MLV). Viral adsorption to the cell membrane was determined by assaying RT activity in the isolated debris of mechanically disrupted NIH/3T3 mouse fibroblasts that had been infected with MLV in the presence of actinomycin D. At 37 C, viral adsorption proceeded at a high initial rate, but after 5 min of incubation with the virus, it gradually slowed down. At 4 C, viral adsorption was slower, but it proceeded linearly. The presence of intracellular virus was determined by centrifuging the cytoplasmic fraction of the disrupted cells at 105,000 x g for 45 min and assaying RT activity in the high-speed pellet. Sucrose gradient analysis of the enzyme activity recovered from the cytoplasm of infected cells indicated that it represented intact virus particles. No appreciable amount of these particles was recovered from the cytoplasm of cells infected at 4 C. This indicates that the virions recovered from the cytoplasm of cells infected at 37

C are indeed intracellular virus particles that penetrated the cells and not just membrane-bound particles mechanically released into the cytoplasmic fraction during cell disruption. The intracellular virus was found to accumulate in the cytoplasm, reaching a max level within 20 min. The accumulated intracellular virus particles gradually disappeared from the cytoplasm, probably as a result of uncoating, which was completed within 80 min. (21 refs)

79-3810 Genetic Control of Mouse Leukemia Virus Replication. (Eng) Jolicoeur, P. (Institut de Recherches Cliniques, 110 West Pine Ave., Montreal, Quebec, H2W 1R7, Canada). *Natl Cancer Inst Monogr* (48): 191-197; 1978.

The site of *Fv-1* gene restriction, which results in restriction of the growth of the murine leukemia virus (MuLV) in vitro, was studied. B-tropic virus was able to infect about 100-fold less NIH nonpermissive mouse cells than BALB permissive cells. The cytoplasm of BALB cells contained a lower level of virus-specific RNA after infection with an N-tropic virus (nonpermissive system) than after infection with a B-tropic virus (permissive system); the opposite results were obtained with NIH cells. Thus, *Fv-1* gene restriction was not a late event. After infection of NIH cells with N-tropic virus, new integrated viral sequences were detected, whereas few new sequences could be found in NIH cells infected with B-tropic virus. Thus, the *Fv-1* gene product prevented integration of proviral DNA. The levels of unintegrated proviral DNA in B-type cells infected with N- or B-tropic viruses were almost identical soon after infection, indicating that provirus DNA was synthesized as efficiently in nonpermissive as in permissive cells. The results indicate that the *Fv-1* gene product interferes with a step after the synthesis of proviral DNA and before its integration. (30 refs)

79-3811 Effect of Interferon on Murine Leukaemia Virus Infection. IV. Formation of Non-infectious Virus in Chronically Infected Cells. (Eng) Pitha, P. M. (Oncology Center, Johns Hopkins Univ. Sch. Medicine, Baltimore, MD 21205); Wivel, N. A.; Fernie, B. F.; Harper, H. P. *J Gen Virol* 42(3): 467-480; 1979.

SC-1 and AKR-2B mouse cells that were chronically infected with murine leukemia virus (MuLV) were treated with interferon (IF: 150 units/ml), and the number of cell-associated virions and released virions produced before and after treatment was compared, along with their infectivity, content of virus genome RNA, and morphology. IF treatment led to a 100-fold decrease in the amount of infectious virus released into the medium and a 10-fold decrease in the number of virus particles measured by the virion-associated reverse transcriptase assay. However, there was little change in the amount of cell-associated infectious virus,

although nearly twice as many cell-associated virions were counted in electron micrographs. With both types of cells, IF blocked MuLV replication at the postbudding stage, but it did not change the morphology of the particles produced or their content of virion 70S RNA. Infectious virus assembled on the cell membranes of IF-treated cells was less stable at 37 C than that grown in the absence of IF. Release of infectious virus from IF-treated cells was not inhibited by actinomycin D or cycloheximide, although both agents inhibited virus production in controls. These results show that IF inhibits MuLV replication through effects on virion assembly; these effects lead to the formation of noninfectious particles and of fewer virions. Kinetics analysis further shows that IF affects MuLV assembly rapidly, and the induction of an antiviral protein may not be required. (34 refs)

- 79-3812 Expression of Murine Leukemia Virus DNA Polymerase in Mouse Uterus During Pregnancy.** (Eng) Strickland, J. E. (Viral Oncology Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701); Fowler, A. K.; Hellman, A. *Biol Reprod* 20(4): 751-756; 1979.

The expression of two murine leukemia virus proteins, p30 and reverse transcriptase, was studied in subcellular fractions prepared from gravid uteri of NIH Swiss mice at time points throughout gestation. Both proteins were most concentrated on the first day after mating; they decreased by 90% or more near midgestation, and they increased dramatically by the first day after parturition. Reverse transcriptase was primarily found in the microsomal fraction, but p30 was distributed throughout the subcellular fractions. The expression of these proteins may be mediated by estrogen. These findings plus those of previous studies support the notion that viruses are involved in mammalian reproduction. (30 refs)

- 79-3813 Involvement of DNA Damage in Hydroxyurea-mediated Induction of Endogenous Murine Retrovirus.** (Eng) Rascati, R. J. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Tennant, R. W. *Virology* 94(2): 273-281; 1979.

The mechanism of the hydroxyurea (HU)-mediated induction of retroviruses from AKR mouse cells was studied. Treatment with HU resulted in virus induction only if serum was added immediately after the removal of HU; no stable intermediate was formed. DNA synthesis was required shortly after HU treatment. Iododeoxyuridine (IdUrd) or bromodeoxyuridine (BrdUrd) enhanced HU-mediated induction by approx 200% and 74%, respectively. Induction by halogenated pyrimidine alone was extremely low. IdUrd was capable of significantly enhancing virus expression only if present when the cells had been exposed to

HU for a sufficient length of time for induction to occur. Since HU inhibited semiconservative replication and since BrdUrd was incorporated into the cellular genome predominantly by unscheduled DNA synthesis (repair replication) under these conditions, this stimulation appeared to be the result of insertion into DNA of the thymidine analogs during the repair of HU-induced alterations in the DNA. The halogenated pyrimidines were able to form a stable induction intermediate when incorporated by repair synthesis, and this intermediate was similar to that formed when the analogs were incorporated during semiconservative replication. Thus, the same sites appear to be involved in induction by damaging agents and by halogenated pyrimidine incorporation. (30 refs)

- 79-3814 Genetic Individuality of Intracisternal A-Particles of *Mus musculus*.** (Eng) Lueders, K. K. (Lab. Biochemistry, NCI, NIH, Bethesda, MD 20014); Kuff, E. L. *J Virol* 30(1): 225-231; 1979.

The nucleic acid sequence relationship between mouse intracisternal A-type particles and C- and B-type RNA tumor viruses of *Mus musculus* was examined by reciprocal complementary DNA-RNA hybridization. Complementary DNA's prepared from the RNA's of intracisternal A particles were hybridized with high-mol-wt RNA's from a variety of murine tumor viruses, and complementary DNA's representing a variety of RNA tumor virus genomes were hybridized with the high-mol-wt RNA's from A particles. The criterion for homology between two types of virus was that the heterologous hybridization reaction occur over the same RNA concentration range as the homologous reaction. The results of these hybridizations indicate that there are no major sequence homologies between the RNA of intracisternal A particles and the RNA of representative members of B- and C-type viruses of *Mus musculus*. (37 refs)

- 79-3815 Virus Particles in Normal Prostate Tissues of Mice from Ten Strains, with Special Reference to Type-B Virus Particles.** (Eng) Ohtsuki, Y. (Dept. Pathology, Okayama Univ. Medical Sch., Shikata-cho 2-5-1 Okayama 700, Japan); Dmochowski, L. *Gann* 70(2): 195-202; 1979.

The prostate tissues of 61 normal mice from 10 different strains, including high (C3H/Dm, RIH/Dm, and A/Dm) and low (BALB/c/Dm, C3Hf/Bi/Dm, and C3Hf/He/TEX) mammary cancer and high (AKR/Dm) and low (CBA/J/Cr, SJL/J/Cr, and C57/BL/6/TEX) leukemia strains, were examined by electron microscopy for the presence of virus particles. B-type virus particles were present in the normal prostate tissues of some old (11 mo-2 yr) mice from all three high and one low (BALB/c/Dm) mammary cancer strain. Varying numbers

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of C-type virus particles were present in the prostate tissues of some young (1-6 mo) and old mice from all 10 strains, especially in the older mice. Intracisternal A-type virus particles were observed in all mice examined. Immunological characterization of the B-type virus particles by fixed immunofluorescence tests and immunoelectron microscopy revealed that these particles in the normal prostate tissues of old C3H/Dm and A/Dm mice are morphologically and immunologically similar to mouse mammary tumor virus. Thus, the prostate tissue can be a potential source of horizontally transmitted mammary tumor virus in mice of at least some high mammary cancer strains. (24 refs)

- 79-3816 Characterization of an Amphotropic Murine C-Type Virus that is NB-Tropic.** (Eng) Kontor, E. J. (Lab. Molecular Oncology, Christ Hosp. Inst. Medical Res., Cincinnati, OH 45219); Krueger, R. G. *Virology* 94(2): 451-459; 1979.

A new retrovirus isolate that can productively infect murine and nonmurine cells is described. The new isolate, S/A-1, was produced by clone A₃₁ BALB/3T3 cells upon cocultivation of these cells with SIPC-2 BALB/c myeloma cells. It possessed a buoyant density of 1.16 g/cm³, a C-type morphology, a high-mol-wt (70S) RNA, and an RNA-dependent DNA polymerase. In addition to murine cells, it productively infected mink, rat, guinea pig, rabbit, human, and Pekin duck cells. Its ecotropic activity and ability to infect hamster and Pekin duck embryo cells distinguished it from other amphotropic virus strains. The S/A-1 virus possessed proteins of 69,000, 47,000, 32,000, 15,000, 12,000, and 10,000 daltons that corresponded to the established gp69/70, gp45, p30, p15, p12, and p10 proteins of C-type viruses. It also possessed two p30 proteins. (25 refs)

- 79-3817 Characterization of a Murine Myeloma (MOPC-315) C-Type Virus by Nucleic Acid Hybridization.** (Eng) Yaniv, A. (Dept. Human Microbiology, Sackler Sch. Medicine, Tel-Aviv Univ., Tel-Aviv, Israel); Gazit, A. *Virology* 93(1): 256-259; 1979.

Nucleotide sequence homology between the murine myeloma MOPC-315 C-type virus and some other known oncornaviruses was assayed by DNA:RNA hybridization. MOPC-315 virus was more homologous to the murine oncornaviruses than to simian sarcoma virus or avian myeloblastosis virus. The MOPC viral genome shared extensive nucleotide sequences with AKR leukemia virus and Kirsten murine sarcoma virus (Ki-MSV: 90% and 77% hybridization values, respectively) and only moderate sequence relationships with Rauscher leukemia virus (RLV: 46%) and Moloney leukemia virus (MoLV: 45%). In DNA:DNA hybridization experiments with cellular DNA's of various animal species (mouse, rat, hamster, guinea pig,

and cat), the MOPC-315 viral genome shared the greatest degree of homology with cellular DNA's of the various mouse species (BALB/c, AKR, C57 black, and NIH Swiss). In association kinetics assays performed between MOPC viral probe and cellular DNA from myeloma cells and from normal uninfected BALB/c cells, identical extents of hybridization (86%) and reannealing kinetics were obtained. The hybrid formed with MOPC cellular DNA was indistinguishable in its melting profile from the hybrid formed with BALB/c cellular DNA. It is concluded that the myeloma MOPC-315 virus is an endogenous virus of the mouse. (20 refs)

- 79-3818 Murine Type-C Virus Expression in Human x Mouse Hybrid Cells.** (Eng) Brown, S. (Dept. Biochemistry, Univ. Nottingham Medical Sch., Queen's Medical Centre, Nottingham NG7 2UH, England); Minna, J. D. *Biochem Soc Trans* 7(2): 384-386; 1979.

Hybrids between human bone marrow or lymphocyte cells and oncornavirus-producing mouse RAG cells were used to study the control of murine virus expression by human chromosomes. The hybrids selectively retained the human X chromosome. No single human chromosome tested was sufficient for the suppression of reverse transcriptase activity in the culture fluid of these hybrids. After growth in nonselective medium for 3 wk followed by transfer to medium containing 6-thioguanine, 16 clones no longer expressed human X chromosome markers, but the reverse transcriptase phenotypes remained the same. Thus, the human X chromosome was not responsible for virus suppression. (10 refs)

- 79-3819 Differences in Pathogenicity among Cloned Sublines of a Murine Leukemia Virus.** (Eng) Manly, K. F. (Dept. Viral Oncology, Roswell Park Memorial Inst., Buffalo, NY 14263); Buffett, R. F. *J Virol* 30(1): 232-240; 1979.

Five clones of the lymphatic leukemia virus 334C were isolated by a procedure designed to maintain the homogeneity of the clones. 334C virus causes lymphatic leukemia within 80-200 days when injected sc into newborn Ha/ICR Swiss or BALB/c mice, and it is antigenically related to the Friend-Moloney-Rauscher subgroup of murine leukemia viruses. Three of the clones induced leukemia in Ha/ICR mice with the time course of the uncloned parental virus, one induced leukemia with a delayed time course, and one seemed to be biologically inactive. When the clone inducing leukemia most rapidly and the clone inducing leukemia least rapidly were subcloned, the subclones retained the leukemogenicity of the parental clones. The electrophoretic patterns of purified virion proteins and hybridization of viral RNA's with virus-specific DNA suggest that these clones are two closely related

variants, not unrelated viruses. Furthermore, in mice infected with these two clones, viral RNA appeared in thymuses and spleens at the same time after infection and at nearly the same concentrations. Thus, variations in leukemogenicity can be determined by a genetic property of an ecotropic leukemia virus, and this property is expressed in a manner that is more subtle than simple control of replication. (34 refs)

- 79-3820 Genetic Mapping of the Ecotropic Murine Leukemia Virus-inducing Locus of BALB/c Mouse to Chromosome 5.** (Eng) Kozak, C. A. (Lab. Viral Diseases, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Rowe, W. P. *Science* 204(4388): 69-71; 1979.

Assignment of the inducibility locus for the infectious ecotropic murine leukemia virus of BALB/c mice was assigned to chromosome 5 using somatic cell genetics, and this assignment was confirmed and the intrachromosomal location of the gene was established by Mendelian breeding studies. Somatic cell hybrids were made between BALB/c mouse peritoneal cells and cells of the Chinese hamster cell line E36. The inducibility locus, designated *Cv*, was mapped to chromosome 5, 23 units from the locus for phosphoglucomutase-I, with the gene order *Cv-Pgm-1-Gus*. This low-efficiency inducibility locus is therefore not allelic with the chromosome 7 loci previously described for two other mouse strains with high virus inducibility. These studies provide further evidence that endogenous ecotropic viruses represent viral genomes inserted at different chromosome sites in the various mouse strains. (22 refs)

- 79-3821 Age Dependence and Genetics of Expression of Ecotropic Murine Leukemia Virus in SJL/J Mice.** (Eng) Colombatti, A. (Dept. Pharmacology and Experimental Therapeutics, Johns Hopkins Univ., 725 N. Wolfe St., Baltimore, MD 21205); De Rossi, A.; Hilken, J.; Collavo, D.; Chieco-Bianchi, L. *J Natl Cancer Inst* 62(6): 1451-1457; 1979.

The presence of ecotropic murine leukemia virus (MuLV) in SJL/J-(V+) mice (SJL/J mice with a high level of MuLV) was studied. The highest titers were found in tail extracts from 2- to 4-mo-old mice, the spleen and thymus having intermediate and very low titers, respectively. The spleen cell virus showed an N-tropism. Nearly all mice older than 1-2 mo had virus titers in the tail tissues $\geq 10^2$ - 10^3 . The titers in the spleen cells of these mice were similar to those in the tail tissues and higher than those in spleen extracts. The frequency of virus increased in 30- to 40-day-old mice compared with 10- to 20-day-old mice. These young mice were divided into two classes: those that were positive at 10-20 days (24%) or 30-40 days (57%), and

those that were consistently negative (43%). The virus titers in spleen homogenates and the number of infectious centers for ecotropic virus in spleens and thymus glands from lymphoma-bearing mice were 10-fold lower than those in the same tissues of normal mice. Virus titers in the tail homogenates of lymphoma-bearing and normal mice were similar. The high-virus phenotype was dominantly expressed in F_1 hybrids of several strains. Backcross experiments indicated that one dominant gene was responsible for the expression of high levels of virus and that no additional major genetic influences were involved. There was no close correlation between high virus phenotype and three coat color markers (*d*, *a*, *b*). (26 refs)

- 79-3822 Unstable Resistance of G Mouse Fibroblasts to Ecotropic Murine Leukemia Virus Infection.** (Eng) Yoshikura, H. (Dept. Genetics, Inst. Medical Science, Univ. Tokyo, P.O. Takanawa, Tokyo, Japan); Naito, Y.; Moriwaki, K. *J Virol* 29(3): 1078-1086; 1979.

Cell lines of G mouse embryo tissues were established and their clones examined with respect to murine leukemia virus (MuLV) sensitivity, karyotype, glucose-6-phosphate dehydrogenase-1 (G6PD-1) activity, and cell morphology. G mouse cells were resistant to N- and NB-tropic Friend leukemia viruses and to the B-tropic MuLV WN1802(B). They were resistant to Moloney murine sarcoma virus (MuSV) focus formation, but they were almost fully permissive to the helper component of the MuSV-MuLV complex. The cells were fully permissive to amphotropic MuLV and also to focus formation by amphotropic MuSV. None of the cells tested were sensitive to a xenotropic virus derived from NZB mice. In cultures made from individual embryos, marked variations (200-fold) in sensitivity to N-tropic Friend leukemia virus were seen. The variations were not due to genetic heterogeneity, because the embryos were from mothers inbred for 15-16 generations. When cell lines were established from individual embryos, 1 resistant, 2 N-type, and 2 cell lines sensitive to both N- and B-tropic MuLV's were obtained. Cloning of the resistant line indicated that the resistant cells constantly segregated sensitive cells during culture. Some sensitive cell clones were devoid of *Fv-1* restriction. All the dually permissive cells derived from G mouse cells and from feral mouse-derived SC-1 cells retained G6PD-1 activity and a normal chromosome 4. The dual permissiveness was due neither to deletion nor mutation of the *Fv-1* locus; rather, *Fv-1* was probably rendered nonfunctional by an unknown mechanism. (25 refs)

- 79-3823 Abelson Virus-transformed Haematopoietic Cell Lines with Pre-B-Cell Characteristics.** (Eng) Boss, M. (Imperial Cancer Res. Fund., Lincoln's Inn Fields, London WC2, England); Greaves, M.; Teich, N. *Nature* 278(5704): 551-553; 1979.

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Two Abelson murine leukemia virus (A-MuLV)-transformed hematopoietic cell lines with pre-B-cell characteristics are described. Line ABC-1 was derived from BALB/c mouse bone marrow cells transformed in vitro by A-MuLV. They were large, vacuolated, undifferentiated blast cells with no cell-surface immunoglobulin (Ig), or detectable levels of Thy-1.2. Thus, there appeared to be no mature B or T lymphocytes present. Cytoplasmic IgM was present in 1%-4% of the cells. The percentage of cells expressing cytoplasmic IgM was increased by lipopolysaccharide (LPS, 5-20 µg/ml), but not by dimethyl sulfoxide (DMSO, 1%). In cells that had been in continuous culture for 16 mo, DMSO, LPS, butyric acid, and conditioned medium from BALB/c spleen cell cultures stimulated with pokeweed mitogen (PWM) all increased the percentage of IgM-positive cells. Dextran sulfate, concanavalin A, PWM, cAMP, cholera toxin, phytohemagglutinin, and purified protein derivative of tuberculin failed to stimulate cytoplasmic IgM. No cell-surface Ig-positive cells were detected after any of the treatments. The results suggest that A-MuLV had transformed immature precursor cells committed to a B-lymphoid lineage and that the virus seems to transform mainly B-lineage cells. (19 refs)

- 79-3824 Structural Studies of Retroviruses: Characterization of Oligomeric Complexes of Murine and Feline Leukemia Virus Envelope and Core Components Formed upon Cross-linking.** (Eng) Pinter, A. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Fleissner, E. *J Virol* 30(1): 157-165; 1979.

Cross-linking experiments demonstrating the presence of distinct associations between leukemia virus envelope components and between viral core proteins are described. To examine the protein proximity and subunit organization of C-type retroviruses, preparations of AKR murine leukemia virus were treated with bifunctional cross-linking reagents and analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE). The cross-linked components obtained were characterized by immunoprecipitation with monospecific antisera against purified viral proteins, followed by SDS-PAGE analysis both before and after cleavage of the cross-links. With these procedures, complexes of both viral envelope and core components were identified. The major envelope subunit obtained was a large (apparent mol wt 450,000-500,000), glycosylated complex composed of four to six gp70-p15(E) subunits. This complex was detected over a 100-fold range of cross-linker concentration and thus seems to represent a particularly stable viral substructure. The cross-linked complexes of the core proteins consisted of oligomers of p30 dimers, suggesting that the p30 dimer is a basic structural unit of the viral core. When virion preparations that had previously been disrupted with the nonionic detergent Nonidet P-40 were cross-linked, the envelope complex was still observed, indicating that this structure is stable in the presence of Nonidet P-40. A similar envelope structure was

observed for feline leukemia virus, which suggests that such a complex may be a conserved feature of oncornavirus structure. (31 refs)

- 79-3825 In Vitro Induction of Continuous Acute Promyelocytic Cell Lines by Friend or Abelson Murine Leukemia Virus.** (Eng) Greenberger, J. S. (Joint Center for Radiation Therapy, 50 Binney Street, Boston, MA 02115); Davisson, P. B.; Gans, P. J.; Moloney, W. C. *Blood* 53(5): 987-1001; 1979.

The in vitro induction of stably transformed lines of leukemogenic promyelocytes in NIH/Swiss mouse bone marrow cultures infected by the anemia-inducing strain of Friend leukemia virus (FLV-A) or by Abelson murine leukemia virus with Moloney virus as helper [A-MuLV(M-MuLV)] was studied. The numbers of colony-stimulating factor (CSF)-dependent colony-forming cells (CFUc) in virus-infected cultures were increased compared with those of control cultures. After 6-7 wk, 4/10 cultures infected with FLV-A and 4/8 infected with A-MuLV(M-MuLV) generated nonadherent cells that replicated rapidly in suspension culture. One week after infection, nonadherent populations from FLV-A cultures showed atypical promyelocytes, lysosome, esterase-M, and myeloperoxidase. Cell lines generated in vitro by A-MuLV(M-MuLV) showed significantly more basophilic promyelocytes. Both virus-induced cell lines formed CFUc in the presence of WEHI-3 CSF with 50%-90% efficiency; the efficiency was 15%-63% in the absence of CSF. Nonadherent cells from FLV-A- and A-MuLV(M-MuLV)-infected cultures showed a peak of virus replication, as reflected by increased reverse transcriptase activity, at 6-8 wk. Virus released by both cell lines grew in K-NIH, K-BALB, and K-NRK mouse cells; virus released by FLV-A-infected lines showed preferential N-tropism and that released by A-MuLV(M-MuLV)-infected lines showed a slight B-tropism. Cells from both lines induced granulocytic leukemias in adult and newborn NIH/Swiss mice after ip inoculation. (46 refs)

- 79-3826 Glycoprotein Encoded by the Friend Spleen Focus-forming Virus.** (Eng) Dresler, S. (Dept. Pathology, Washington Univ., St. Louis, MO 63110); Ruta, M.; Murray, M. J.; Kabat, D. *J Virol* 30(2): 564-575; 1979.

Evidence that the Friend spleen focus-forming virus (F-SFFV) encodes a glycoprotein is presented. Extracts of F4-6/K Friend erythroleukemia cells contained three components that precipitated specifically with monospecific antiserum made to the Friend murine leukemia virus (F-MuLV) virion envelope glycoprotein: two, gp75 and gPr90env (the biosynthetic precursor of gp75 and p15E), are known to be encoded by the F-MuLV genome; the third

was a glycoprotein of apparent mol wt 55,000 (gp55). The virus released from the F4-6/K cells was adsorbed to Sc-1 mouse embryo fibroblasts, cloned and examined for F-SFFV, F-MuLV, and for gp55 synthesis. All of the Sc-1 cell lines that contained F-SFFV also contained gp55, whereas the lines that lacked F-SFFV also lacked gp55. There was also evidence of a possible correlation between gp55 synthesis and the leukemogenic potential of different F-SFFV-infected nonproducer cell lines. Examination of other cell lines infected with Friend virus indicated that gp55 is probably encoded by the F-SFFV genome. The gp55-like protein in different cell lines varied somewhat in size, probably as a result of differences in processing. Peptide maps of gp55 and F-MuLV gp75 support the idea that gp55 contains amino acid sequences that occur in gp75 and that it may contain additional amino acid sequences. The unglycosylated polypeptide of gp55, formed in cells treated with 2-deoxy-D-glucose, had a mol wt of approx 45,000. gp55 appeared to be absent or present only in low concentrations in erythroleukemia cell plasma membranes, but it accumulated intracellularly in large amounts. It was absent from released virions. Most of the cellular gp55 had an isoelectric point of 8.5-9.0. The results support the hypothesis that an *env* gene recombination event was involved in the origin of F-SFFV. (52 refs)

- 79-3827 **Changes of Serum Thymic Factor Levels in Friend Leukemia Virus-infected Mice.** (Eng) Garaci, E. (Inst. Microbiology, Piazzale delle Scienze, Rome Univ., 00100 Rome, Italy); del Gobbo, V.; Santucci, L.; Rossi, G. B.; Rinaldi-Garaci, C. *Leuk Res* 3(2): 67-74; 1979.

In view of the prominent role of the thymus or its hormonal products in the immune response and, specifically, in the maturation and regulation of T cells, the effects of Friend leukemia virus (FLV) infection on levels of serum thymic factor (STF: a thymus-derived hormone isolated from human and murine sera) and θ -positive spleen cells were studied in male DBA/2 and BALB/c mice. Sera and spleen cells were sampled at various time intervals after infection of the mice with the polycythemic strain of FLV. A pronounced, dose-dependent decrease of STF levels was observed in DBA/2 mice as early as 48 hr after infection; STF levels were also decreased in BALB/c mice, but the effect was less marked and long-lived than that in DBA/2 mice. Parallel to the fall of STF levels in the sera, θ -positive cells were markedly decreased in the spleens of DBA/2 mice and, to a lesser extent, in BALB/c mice. The θ -positive cells were assayed by measuring the sensitivity of spleen rosette-forming cells to azathioprine. Both the fall of STF levels and the decrease of θ -positive spleen cells did not persist >4 wk and were not, therefore, related to the state of overt leukemia. Conceivably, however, these changes, which appear to be virus-related may influence the early fate of FLV-transformed cells by lowering some thymus-dependent functions that may be relevant to immunological surveillance. (34 refs)

- 79-3828 **Inhibition of In Vitro Friend Murine Leukemia Virus Infection of Lipopolysaccharide-activated B-Cells with Concanavalin A.** (Eng) Bowen, D. L. (Harvard Medical Sch. IV, Boston, MA 02115); Isaak, D. D.; Cerny, J. *J Natl Cancer Inst* 62(6): 1497-1502; 1979.

The effect of concanavalin A (Con A) on murine spleen cell cultures infected in vitro with Friend murine leukemia virus (MuLV) was studied. The addition of lipopolysaccharide (LPS) to the culture medium stimulated MuLV replication, as indicated by infectious centers (IC), 10-fold. Con A (2.5 μ g/ml) reduced the number of IC in LPS-stimulated cultures two- and sixfold on days 3 and 6, respectively, after infection. A similar effect was observed when lymphocytes were exposed to Con A prior to infection. The concentration of Con A that inhibited the infection did not neutralize the virus, however, and pretreatment of B cells with Con A inhibited the subsequent infection with MuLV. The latter effect was reversed by specific removal of Con A from the B-cell surfaces with α -methyl-D-mannopyranoside. The results suggest that the lectin interferes with MuLV on the membranes of B cells. (29 refs)

- 79-3829 **Moloney Leukemia Virus Gene Expression and Gene Amplification in Preleukemic and Leukemic BALB/Mo Mice.** (Eng) Jaenisch, R. (Heinrich Pette-Institut für Experimentelle Virologie und Immunologie, Universität Hamburg, Martinistrasse 52, 2000 Hamburg 20, W. Germany). *Virology* 93(1): 80-90; 1979.

The relation between virus expression and virus-specific gene amplification was investigated in BALB/Mo mice, a strain that carries the exogenous Moloney leukemia virus (M-MuLV) as an endogenous virus. Molecular hybridization experiments indicated that spleen and thymus cells of BALB/Mo mice synthesize M-MuLV-specific RNA soon after birth. Only very low amounts (100- to 500-fold lower) of virus-specific RNA were present in liver, kidneys, or brain of BALB/Mo mice of any age. Concentrations of virus-specific RNA were 150- to 400-fold higher in the spleen and thymus of preleukemic (4-wk-old) or leukemic animals than in the livers of newborn mice. Virus gene expression in the spleen and thymus reached high levels at 3-4 wk of age and did not increase substantially in older animals. Similar results were obtained using animals heterozygous or homozygous at the *Mov-1* locus. Virus gene expression was correlated with somatic amplification of M-MuLV-specific DNA sequences during the preleukemic and leukemic phases. DNA amplification occurred in two steps in the target tissues: a first step to approx two copies per haploid genome equivalent in preleukemic mice and a second step to three to four copies in leukemic tissues. Nontarget tissues carried one copy of M-MuLV-specific DNA sequences regardless of the age of the animal. These results suggest that M-MuLV-specific

gene expression is not sufficient for leukemic transformation and is related to virus-specific DNA amplification in preleukemic animals. The second amplification of M-MuLV DNA sequences appears to be related to transformation. (39 refs)

- 79-3830 Highly Inducible Cell Lines Derived from Mice Genetically Transmitting the Moloney Murine Leukemia Virus Genome.** (Eng) Bacheler, L. (Tumor Virology Lab., Salk Inst., San Diego, CA 92112); Jaenisch, R.; Fan, H. *J Virol* 29(3): 899-906; 1979.

Permanent, non-virus-producing fibroblast cell lines were established from a BALB/Mo (male) x BALB/c (female) mouse embryo carrying an endogenous, genetically transmitted Moloney murine leukemia virus (M-MuLV) genome. These cells carried the M-MuLV genome, as demonstrated by hybridization of cellular DNA to M-MuLV complementary DNA, but did not express it at the levels of virus production, accumulation of intracellular viral p30, or M-MuLV-specific RNA. Treatment with bromodeoxyuridine (50 µg/ml for 24 hr) resulted in the induction of XC-positive NB-tropic virus, although only a small fraction of the cells released virus (<0.1% after 48 hr). Immunofluorescent staining and flow microfluorometry indicated that a wave of p30 accumulation occurred in the induced cells, with a max at 24-48 hr after the addition of bromodeoxyuridine. Furthermore, most, if not all, cells were induced to produce p30 protein. Similar kinetics were found for the accumulation of M-MuLV-specific RNA in the cytoplasm of induced cells. This rapid induction of virus expression in a majority of cells was dependent on the presence of the M-MuLV genome and probably represents primarily the expression of this endogenous virus, since induction was not observed in cells similarly derived from a sibling embryo lacking the M-MuLV genome. (19 refs)

- 79-3831 In Vitro Synthesis of a 9 kbp Terminally Redundant DNA Carrying the Infectivity of Moloney Murine Leukemia Virus.** (Eng) Gilboa, E. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Goff, S.; Shields, A.; Yoshimura, F.; Mitra, S.; Baltimore, D. *Cell* 16(4): 863-874; 1979.

³²P-labeled DNA was synthesized in purified, detergent-disrupted virions of Moloney murine leukemia virus (M-MuLV) and the size of the undenatured ³²P-DNA molecules was determined by neutral agarose gel electrophoresis. A band of DNA approx 9 kilobase pairs (kbp) long was detected along with a heterogeneous collection of other molecules. The 9-kbp DNA was double-stranded, as shown by its resistance to single-strain-specific S1 nuclease, and it was infective. Analysis of fragmentation of the DNA using restriction endonucleases showed that it is in-

distinguishable from the linear double-stranded DNA synthesized in infected cells. On the basis of the cleavage site positions for a number of enzymes, the 9-kbp DNA has a 575-base direct terminal repetition. It is longer than the viral RNA at both ends, evidently as a result of repetitive copying of segments of the RNA. The virions also synthesized an 8.4-kbp double-stranded circular DNA that lacks one copy of the terminal repetition, as well as viral DNA longer than 9 kbp. These results show that the virions contain all of the enzymatic machinery needed to generate fully double-stranded linear and one form of circular DNA. (26 refs)

- 79-3832 Structure of Products of the Moloney Murine Leukemia Virus Endogenous DNA Polymerase Reaction.** (Eng) Haseltine, W. A. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA 02115); Coffin, J. M.; Hageman, T. C. *J Virol* 30(1): 375-383; 1979.

The process by which the single-stranded RNA genome of Moloney murine leukemia virus is copied into DNA in vitro was investigated. DNA synthesis is initiated near the 5' end of the genome, and elongation of the growing chain occurs by a jumping mechanism whereby the DNA synthesized at the 5' end of the genome is elongated along the 3' end. Unique DNA fragments synthesized beyond the 5' end of the genome in vitro have, at their 5' and 3' ends, copies of unique sequences from the 5' and 3' ends of the genome. These flank a copy of the 49- to 60-nucleotide terminally redundant sequence. These results indicate that the terminal redundancy serves as a "bridge" to allow a DNA molecule synthesized at the 5' end of the genome to serve as a primer for synthesis from the 3' end. (16 refs)

- 79-3833 gag-related Polyproteins of Moloney Murine Leukemia Virus: Evidence for Independent Synthesis of Glycosylated and Unglycosylated Forms.** (Eng) Edwards, S. A. (Tumor Virology Lab., Salk Inst., San Diego, CA 92112); Fan, H. *J Virol* 30(2): 551-563; 1979.

The gag-related polyproteins of Moloney murine leukemia virus (M-MuLV)-infected NIH-3T3 cells were studied. Immunoprecipitation of the gag-related polypeptides from infected cells revealed major gag-related polyproteins of 80,000 daltons (80K: (Pr80gag) and 65K (Pr65gag) and the reverse transcriptase precursor Pr180gag-pol. (Since Pr80gag was shown here to be glycosylated, it was renamed Gp80gag, indicating a glycosylated gag-related polyprotein of 80K.) A minor gag-related polypeptide of 73K and several minor polypeptides smaller than Pr65gag were also found. Labeling with (³H)mannose and changes in electrophoretic mobility following treatment of the cells with tunicamycin or digestion with endoglycosidase II indicated

that GpP80gag was glycosylated. Mannose and galactose labeling experiments suggested that during the synthesis of GpP80gag, core oligosaccharides containing mannose are added. Subsequent glycosylation of this polypeptide apparently then takes place to yield a final product of 95K, presumably present at the cell surface. This glycopeptide is then cleaved to produce the 55K and 40K gag-related polypeptides, which are released into the culture medium as soluble proteins. The polypeptide protein of GpP80gag appeared to be a 75K polypeptide that differed from Pr65gag. The 65K gag-related cell-free translation product comigrated with Pr65gag, and the 75K cell-free product comigrated with the unglycosylated form of Gp80gag. Both of the gag-related cell-free translation products could be labeled with (35S)formylmethionine, which was incorporated only as the N-terminal amino acid during translation. The results suggest that GpP80gag and Pr65gag are translated from two separate initiation sites in M-MuLV RNA. (41 refs)

- 79-3834 Immune Responses to Weakly Immunogenic Virally Induced Tumors. V. Short In Vitro Cultivation of YAC Changes Its Antigenic Properties. (Eng) Devens, B. (Lautenberg Center General and Tumor Immunology, Hebrew Univ. — Hadassah Medical Sch., Jerusalem, Israel); Schochot, L.; Naor, D. *Cell Immunol* 44(2): 442-453; 1979.

Differences in the antigenic structures of the Moloney-induced YAC tumor of A/J mice and the in vitro YAC-1 tumor line were studied. Distinct populations of effector cells were raised to YAC and YAC-1 following priming of female A mice by either YAC-1 or Rauscher-induced RBL5 lymphoma cells and cultivation of the splenocytes for 6 days. This was demonstrated by the fact that, when YAC was the labeled target, unlabeled YAC-1 did not block the cytotoxic effector cells as well as unlabeled YAC. When YAC-1 was the labeled target, YAC showed virtually no blocking activity, whereas YAC-1 effectively blocked the cytotoxic effector cells. RBL5 generated anti-YAC and anti-YAC-1, and anti-RBL5 reactive cells in A mice. Priming with either YAC-1 or RBL5 generated multiple lines of effector cells after in vitro cultivation of the primed splenocytes. Competition experiments with YAC, YAC-1, or RBL5 as the primary target showed that the best competition for each labeled target was the same tumor as that used as the target. It is concluded that both YAC-1 and RBL5 stimulate different populations of effector cells to each of the targets. (9 refs)

- 79-3835 Application of Antisera to Interferon in Studying Oncogenic Viruses. (Eng) Ingnot, A. D. (Lab. Tumor Viruses, Dept. Tumor Immunology, Inst. Immunology and Experimental Therapy, Polish Acad. Sciences, 53-114 Wrocław, Poland); Oleszak, E. *Arch Immunol Ther Exp (Warsz)* 26(1-6): 529-536; 1978.

The effects of sheep anti-mouse interferon (IF) serum on

the growth of lesions induced by Moloney sarcoma virus (MSV) and/or herpes simplex virus type 2 (HSV-2) were studied in male BALB/c mice. Anti-IF serum potentiated the growth of MSV-induced tumors, although tumors in all mice regressed spontaneously starting 2 wk after inoculation. After approx 1 mo, all 4- to 5-wk old mice treated with anti-IF serum or untreated were tumor-free. In contrast, in 3- to 4-wk-old mice treated with anti-IF serum, large tumors reappeared at the site of MSV inoculation and metastasized, killing approx 80% of the animals. This metastatic proliferation occurred in only 10% of mice not given anti-IF serum. Anti-IF serum also aggravated the signs of HSV-2-induced ear lesions and increased mortality due to CNS damage following infection. Virus titers in the brain stem and spinal cord were also higher in mice given anti-IF serum. IF protected against HSV-2-induced disease and nullified the enhancing effect of anti-IF serum. Anti-IF serum did not potentiate recurrent HSV-2-induced ear infection or affect apparently healthy mice that had survived HSV-2 infection. Superinfection of Rauscher murine leukemia virus (MuLV-R)-infected mice with HSV-2 inhibited the splenomegaly seen with MuLV-R, and anti-IF serum further amplified this effect. Most mice infected with MuLV-R and HSV-2 died of CNS involvement; anti-IF serum increased the mortality. Mortality due to CNS damage in HSV-2-infected mice was prevented by administration of exogenous IF. (14 refs)

- 79-3836 Purification of RNA-directed DNA Polymerase from Mouse Spleen Infected with Rauscher Leukemia Virus. (Eng) Drescher, B. (Abteilung Zellregulation, Bereich Bioregulation, Zentralinstitut für Molekularbiologie, Akademie der Wissenschaften der DDR, 1115 Berlin-Buch, E. Germany); Riedel, H.; Niemiec, P. *Acta Biol Med Ger* 37(11/12): 1655-1663; 1978.

A method involving cell fractionation, lysis of the microsomal fraction, and Sephadex G-200 and phosphocellulose chromatography was used to purify RNA-directed DNA polymerase from the spleens of BALB/c and NMRI mice infected with Rauscher murine leukemia virus (R-MuLV). Purified splenic R-MuLV DNA polymerase could transcribe ribopolymers, deoxyribopolymers, and heteropolymers as efficiently as purified DNA polymerase from purified virions. (25 refs)

- 79-3837 Effect of Methotrexate on the Activity of DNA-Dependent RNA Polymerases A and B in the Spleens of Mice Infected with Rauscher Leukemia Virus and on the Development of Virus-induced Leukemia. (Rus) Pravdina, N. F. (D. I. Ivanovskii Inst. Virology, Moscow, USSR); Shobukhov, V. M.; Veselovskaia, T. V.; Smirnova, N. R.; Galegov, G. A. *Vopr Med Khim* 25(2): 193-198; 1979.

The effect of the antitumor drug methotrexate (MT) on DNA-dependent RNA-polymerase activity was studied in

BALB/c mice with Rauscher leukemia virus (RLV)-induced leukemia. Animals were inoculated ip with a series of 10-fold dilutions of an extract of leukemic spleens (1 ml of the extract contained 10^7 infectious units). At various times postinfection (PI), mice received ip or sc injections of MT (2 doses of 2.5 μ g/g at 3-day intervals). MT increased the latent period of leukemia development and the life-span of leukemic mice. The spleens of mice treated with MT weighed 2-2.5 times less than those of untreated (leukemic control) mice. The spleens of healthy control mice had three peaks of RNA polymerase activity (A-I, A-II, and B). On day 5 PI in the leukemic controls, when the spleen weighed 0.3 g, there were still three peaks of RNA polymerase activity, but polymerase B activity accounted for 64.1% of the total enzyme activity (compared with 47.1% in healthy controls). On day 11 PI, when the spleen weighed 0.8 g, there were only two peaks, and polymerase A accounted for 75.8% of the total enzyme activity. On day 11 PI, the spleen wt in mice treated with MT was 0.31 g and the RNA polymerase profile corresponded to that for leukemic control mice on day 5 PI (two-fold increase in polymerase B activity). (8 refs)

- 79-3838 Tryptic Peptide Analysis of *gag* and *gag-pol* Gene Products of Rauscher Murine Leukemia Virus.** (Eng) Kopchick, J. J. (Dept. Biology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Karshin, W. L.; Arlinghaus, R. B. *J Virol* 30(2): 610-623; 1979.

(3 H)Tyrosine-labeled viral precursor polyproteins and known mature viral proteins derived from the Rauscher murine leukemia virus *gag* and *pol* genes were examined by two-dimensional tryptic peptide mapping. Pr200 *gag-pol* contained peptide sequences of the viral core proteins p30, p15, p12, and p10, as well as peptide sequences found in the cell-associated reverse transcriptase (RT). Intermediate RT precursor Pr125 *pol* lacked peptide sequences of the four core proteins, but it contained RT-specific tryptic peptides plus two additional tyrosine-containing tryptic peptides not related to *gag* or *pol* gene products. Methionine-containing tryptic peptide analysis also suggested the presence of additional protein material in Pr125 *pol*. Pr200 *gag-pol* contained two additional classes of tryptic peptides: those associated with Pr125 *pol* but not Pr80 *pol*; and those not found in Pr125 *pol* or any known viral protein. Pr200 *gag-pol* may, therefore, contain additional gene products aside from the *gag* and *pol* genes. Pr80 *gag* and Pr65 *gag* maps had sequences of all four core proteins. Pr65 *gag* contained two p30 tyrosine tryptic peptides that were not present in Pr80 *gag*, suggesting that Pr80 *gag* may not be the precursor to Pr65 *gag*. Pr80 *gag* also contained tryptic peptides not found in Pr65 *gag*. Two of these were present in Pr80 *pol*, but not in any of the viral core proteins, suggesting that Pr80 *gag* and Pr80 *pol* may have overlapping peptide sequences. Consistent with this conclusion was the finding that Pr80 *gag* terminated with the *pol* gene. A

model that describes the relationship of these findings to viral gene products is presented. (35 refs)

- 79-3839 Post-translational Modification of Rauscher Leukemia Virus Precursor Polyproteins Encoded by the *gag* Gene.** (Eng) Schultz, A. M. (Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Rabin, E. H.; Oroszlan, S. *J Virol* 30(1): 255-266; 1979.

A detailed study was made of three kinds of major post-translational modifications that occur in the course of processing *gag* gene-coded precursor polyproteins in Rauscher murine leukemia virus (R-MuLV)-infected JLS-V9 cells: phosphorylation, glycosylation, and proteolytic cleavage. To study the sequence of these events, chronically infected JLS-V9 cells were labeled in pulse-chase experiments with the radioactive precursors [35 S]methionine, [14 C]mannose, [3 H]glucosamine, and [32 P]phosphate. Newly synthesized *gag* polyproteins that incorporated label and the modified products derived from them were identified by immunoprecipitation of cell lysates with anti-p30 rabbit serum, followed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and autoradiography. Pulse-chase experiments were carried out in the presence and absence of tunicamycin (TM), an inhibitor of glycosylation. Among the three major polyproteins synthesized in the absence of TM, two were found to be glycosylated but not phosphorylated. These were designated gPr80 *gag* and gP94 *gag*. Both shared identical [35 S]methionine peptides with Pr65 *gag* and p30. Of the two nonglycosylated precursors, Pr65 *gag* and Pr75 *gag*, only Pr65 *gag* was found to be detectably phosphorylated, and Pr75 *gag* could be readily identified only when glycosylation was inhibited. On the basis of these results, a scheme for the posttranslational modification of *gag* polyproteins is proposed. According to this scheme, the *gag* gene-encoded polyproteins are processed from a common precursor, Pr75 *gag*, by two divergent pathways: one leading through the intermediate Pr65 *gag* to internal virion components via cleavage and phosphorylation and the other via TM-sensitive mannosylation to the intermediate gPr80 *gag*, which is further glycosylated to yield the cell-surface polyprotein gP94 *gag*. (52 refs)

- 79-3840 Translating Ability of 35S RNA from Cells Infected with Rauscher Leukemia Virus.** (Ukr) Sherban, S. D. (Inst. Problems Oncology, Kiev, USSR); Smirnova, I. A. *Dopov Akad Nauk Ukr RSR [B]* (12): 1123-1126; 1978.

The translating ability of virus-specific RNA was studied in a cell-free extract of Ehrlich ascites carcinoma (EAC) cells. Spleen cells from mice infected with Rauscher leukemia virus (RLV) were subjected to electrophoresis in

polyacrylamide gels; the 35S RNA fraction was then subjected to repeated ultracentrifugation in a sucrose gradient. The purified 35S RNA fraction was added to a fraction of the cell-free extract of EAC cells. Translation was measured by the rate of ^{14}C -lysine and ^{14}C -phenylalanine incorporation. The 35S RNA fraction caused a 4.25-fold increase in polypeptide synthesis. Incorporation of label into the acid-insoluble fraction was increased in the presence of Mg^{2+} (2 mM) and spermine (80 μM). (13 refs)

- 79-3841** Glucose-regulated Membrane Properties of Untransformed and Virus-transformed BHK21 Cells. (Eng) Lage-Davila, A. (Instituto Nacional de Oncologia y Radiobiologica, 29 y E Vedado, Havana, Cuba); Hofmann-Clerc, F.; Torpier, G.; Montagnier, L. *Exp Cell Res* 120(1): 181-189; 1979.

The biochemical properties of two asparagine-dependent clones of BHK21 cells, one untransformed (C13/8) and the other (HS5) transformed by hamster sarcoma virus (HSV), were studied under conditions of glucose excess and glucose starvation. In normal medium, the uptake of 2-deoxyglucose (2-DOG) by HS5 cells was four- to ten-fold greater than that by C13/8 cells. 2-DOG uptake could be derepressed by glucose starvation in both cell types, but it could be decreased by glucose excess only in HS5 cells. The rate of uptake in fully derepressed C13/8 cells did not equal that in transformed cells under any condition. Starvation-induced changes in the plasma membrane proteins of HS5 and C13/8 cells included increases in two proteins of mol wt 95,000 and 78,000. The loss of the large, external transformation-sensitive protein, the high density of intramembranous particles, the increase in the amount of a 177K integral plasma membrane protein, and the increase in the amount of a high mol wt surface glycopeptide in the HS5 cells were not altered by glucose depletion. Another iodinated protein of mol wt 160,000 was decreased in transformed cells. This protein increased in both normal and transformed cells when arrested in G_1 by asparagine deprivation. (30 refs)

- 79-3842** Cellular Regulation of Mammalian Sarcoma Virus Expression: A Gene Regulation Model for Oncogenesis. (Eng) Porzig, K. J. (Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014); Robbins, K. C.; Aaronson, S. A. *Cell* 16(4): 875-884; 1979.

In a study of replication-defective transforming retroviruses of feline origin, morphological revertants were encountered at a relatively high frequency in clonal lines transformed by Snyder-Theilen feline sarcoma virus (ST-FeSV). The block to expression of the transformed state in these cellular revertants was spontaneously reversible at low frequency. Infection with certain C-type helper viruses reversed the block at very high efficiency and led to focus

formation. Helper virus complementation was not a direct effect of helper virus functions expressed in the initially infected revertant cells. The helper virus acted indirectly by rescuing sarcoma virus and permitting it to infect and transform another cell within the revertant population. A specific and very marked reduction in transcriptional and translational products of the sarcoma virus genome was demonstrated in the revertant cells by biochemical and immunological techniques. The findings suggest that reversion results from cellular transcriptional regulation of the integrated sarcoma virus genome. Morphological reversion in this viral transformation system provides a potentially important model for oncogenesis resulting from derepression of cellular genes that possess malignant potential. (53 refs)

- 79-3843** Glucocorticoid Effects on Peripheral Blood Lymphocytes in Cows Infected with Bovine Leukemia Virus. (Eng) Bloom, J. C. (New Bolton Center, 382 W. Street Road, Kennett Square, PA 19348); Kenyon, S. J.; Gabuzda, T. G. *Blood* 53(5): 899-912; 1979.

The effects of glucocorticoids on peripheral blood lymphocytes (PBL) of cattle with lymphoproliferative conditions associated with bovine leukemia virus (BLV)—persistent lymphocytosis (PL) and lymphosarcoma cell leukemia (BLSL)—were studied. The spontaneous incorporation of ^{14}C -thymidine by PBL from all five cows with PL and 2/3 cows with BLSL was markedly inhibited by concentrations of hydrocortisone 21-sodium succinate (HSS) as low as 10^{-7} M. Mitogen-stimulated thymidine incorporation was more resistant to HSS than was spontaneous incorporation. Absolute lymphocyte counts following administration of prednisolone (0.33 g tid, im, for 3 wk) to three cows with PL were reduced by 80%-90% and remained at this level for 40-60 days after cessation of treatment. Similar results were seen in 2/3 cows with BLSL, except that rapid progression of disease was evident within 5 days after cessation of treatment. The lymphocyte count increased in a third cow with BLSL after prednisolone treatment. Tumor expansion in the BLSL cows was associated with a rapid increase in the number of peripheral atypical lymphocytes. Decreases in B cells accounted for 85%-98% of the decreased lymphocyte counts in the PL cows but only a small proportion of the decreased counts in the BLSL cows. The circulating B-cell population reduced by the steroid appeared to be the same population that spontaneously incorporated thymidine. (30 refs)

- 79-3844** Seroepidemiologic Studies of Bovine Papillomavirus Infections. (Eng) Pfister, H. (Institut für Virologie, Zentrum für Hygiene, Universität Freiburg, Hermann-Herder-Strasse 11, 7800 Freiburg, W. Germany); Huchthausen, B.; Gross, G.; zur Hausen, H. *J Natl Cancer Inst* 62(6): 1423-1425; 1979.

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Bovine and human sera were analyzed for the presence of antibodies against bovine papillomavirus types 1 and 2 (BPV 1 and 2) and human papillomavirus type 1 (HPV 1) by solid-phase radioimmunoassay. The bovine sera were obtained from slaughterhouses, and the human sera were obtained from butchers working in the slaughterhouses, farmers, and from routine diagnostic laboratory specimens. Rabbit antiserum raised against BPV from bovine cutaneous warts showed no cross-activity with HPV 1 or HPV 4 and reacted with all of 10 BPV isolates. The DNA of one virus isolate was identical to BPV 2 by restriction enzyme analysis. Of 110 cattle sera examined, 19% showed low titers of antibodies against BPV, whereas none of the human sera were positive. Among the butchers, 5/17 had verrucae at the time of serum sampling and 5 others had a history of warts. Five of the farmers also had histories of warts. The bovine sera did not contain antibodies against HPV 1, but 53% of the sera from the butchers did. (13 refs)

- 79-3845 Latent Infection of Cattle with Bovid Herpesvirus 2. (Eng) Martin, W. B. (Animals Diseases Res. Assoc., Moredun Inst., Edinburgh, England); Scott, F. M. *Arch Virol* 60(1): 51-58; 1979.

A series of experiments was conducted to determine whether bovid herpesvirus 2 (BHVS) can remain as a latent infection in cattle. Ten calves were infected by different routes with the bovine mammillitis strain of BHV2. Nine of the calves developed lesions, and BHV2 was isolated from eight of these animals. At intervals after clinical recovery, the calves were given a course of corticosteroid (dexamethasone: 10 mg/day for 5 days) by iv injection. Subsequently, seven calves developed lesions, and BHV2 was reisolated from three of these animals. The shortest interval between initial infection and reisolation of virus was 10 wk. In one calf, virus was recovered on three occasions, at intervals of 20, 44, and 74 wk postinfection. Thus, it is probable that BHV2 can occur as a latent infection in calves and that BHV2 should be included among the herpesviruses known to remain latent. (17 refs)

- 79-3846 Expression of Baboon Endogenous Virus in Exogenously Infected Baboon Cells. (Eng) Lavelle, G. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Foote, L.; Heberling, R. L.; Kalter, S. *J Virol* 30(1): 390-393; 1979.

The susceptibility of baboon cells and of heterologous cells to baboon endogenous virus (BaEV) was compared in a series of virus titration experiments using an immunofluorescence assay. Strains of low-passage, fetal diploid, baboon (*Papio cynocephalus*) fibroblasts were susceptible to exogenous infection with three independent isolates of BaEV, as measured by an immunofluorescence

assay specific for the major internal structural polypeptide (p28) of BaEV. Infectivity of the M7 strain of BaEV for baboon cells of fetal skin muscle origin was equivalent to that for human and dog cells in that similar, linear, single-hit titration patterns were obtained. The assay for supernatant RNA-dependent DNA polymerase, however, showed that baboon cells produced only low levels of virus after infection compared with virus production by heterologous cells. The results showed that BaEV was capable of penetrating baboon cells and that viral genes were expressed in infected cells. However, replication of complete infectious virus was restricted, indicating that in this primate system homologous cells differentially regulate the expression of viral genes. (18 refs)

- 79-3847 Retrovirus Sequences in a Leukemic Gibbon and Its Contact: Evidence for Partial Provirus in the Nonleukemic Gibbon. (Eng) Wong-Staal, F. (Lab. Tumor Cell Biology, NCI, NIH, Bethesda, MD 20205); Reitz, M. S.; Gallo, R. C. *Proc Natl Acad Sci USA* 76(4): 2032-2036; 1979.

The results of molecular hybridization experiments showing the presence of proviral sequences in tissues from two gibbon apes derived from a colony from Halls' Island, Bermuda, are reported. Integrated DNA sequences were detected in tissues from a leukemic gibbon (6G-1), from whose WBC a distinct strain of gibbon ape leukemia virus (GaLV-H) was isolated, and gibbon 6G-4, a contact of 6G-1 that had uremia and cachexia of unknown origin. Although 6G-4 had no detectable neoplasia or viral proteins, its serum contained persistent antibody against GaLV antigens. Whereas DNA from most of the tissues of 6G-1 contained GaLV provirus, DNA from only three tissues (kidney, spleen, and liver) of 6G-4 showed detectable viral sequences, and the extent of hybridization in each case was lower than that with 6G-1. After cleavage with *Bam*HI, two virus-specific DNA fragments were detected in the tissues of 6G-1. Only one of these fragments was detected in the positive tissues of 6G-4. The results indicate that: (1) 6G-4 was exposed to and infected by GaLV; (2) early target sites for infection of gibbons by GaLV may be limited to a few tissues; and (3) infection can be contained by integration of only partial provirus in a few tissues. (29 refs)

- 79-3848 Mapping of Related and Nonrelated Sequences of RNA from Woolly Monkey Virus and Gibbon Ape Leukemia Virus. (Eng) Reitz, M. S. (Lab. Tumor Cell Biology, NCI, NIH, Bethesda, MD 20014); Luczak, J. C.; Gallo, R. C. *Virology* 93(1): 48-56; 1979.

Experiments were conducted to determine if the previously detected nucleotide sequence differences (20%-40%) in RNA's from woolly monkey virus (SSV) and gibbon ape

leukemia virus (GaLV) are concentrated within a distinct region of the genome. Poly(A)-containing subgenomic fragments of 35S RNA were prepared from SSV-1, and the fraction of the whole viral genome that each fragment represented was determined by several criteria. The characterized RNA fragments were labeled with iodine-125 and used in competition hybridization experiments with complete RNA from SSV-1 and from two different isolates of GaLV [Hall's Island (GaLV-H) and San Francisco (GaLV-SF) strains] to determine the position, with respect to the poly(A) (3') end, of RNA sequences in the SSV genome that are not shared with these GaLV isolates. The bulk of the unshared sequences was found to be concentrated in the 20%-40% of the RNA closest to the 3' end of the RNA. In this region, GaLV-H differed from GaLV-SF in that it was more related to SSV. If the gene order in the infectious primate viruses is the same as that established for the avian leukosis viruses and suggested for the murine viruses, these results indicate that the unshared sequences of the SSV genome are concentrated within the *env* gene. (28 refs)

- 79-3849 Detection of Virus-specific RNA in Simian Sarcoma-Leukemia Virus-infected Cells by In Situ Hybridization to Viral Complementary DNA. (Eng) Kaufman, S. L. (Lab. Tumor Cell Biology, NCI, Bethesda, MD 20014); Gallo, R. C.; Miller, N. R. *J Virol* 30(2): 637-641; 1979.

An in situ molecular hybridization system that will detect retrovirus RNA in the cytoplasm of individual virus-infected cells is described. The technique was applied to the study of cells infected with simian sarcoma-leukemia virus, in which the virus-specific RNA was detected by hybridization to simian sarcoma-leukemia virus ³H-labeled complementary DNA. The procedure is specific for viral RNA, has a low background that does not interfere with the detection of low levels of positive hybridization, and is independent of the number of virus-negative cells in the population. Thus, the system is useful for detecting viral RNA-containing cells in the presence of an excess of virus-negative cells and for determining which type of cell in a heterogeneous population is expressing viral RNA. (16 refs)

- 79-3850 Isolation of an Endogenous Type C Virus Related to the Infectious Primate Type C Viruses from the Asian Rodent *Vandeleuria oleracea*. (Eng) Callahan, R. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014); Meade, C.; Todaro, G. J. *J Virol* 30(1): 124-131; 1979.

The properties of an infectious, xenotropic, C-type virus isolated from a tissue culture cell line derived from the Asian rodent *Vandeleuria oleracea* (long-tailed tree mouse)

are described. The virus-associated reverse transcriptase (RNA-dependent DNA nucleotidyltransferase) and the major internal protein p30 are immunologically related to the respective proteins of the woolly monkey-gibbon ape group of infectious primate viruses. By these criteria, the *V. oleracea* viral isolate is similar to the murine type C-I class of endogenous retroviruses, and it has been designated Vand C-I. Nucleic acid homology studies show that *V. oleracea* cellular DNA shares similar levels of homology with DNA from members of the *Mus* and *Rattus* genera and lower levels of homology with other rodent genera. The Vand C-I viral genome is present in *V. oleracea* cellular DNA in multiple copies, and partially related sequences can be detected in other rodent genera. These results support the conclusion that the Vand C-I viral genome is genetically transmitted in *V. oleracea* and that the type C-I class of endogenous retroviral genes has been highly conserved during evolution. (27 refs)

- 79-3851 Ultrastructural Studies on the Replication of Herpes Virus Ateles-73 in Owl Monkey Kidney Cells. (Eng) Luetzeler, J. (Immunopathology Section, Univ. Cologne, Cologne, W. Germany); Heine, U. I.; Wendel, E.; Prasad, U.; Ablashi, D. V. *Arch Virol* 60(1): 59-73; 1979.

The morphology and replicative cycle of *herpesvirus ateles* strain 73 (HVA-73) in subhuman primate cells were examined electron microscopically and compared with those of other oncogenic herpesviruses. A relatively slow replicative cycle of HVA-73 in owl monkey kidney (OMK) cells allowed the cytoplasmic and nuclear stages of replication, comprising virus uptake, transport, maturation, and extrusion to be distinguished. Virus uptake was observed within 10 hr of infection, and it occurred both as a result of fusion between virus and cell membranes and by phagocytosis. Morphologic evidence for the transfer of viral DNA from nucleocapsids to the nucleus at the nuclear membrane is presented. This was shown by the location of numerous empty capsids in front of nuclear pores early during infection. Toward the end of the eclipse phase, at about 48 hr after infection, two different types of nuclear inclusion bodies were observed: one composed of fibrillar material and one that was starlike and of high electron density. Progeny nucleocapsids were detected in the nucleus at the same time. Envelopment of the nucleocapsids occurred both at the nuclear membrane and at proliferating Golgi lamellae in the cytoplasm. Each site of envelopment was associated with the maturation of a characteristic, morphologically distinguishable virus particle. The assembly of HVA-73 resembled that of other oncogenic herpesviruses. (30 refs)

- 79-3852 Replication of Herpesvirus DNA: IV. Analysis of Concatemers. (Eng) Ben-Porat, T. (Dept.

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Microbiology, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Rixon, F. J. *Virology* 94(1): 61-70; 1979.

At late stages of infection, pseudorabies virus DNA replicates in the form of large, tangled masses. These tangles were analyzed by cleavage with restriction endonucleases. The results indicate that replicating viral DNA is associated with linear concatemers joined head to tail. The av length of the concatemers varied between 3 and 5 unit-length molecules to much longer arrays. However, DNA consisting of arrays of 3-5 unit-length molecules had S values similar to those of the longer concatemers. Therefore, the sedimentation characteristics of replicative DNA are not an indication of the length of the linear concatemers in the structure. (21 refs)

- 79-3853 Purification and Concentration of Herpes Simplex Virus Types 1 and 2.** (Rus) Kitsak, V. I. (Central Inst. Advanced Training Physicians, Moscow, USSR); Moisiadi, S. A.; Bocharov, A. F.; Landin, L. K.; Sklianskaia, E. I. *Vopr Virusol* (2): 142-148; 1979.

The purification and concentration of extra- and intracellular herpes simplex virus types 1 and 2 (HSV-1 and HSV-2) are described. The HSV was isolated from cultures of human embryo skin-muscle cells. Virus-containing culture liquid was precipitated with ammonium sulfate and then centrifuged in a Ficoll gradient. The ³H-Thymidine-labeled extracellular virus was detected in two main zones of the gradient at 1.110-1.114 and 1.083-1.085 g/cm³, respectively. The amount of infectious HSV-1 detected in both zones was 38.4% of the total, as determined by intracranial inoculation experiments with albino mice. The corresponding amount of infectious HSV-2 was 26.3%. (21 refs)

- 79-3854 Synthesis of Herpes Simplex Virus DNA in Isolated Chromatin.** (Eng) Knopf, K. (Inst. Virus Res., German Cancer Res. Center, Heidelberg, W. Germany). *Biochemistry* 18(9): 1776-1781; 1979.

Herpes simplex virus (HSV) DNA synthesis in isolated chromatin of African green monkey kidney (RC-37) cells was studied after HSV type 1 infection. HSV chromatin synthesized DNA at a 35-fold higher rate than the corresponding chromatin from uninfected cells (mock chromatin). Optimal DNA synthesis was observed in the presence of all four ribonucleoside triphosphates and a triphosphate generating system. DNA synthesis was reduced about twofold in the presence of pancreatic RNase and was stimulated by salt. The pH optimum was 7.5. The optimal reaction temperature was 41 C for HSV chromatin and 38.7 C for mock chromatin. The rate of HSV DNA synthesis was max at 8-12 hr after infection. HSV DNA polymerase or HSV cytoplasm stimulated HSV chromatin

only slightly, but they stimulated mock chromatin DNA synthesis ninefold. HSV DNA polymerase was shown to be the major DNA synthesizing activity in HSV chromatin. HSV chromatin synthesized viral DNA (65.5%) and cellular DNA sequences at ratios identical with those seen in vivo. More than 60% of the newly synthesized single-stranded DNA fragments sedimented under alkaline and neutral conditions with a sedimentation constant >10S. (16 refs)

- 79-3855 Acute Infection of Differentiated Neuroblastoma Cells by Latency-positive and Latency-negative Herpes Simplex Virus *ts* Mutants.** (Eng) Gerdes, J. C. (Dept. Microbiology and Immunology, Univ. Colorado Medical Center, Denver, CO 80262); Marsden, H. S.; Cook, M. L.; Stevens, J. G. *Virology* 94(2): 430-441; 1979.

The phenotypes of wild-type and temperature-sensitive (*ts*) mutants of herpes simplex virus type 1 (HSV-1) were compared following infection of C1300 neuroblastoma cells, baby hamster kidney (BHK) cells, and mouse brain cells in situ at the nonpermissive temperature. Differentiated C1300 cells were productively infected by the syncytial variants of strain 17 HSV-1. The *ts* mutants of this virus were equally temperature-sensitive in BHK cells and C1300 cells, but they were restricted following infection of mouse brains in situ. The full range of virus-specific products was detectable in brain neurons or C1300 cells infected with wild-type virus. However, the morphologic phenotypes for mutant *ts* G were different in the three systems, with the neurons apparently being more restrictive for this virus. The phenotypes of mutant *ts* I were similar in BHK and C1300 cells, but different in brain neurons in situ. The phenotypes of *ts* J and *ts* D were similar in all three systems. In general, the DNA phenotypes of the *ts* mutants were the same in C1300 cells as reportedly in BHK cells. All of the viral-induced polypeptides previously reported for infected BHK cells were detected in C1300 cells infected with wild-type virus, but there were quantitative differences. Similar results were obtained with the *ts* mutants, and there seemed to be a unique block in the processing of some early polypeptides in C1300 cells infected with *ts* D or *ts* K. (21 refs)

- 79-3856 Herpes Simplex Virus Type 1 Infection of Isogenic Epstein-Barr Virus Genome-negative and -positive Burkitt's Lymphoma-derived Cell Lines.** (Eng) Leinbach, S. S. (Dept. Therapeutic Radiology, Yale Univ. Sch. Medicine, New Haven, CT 06510); Summers, W. C. *J Virol* 30(1): 248-254; 1979.

The effect of high multiplicities of herpes simplex virus type 1 (HSV-1; 10⁷ plaque-forming units/cell) on the Epstein-Barr virus (EBV) genome-negative Burkitt's

lymphoma-derived cell lines BJAB and Ramos and their in vitro EBV-converted sublines BJAB-B1, BJAB-A5, BJAB-B95-8, and AW-Ramos was investigated. Specifically, cultures were monitored for cell growth and HSV-1 DNA synthesis. EBV-converted BJAB cultures were more permissive for HSV-1 infection than BJAB cultures. Significant cell killing and HSV-1 DNA synthesis were observed during the first 48 hr of infection in the EBV-converted BJAB cultures but not in the BJAB cultures. The EBV-converted BJAB-B1 cell line contains an appreciable fraction of EBV-negative cells. Therefore, it was cloned. EBV-positive and -negative cells were identified by EBV-determined nuclear (EBNA) antigen anticomplement immunofluorescence. Two types of subclones were identified: (1) those that contained both EBNA-positive and EBNA-negative cells and (2) those that contained only EBNA-negative cells. When levels of HSV-1 DNA synthesis were measured in these subclones, it was found that the former were more permissive for HSV-1 infection than the latter. Thus, the presence of the EBV genome in BJAB cells correlates with the increased permissiveness of these cells for HSV-1 during the first 48 hr of infection. Nonetheless, persistent HSV-1 infections were established in both BJAB and EBV-converted BJAB-B1 cultures. No differences in extent of permissiveness for HSV-1 infection were found for Ramos and EBV-converted AW-Ramos cells. (20 refs)

- 79-3857 Partial Characterization of Herpes Simplex Virus Type 2 (HSV-2)-specific Poly(A) + RNA by Hybridization to *Eco*RI-generated HSV-2 DNA Fragments.** (Eng) Bodemer, W. W. (Radiobiology Labs., Yale Univ. Sch. Medicine, 333 Cedar St., New Haven, CT); Bodemer, M. *Virology* 92(2): 507-517; 1979.

DNA-RNA filter hybridization studies were performed to determine the quantity and origin of the polyadenylated RNA [poly(A)+RNA] in polyribosomes from herpes simplex virus type 2 (HSV-2)-infected Vero cells at different stages of infection. RNA was labeled in infected cells and hybridized to uncleaved HSV-2 DNA as well as to its *Eco*RI fragments. Templates coding for early virus-specific RNA were widely dispersed within the HSV-2 genome in the short and long segments, since early poly(A)+RNA contained transcripts hybridizing to all *Eco*RI fragments. The virus-specific fraction of labeled poly(A)+RNA that hybridized to each of the *Eco*RI fragments was increased in 1- β -D-arabinofuranosylcytosine (ara-C)-treated poly(A)+RNA as well as in late poly(A)+RNA. Poly(A)+RNA from infected, ara-C-treated cells contained virus-specific RNA that hybridized to each of the *Eco*RI fragments. Transcripts from some *Eco*RI fragments were exceptionally well-represented at early and/or late times after infection, which indicates that at least some fragments encode primarily for early or late RNA. These hybridizations showed that the relative amounts of transcription from one region of the genome vary with time after infection compared with the relative amounts of transcription from other

regions. Thus, at specific stages of infection, some genomic regions seem to be preferentially transcribed quantitatively. (31 refs)

- 79-3858 Neoplastic Transformation of Cultured Syrian Hamster Embryo Cells by DNA of Herpes Simplex Virus Type 2.** (Eng) Jariwalla, R. J. (Div. Biophysics, Johns Hopkins Univ. Schs. Hygiene and Public Health and Medicine, Baltimore, MD 21205); Aurelian, L.; Ts'o, P. O. *J Virol* 30(1): 404-409; 1979.

The neoplastic transformation of cultured Syrian hamster embryo cells by purified herpes simplex virus type 2 (HSV-2) DNA at dilutions that lack detectable infectivity, ie, under conditions most closely reflecting those that may operate in vivo, is reported. The cells were transformed to a neoplastic phenotype after exposure to HSV-2 (S-1) DNA at concentrations ($\leq 0.01 \mu\text{g}/60\text{-mm dish}$) at which infectivity was no longer demonstrable. Transformed cells manifested in vitro phenotypic properties characteristic of the neoplastic state, expressed HSV-specific antigens, and induced invasive tumors in vivo. Transfection and transformation of Syrian hamster embryo cells with HSV-2 DNA or its fragments is a suitable system for investigating the structure and function of the HSV transforming gene(s). (29 refs)

- 79-3859 Identification of Disulfide-linked Protein Complexes in the Nucleocapsids of Herpes Simplex Virus Type 2.** (Eng) Zweig, M. (Carcinogenesis Intramural Program, Frederick Cancer Res. Center, Frederick, MD 21701); Heilman, C. J.; Hampar, B. *Virology* 94(2): 442-450; 1979.

High-mol-wt disulfide-linked protein complexes in herpes simplex virus type 2 (HSV-2) nucleocapsids were demonstrated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) analysis under nonreducing conditions. In gels containing the nonreduced proteins, the intensities of the p155, p50, and p40 bands were reduced 2-, 5-, and 10-fold, respectively, whereas the p32, p25, and p12 bands were unchanged relative to gels containing reduced proteins. Using diagonal gel electrophoresis, the p350 component dissociated into two major constituents, p155 and p50. However, neither SDS-PAGE nor gel filtration in guanidine hydrochloride reproducibly resolved distinct disulfide-linked components containing p40. (16 refs)

- 79-3860 Organ Culture Model for the Study of HVH-II Infections in Carcinoma of the Cervix.** (Eng) Tobin, S. M. (Dept. Res., Univ. Ontario Medical Sch., 600 University Ave., Toronto, Ontario M5G 1X5, Canada);

Fish, E. N.; Wilson, W. D.; Papsin, F. R. *Obstet Gynecol* 53(5): 559-564; 1979.

An experimental technique for the maintenance of human and monkey cervical tissues in organ cultures is described, and the effects of herpesvirus hominis type II (HVH-II) infection on these tissues are reported. Fully viable basal cells could be routinely maintained in culture for up to 21 or 40 days in the case of human and monkey tissue, respectively. Early degenerative changes included pyknosis and loss of cells in the squamous zone of the ectocervical epithelium. The final stage of degeneration involved the lifting off of basal cells from the basement membrane and rapid tissue necrosis. Following infection with HVH-II, human and monkey tissues showed cytoplasmic and nuclear hypertrophy preceded by the appearance of HVH-II-specific immunofluorescence in the basal zone of the epithelium. The nucleoli then fragmented and there was a progressive increase in B inclusion bodies. Multinucleation and the appearance of nuclei with a ground-glass appearance followed. The subsequent appearance of intranuclear A inclusion bodies indicated the later stages of infection. The infected cells then separated from the adjacent normal cells, exfoliated, and underwent degeneration around 20 days after infection. (13 refs)

79-3861 **Relation of HVH-II to Carcinoma of the Cervix.** (Eng) Tobin, S. M. (Dept. Res., Mount Sinai Hosp., 600 University Ave., Toronto, Ontario M5G 1X5, Canada); Fish, E. N.; Cooter, N. B.; Papsin, F. R. *Obstet Gynecol* 53(5): 553-558; 1979.

The oncogenic potential of herpesvirus hominis type II (HVH-II) was studied in female Wistar rats. All 20 animals inoculated with HVH-II gave positive cervico-vaginal HVH-II-specific immunofluorescence. Changes in the cervical epithelium ranged from nonspecific inflammatory changes with some increase in the basal cell layer and enlargement of the nuclei to more severe inflammatory changes with a marked increase in the basal cell layer. The changes were a direct consequence of increased activity of the basal cells. Basal cell hyperplasia was evident in 15 of the infected animals, and there was evidence of accompanying metaplasia in 14 and of dysplasia in 7. All but five of the infected animals showed cytologic evidence of HVH-II infection. This included hypertrophy of the cytoplasm and nuclei, evidence of B and A inclusion bodies, multinucleated giant cells, and the appearance of a granular texture in the nuclei. The results suggest that multiple etiologic factors are involved in the multistep pathogenesis of cervical cancer and that HVH-II functions as an etiologic agent to initiate the multistep process to proceed as far as dysplasia. (35 refs)

79-3862 **Heterogeneity of Epstein-Barr Virus Derived from P3HR-1 Cells.** (Eng) zur Hausen, H.

(Institut für Virologie, Universität Freiburg, Zentrum für Hygiene, D-7800 Freiburg, W. Germany); Fresen, K. O. *IARC Sci Publ* 20: 391-396; 1978.

The reason for the difference in Epstein-Barr virus (EBV) nuclear antigen (EBNA) patterns between cell lines transformed by EBV strains P3HR-1 and B95-8 was studied. B95-8-transformed cells all stain brilliantly for EBNA, but the pattern in P3HR-1 cells is more heterogeneous: apart from a few brilliantly stained nuclei, a faintly granular nuclear pattern predominates. Individual P3HR-1 virus-converted BJAB cells were cloned, and the 27 subclones were analyzed for EBNA expression. All subclones except one revealed the same EBNA pattern as the parental clone: 30%-80% were EBNA-positive, and a corresponding percentage were EBNA-negative; ie, they showed a faintly granular EBNA pattern. The one exception showed no detectable EBNA. The data indicate that in these subclones, brilliant EBNA staining accompanied the segregation of EBNA-negative cells. Nucleic acid hybridization studies indicated that segregation of EBNA-negative cells in the clones showing intense EBNA expression was due to a loss of viral DNA. However, there was no correlation between the intensity of EBNA expression and the number of EBV genome equivalents per cell. EBV genome-containing cells had an av of 14 times more cells showing early antigen (EA) synthesis after superinfection by P3HR-1 virus compared with EBNA-negative cells infected under identical conditions. Studies of the kinetics of EA induction in EBNA-positive and EBNA-negative cells indicated that complementation was required for EA induction after superinfection. (6 refs)

79-3863 **Expression of Latent Epstein-Barr Virus Genomes in Human Epithelial/Burkitt's Lymphoblastoid Hybrid Cells.** (Eng) Nonoyama, M. (Lab. Molecular Virology, Life Sciences Biomedical Res. Inst., Inc., St. Petersburg, FL 33710); Tanaka, A.; Glaser, R. *IARC Sci Publ* 20: 397-402; 1978.

Expression of latent Epstein-Barr virus genomes in somatic cell hybrids of Burkitt's lymphoblastoid cells (non-virus-producer Raji and virus-producer HR-1) with D98 cells (HeLa variant) was studied. Treatment of the hybrid D98/Raji and D98/HR-1 cells with iododeoxyuridine (IUDR: 60 µg/ml for 3 days) induced the formation of early antigen (EA) and virus capsid antigen and replication of virus DNA, whereas the same treatment of Raji cells induced only the formation of EA. The patterns of transcription of the virus genomes in these three cell lines were, however, very similar: 25% without IUDR treatment, 30% immediately after treatment, and 50% (entire genome transcription) 3 days after being transferred to fresh medium. The amount of virus RNA in the cells, calculated from DNA-RNA hybridization kinetics, was proportional to the number of virus genomes per cell, suggesting that

every copy of virus DNA in these cells is actively transcribed. (8 refs)

- 79-3864 X-linked Immunocompetence and Epstein-Barr Virus Oncogenesis (Letter to Editor).** (Eng) Hecht, F. (Southwest Biomedical Res. Inst. and Genetics Center, Tempe, AZ 85281). *Lancet* 1(8121): 881; 1979.

Although X-linked genes clearly play an important role in immunocompetence, the conclusion by a previous author that Epstein-Barr virus is an oncogen in the X-linked lymphoproliferative syndrome or in Burkitt's lymphoma seems premature. (3 refs)

- 79-3865 Retinoic Acid Inhibition of Epstein-Barr Virus Induction.** (Eng) Yamamoto, N. (Institut für Virologie, Zentrum für Hygiene, Universität Freiburg, Hermann-Herder-Str. 11, 7800 Freiburg, W. Germany); Bister, K.; zur Hausen, H. *Nature* 278(5704): 553-554; 1979.

The possible interaction of retinoic acid (RA) with Epstein-Barr virus (EBV) induction by tumor promoters was studied using Raji cells. Concomitant treatment with 12-O-tetradecanoylphorbol-13-acetate (TPA: 20 nanograms/ml) and RA (0.01-50 μ M) resulted in effective inhibition of the induction of early antigens (EA) by TPA. At 10^{-5} M RA, >90% of EA induction by TPA was suppressed. The same degrees of inhibition were observed when the cells were pretreated with the various concentrations of RA. EA induction by anti-IgM and, to a lesser extent, by iododeoxyuridine was also inhibited by retinoic acid. In Raji cells superinfected with EBV derived from P3HR-1 cells, RA did not affect the percentage of EA-positive cells, even when applied at 10^{-5} M. The spontaneous induction rate of P3HR-1 and B95-8 cells was also not significantly altered by RA. The data suggest that the different inducing reagents activate the persisting genomes indirectly by a common mediator that seems to be inhibited by RA. EA induction by superinfection or spontaneous induction of virus antigens in tissue culture cells apparently follows different pathways. (15 refs)

- 79-3866 The Association Between Undifferentiated Nasopharyngeal Carcinoma and Epstein-Barr Virus Shown by Correlated Nucleic Acid Hybridization and Histopathological Studies.** (Eng) Andersson-Anvret, M. (Dept. Tumor Biology, Karolinska Institutet, S104 01 Stockholm 60, Sweden); Klein, G.; Forsby, N.; Henle, W. *IARC Sci Publ* 20: 347-357; 1978.

The association between undifferentiated nasopharyngeal

carcinoma (NPC) and Epstein-Barr virus (EBV) was investigated in parallel histological and nucleic acid hybridization studies. Biopsies were obtained in Nairobi from primary tumors of the nasopharynx and other regions of the head and neck. Serum from each patient was tested for antibodies against viral capsid antigen (VCA), the diffuse component of the early antigen complex [EA(D)], and Epstein-Barr nuclear antigen (EBNA). All 51 undifferentiated NPC biopsies were EBV-DNA-positive, and there was a mean equivalent of 31.5 viral genomes/cell. Sera from the undifferentiated NPC patients tested were EBNA-positive. Most of these patients also had high anti-EBV [VCA and EA(D)] antibody titers. Of four patients who had somewhat differentiated NPC's, two were EBV-DNA-positive with a corresponding serological picture, and two were negative with an EBV serology comparable to that of healthy donors. Of seven lymphomas localized in the nasopharynx, one was EBV-DNA-positive and corresponded histologically to Burkitt's lymphoma, whereas six others were EBV-DNA-negative. Fourteen head and neck carcinomas outside the nasopharynx were all EBV-DNA-negative. These results confirm the association of EBV DNA with undifferentiated NPC. (29 refs)

- 79-3867 Morphological Transformation of Nasopharyngeal Epithelial Cells In Vitro by Epstein-Barr Virus from B95-8 Cells.** (Eng) Huang, D. P. (Medical and Health Dept., Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong); Ho, J. H.; Ng, M. H. *IARC Sci Publ* 20: 359-368; 1978.

Attempts were made to produce experimental tumors in vitro by infecting fragments of human nasopharyngeal mucosa (NP), tonsillar mucosa, primary nasopharyngeal tumors (NPC), and primary upper respiratory or alimentary tract lesions with Epstein-Barr virus (EBV). All specimens were from Chinese subjects. According to the outgrowth ratios (the ratio of the av diameter of the infected fragment outgrowth to that of the uninfected fragment outgrowth), the NP specimens were highly susceptible to EBV infection; 19/20 showed growth stimulation in response to EBV. The other specimens responded at significantly lower frequencies. All the NPC specimens showed the presence of EBV nuclear antigen (EBNA) before EBV infection, and some of the infected NP cells displayed EBNA. After EBV infection, the NP explants showed marked changes in growth and cellular morphology. They proliferated at a much higher rate, and there were foci of cell piling and a disoriented cell distribution pattern. They showed marked cellular pleomorphism, an increased nucleus:cytoplasm ratio, many mitotic figures, and a few vesicular nuclei. Occasional multinucleate giant cells and nuclear hyperchromasia were also noted. These features indicate that the cells underwent morphological transformation. Whether malignant transformation also occurred was not determined. (22 refs)

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- 79-3868** Epstein-Barr Virus-Epithelial Cell Interaction and Its Implications in the Etiology of Nasopharyngeal Carcinoma. (Eng) Lenoir, G. (International Agency Res. on Cancer, Lyon, France); De-The, G. *IARC Sci Publ* 20: 377-384; 1978.

Three hypotheses are proposed to explain how the DNA of Epstein-Barr virus (EBV) becomes associated with nasopharyngeal carcinoma (NPC) cells and whether the presence of EBV nuclear antigen (EBNA) in the epithelial cell is a marker of the malignant state. The first hypothesis suggests that the normal epithelial cell from the nasopharyngeal mucosa is permissive for EBV infection; ie, the cell possesses EBV receptors and can replicate the virus. The second hypothesis proposes that a malignant or premalignant change is required in the epithelial cells for them to become permissive for EBV infection. If the cell were already malignant when it was infected, the virus would have no etiologic role in the transformation process (passenger hypothesis), whereas if the cell were in a premalignant state prior to infection, the virus would act as a promoting factor for tumor development. The third hypothesis states that the epithelial cells have no receptors for EBV and that infection results from a specific interaction of these cells with EBV-infected lymphocytes, involving either cell hybrid formation or a transfection process. Here again, the virus might be passive or it might act as an oncogenic factor. Recent evidence for and against these hypotheses is presented. (16 refs)

- 79-3869** Identification of the Primate Papovavirus HD as the Stump-tailed Macaque Virus. (Eng) Howley, P. M. (Lab. Pathology, NCI, Bethesda, MD 20014); Newell, N.; Shah, K. V.; Law, M. F.; Gruss, P.; Sauer, G.; Kelly, T. J. *J Virol* 30(1): 400-403; 1979.

The recently isolated primate papovavirus HD was shown to be indistinguishable from the stump-tailed macaque virus by immunofluorescent reactivity, by restriction endonuclease analysis, and by nucleic acid hybridization assay. The size and restriction pattern of the smaller HD species DNA were identical to those of the wild-type stump-tailed macaque virus; the larger HD DNA species contained a 6% insertion that may be a tandem repeat of a portion of the viral genome. (19 refs)

- 79-3870** Localization of Viral DNA-Replication in Sections of Human Warts by Nucleic Acid Hybridization with Complementary RNA of Human Papilloma Virus Type 1. (Eng) Grubendorf, E. I. (Abteilung Dermatologie der Medizinischen Fakultät der RWTH Aachen, Goethestrasse 27-29 D-5100 Aachen, W. Germany); zur Hausen, H. *Arch Dermatol Res* 264(1): 55-63; 1979.

Using complementary RNA of human papilloma virus type 1 (HPV1) and in situ hybridization techniques, the localization of viral DNA replication was studied in sections of 38 human virus acanthomas from 31 different patients. In five cases, significant labeling was detected by autoradiography. The labeling began in the first or second suprabasal cell layer and was strongly limited to the nuclei. A remarkable early beginning of the vacuolated process seemed to be correlated with the visible DNA replication. No labeling could be detected in the basal cell layer, which suggests that these cells contain only a small number of viral genomes. These findings represent the only instance of human warts definitely caused by HPV1. It is not known whether the negative hybridization results in the remaining warts indicate a scarcity of viral genomes within the sections or an infection by a different agent. (13 refs)

- 79-3871** A Map of the Sites in the Polyoma Genome Cleaved by Endonuclease *A₁l*. (Eng) Berkner, K. L. (Dept. Biological Chemistry, Univ. Michigan, Ann Arbor, MI 48109); Folk, W. R. *Virology* 92(2): 482-494; 1979.

The 29 sites in the polyoma virus genome cleaved by endonuclease (endo) *A₁l* and the 1 site cleaved by endo *Xba*I were identified. The *A₁l* fragments ranged in size from 0.9 to 37 x 10⁴ daltons. The locations of the 29 *A₁l* fragments were generally specified by at least two independent techniques. Seventeen sites were located in the early region of the genome, 12 in the late region. The *Xba*I site was located 17% of the genome away from the *Eco*RI site, toward the terminus of DNA replication. Cleavage of the early region of the genome into 17 fragments should be valuable for mapping early gene functions. (31 refs)

- 79-3872** Topography of Polyoma Virus-specific Giant Nuclear RNA Molecules Containing Poly(A) Sequences. (Eng) Lev, Z. (Div. Biology, California Inst. Tech., Pasadena, CA 91125); Kamen, R.; Manor, H. *Virology* 93(2): 445-457; 1979.

Experiments were conducted to identify the sequences adjacent to the polyadenylated [poly(A)] tracts in giant polyoma-virus specific nuclear RNA molecules to determine the order of sequences at increasing distance from the poly(A). Poly(A) nuclear RNA sedimenting faster than 30S was isolated from polyoma virus-infected cells and cleaved into fragments of various sizes by partial alkaline hydrolysis. Fragments containing poly(A) were separated from nonpoly(A) RNA. The viral sequence composition of poly(A) or nonpoly(A) fragments and of total poly(A) giant RNA was determined by hybridization to ³²P-labeled separated strands of specific restriction endonuclease fragments of polyoma virus DNA. The results of this analysis showed that in all poly(A) giant RNA molecules

transcribed from the L DNA strand, the portion adjacent to the poly(A) segment hybridized to the 3'-terminal half of the late region of the viral genome. The next portion of these chains hybridized to the remainder of the late region, the more distal part to the early region, and the portions furthest from the poly(A) contained sequences complementary to the L DNA strand of both early and late regions. This finding shows that polyadenylation of polyoma giant nuclear RNA is nonrandom and suggests that the poly(A)-linked RNA sequence(s) maps within the same region of the viral genome as the poly(A)-linked sequence found in mature cytoplasmic messenger RNA. (34 refs)

- 79-3873 Polyoma Viruses with Mutations at Endonuclease *Hind*II Site 1: Alterations at the COOH Terminus of VP1.** (Eng) Bendig, M. M. (Dept. Biological Chemistry, Univ. Michigan Medical Sch., Ann Arbor, MI 48109); Folk, W. R.; Gibson, W. *J Virol* 30(2): 515-522; 1979.

Four mutants of polyoma virus lacking endonuclease *Hind*II site 1 were isolated and characterized with respect to the VP1 coding sequence. DNA from each mutant was digested with endo *Hind*II plus endo *Eco*RI or *Bam*HI, and the products were separated by agarose gel electrophoresis. The results indicated that all four mutants retained *Hind*II site 2 but lacked *Hind*II site 1. However, none of the deletions in the mutants included the nearby *Hha*I site, which is within 20 base pairs of *Hind*II site 1. Three of the mutants had deletions that removed 0.2%-0.3% of the genome. All three deletion mutants encoded VP1 proteins that were smaller than wild-type VP1 protein and lacked one or more tryptic peptides normally found in the latter. The mutants did not differ significantly from the wild-type virus in the decay of infectivity after heating, and the mutations had no apparent effect on virus assembly or ability to transform BHK 21/13 cells. The results suggest that *Hind*II site I is at, or very near, the carboxy terminal end of the coding sequence for VP1. A model for the peptide organization in that region is presented. (31 refs)

- 79-3874 BK Virus DNA Sequence Coding for the Amino-Terminus of the T-Antigen.** (Eng) Yang, R. C. (Dept. Biochemistry, Molecular and Cell Biology, Cornell Univ., Ithaca, NY 14853); Wu, R. *Virology* 92(2): 340-352; 1979.

The nucleotide sequence between map positions of 0.540 and 0.604 on the human papovavirus BK[MM strain:BKV(MM)] genome was determined. A total of 332 strand nucleotides with the same polarity as early RNA are presented. A unique reading frame for translation was identified that starts with a potential initiation signal (ATG) at map position 0.595 and does not include any termination codon. From this initiation codon, a polypeptide

sequence of 95 amino acids could be deduced. The homology of the predicted amino acid sequences for the tumor (T) antigens of BKV(MM) and simian virus 40 (SV40) was determined. The first 10 amino acids at the amino terminus were identical. Between amino acids 11-74, there was a 78% homology of the nucleotide sequences and an 84% homology of the amino acid sequences. In the predicted amino acid sequences, between positions 1-95 for BKV(MM) and 1-98 for SV40, there were 16 arginine and lysine molecules common to both proteins. Six of 12 predicted tryptic peptides were common to both polypeptides, as were 3/6 methionine-containing tryptic peptides. At the 5' noncoding region of the T-antigen genes, a 20-nucleotide sequence that contained a high degree of self-complementation was identified just before the ATG initiation signal for each viral DNA molecule. This allowed the formation of a stable secondary structure that may be involved in regulation of the transcription or translation of T-antigen messenger RNA. (33 refs)

- 79-3875 Studies on the Defectiveness of Adeno-associated Virus (AAV). II. Effect of Growth in CV-1 Cells on the Replication of Adeno-associated Virus.** (Eng) Young, J. F. (Dept. Microbiology and Immunology, Baylor Coll. Medicine, Houston, TX 77030); Mayor, H. D. *Virology* 94(2): 342-351; 1979.

The mechanisms by which helper viruses can promote adeno-associated virus (AAV) macromolecular synthesis were studied using two lines of African green monkey kidney cells (CV-1 and Vero). AAV1 antigen production was good in CV-1 cells infected with AAV1 and simian virus 15 (SV15) and in Vero cells infected with AAV1 and herpes simplex virus type 1 (HSV1), but not in CV-1 cells infected with AAV1 and HSV1. In CV-1 cells infected with AAV1 and SV15, normal yields of AAV1 antigen were observed when HSV1 was added 6 hr later. Potentiation of AAV1 structural protein synthesis and AAV1 DNA synthesis in CV-1 cells could be accomplished with SV15, but not HSV1, helper virus. Thus, the HSV helper function for AAV protein synthesis is absent in CV-1 cells. HSV1 replication appeared to be normal in CV-1 cells, however, and AAV1 had little or no effect on its replication in CV-1 or Vero cells. Comparison of HSV protein production in CV-1 and in Hep-2 cells (an epidermoid cancer line) demonstrated the absence of HSV proteins ICPO and ICP46 in the CV-1 line following the release of a cycloheximide block. The data suggest that at least one of the HSV1 helper functions for AAV is not required by HSV1 for its own lytic cycle. (24 refs)

- 79-3876 Physicochemical Properties of DNA from Simian Adenovirus Type 38.** (Rus) Dubichev, A. G. (D. I. Ivanovskii Inst. Virology, Moscow, USSR); Dimotrov, D. H.; Parfenov, N. N.; Grigor'ev, V. B.;

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Naroditskii, B. S.; Tikhonenko, T. I. *Vopr Virusol* (2): 115-118; 1979.

Simian adenovirus type 38 (SAV-38) was isolated from a monolayer culture of primary cells and its DNA was separated by centrifugation in a CsCl gradient. The DNA preparations were assayed by high-velocity sedimentation, viscosimetry, and temperature denaturation. SAV-38 DNA had a sedimentation constant of 31.6S, a characteristic viscosity of 86.5 daltons/g, and a mol wt of 21.5 megadaltons. The av size of the DNA molecule was 10.4 nanometers. Its av buoyant density in CsCl and Cs₂SO₄ gradients was 1.7185 and 1.4295 g/cm³, respectively, which corresponds to 59.1% and 60.0% G-C pairs. (16 refs)

79-3877 Preparation of Simian Virus 40 and Its DNA. (Eng) Khoury, G. (Lab. DNA Tumor Viruses, NCI, NIH, Bethesda, MD 20014); Lai, C. J. *Methods Enzymol* 48: 404-412; 1979.

Simple methods for the production of simian virus 40 (SV40) and SV40 DNA are described. It is convenient to infect confluent monolayers of kidney cells at a multiplicity of approx 10 plaque-forming units/cell to prepare SV40 DNA. The DNA can be isolated by the selective extraction method of Hirt, which is based on the removal of high-mol-wt cellular DNA by precipitation in the presence of high salt concentrations and sodium dodecyl sulfate, or the DNA can be isolated from purified SV40 virions. The latter technique yields highly purified SV40 DNA, but it requires more time and yields less DNA than the Hirt technique. Techniques for radiolabeling and in vitro labeling of SV40 DNA are discussed. In vitro labeling has been used to obtain qualitative and quantitative information related to the integrated viral genomes in transformed cells. The yield of SV40 DNA is most easily determined spectrophotometrically. The restriction enzyme cleavage pattern of the DNA is perhaps the best criterion of its purity. (20 refs)

79-3878 Histone Modifications in Simian Virus 40 and in Nucleoprotein Complexes Containing Supercoiled Viral DNA. (Eng) Chen, Y. H. (Dept. Cell and Molecular Biology, Medical Coll. Georgia, Augusta, GA 30902); MacGregor, J. P.; Goldstein, D. A.; Hall, M. R. *J Virol* 30(1): 218-224; 1979.

This study was performed to determine whether the electrophoretic complexity of simian virus 40 (SV40) virion histones was correlated with specific degrees of acetylation. An additional aim was to determine whether SV40 nucleoprotein (NP) complexes from virus-infected cells contained similarly modified histones. NP complexes containing circular supercoiled viral DNA were extracted from infected cells and purified by differential centrifugation. The protein content of these complexes was compared by

electrophoresis on 15% acrylamide gels with the protein content of purified SV40 virions and with histones from virus-infected cells. The electrophoretic patterns of histones from each of the sources revealed several major differences. SV40 virions contained histones H3, H2B, H2A, and H4 but not H1. NP complexes and host cells contained all five major histone groups. Relative to cellular histones, virion and NP complex histones were enriched 15%-40% in histones H3 and H4. In addition to the major classes of histones, several subfractions of histones H1, H3, and H4 were observed in acrylamide gels of proteins from SV40 virions and viral NP complexes. Acetate labeling experiments indicated that each subfraction of histones H3 and H4 had a different level of acetylation. The histones from SV40 virions and NP complexes were acetylated to significantly higher levels than those of infected host cells. No apparent differences in phosphorylation of the major histone groups were observed. (43 refs)

79-3879 Simian Virus 40 Gene A Regulation of Cellular DNA Synthesis. I. In Permissive Cells. (Eng) Hiscott, J. B. (Dept. Pathology, New York Univ. Sch. Medicine, New York, NY 10016); Defendi, V. *J Virol* 30(2): 590-599; 1979.

The kinetics of host cellular DNA stimulation by simian virus 40 (SV40) *tsA58* infection was studied in two types of productively infected monkey kidney cells (AGMK secondary passage; and the TC-7 cell line) by flow microfluorometry and autoradiography. Prior to infection, the cells were maintained predominantly in the G₀-G₁ phase of the cell cycle by low (0.25%) serum concentration. Induction of host DNA synthesis and replication of viral DNA occurred in resting cultures after infection by wild-type (wt) SV40 or SV40 *dl890* at 33, 37, or 41 C, or by SV40 *tsA58* at 33 C. AGMK or TC-7 cells induced into DNA synthesis by SV40 infection did not continue cell cycle traverse but arrested in the G₂ phase with a 4N DNA content. Infection of TC-7 cells at 41 C with SV40 *tsA58* resulted in a transient induction of DNA synthesis involving a round of synthesis in approx 30% of the cells at 20-24 hr postinfection. At later times, these cells returned to the G₁ resting state, although they remained tumor antigen positive by immunofluorescence. Infection of AGMK cells with the *tsA* mutant at 41 C induced approx 50% of the cell population to progress through the S phase to G₂ + M. However, the 4N DNA population of these cells did not return to a G₁ state but remained at G₂. The results indicate that the mitogenic effect of the *A* gene product on cellular DNA is more heat resistant than its regulatory activity on viral DNA synthesis and that the extent of induction of cell DNA synthesis by the *A* gene product may be influenced by the host cell. (32 refs)

79-3880 Inhibition of SV40 Replication by 5-Azacytidine: Effect on DNA Synthesis and

Conformation. (Eng) Johnson-Thompson, M. (Dept. Biology, Univ. District Columbia, Washington, DC); Rosenthal, L. J. *Virology* 93(2): 605-608; 1979.

Simian virus 40 (SV40) DNA synthesis was inhibited by approx 50% following a 3-hr treatment with 100 µg/ml 5-azacytidine (5-AzaCR), a potent antineoplastic and antileukemic agent. There was a max inhibition of 90% by 9 hr. Cesium chloride-ethidium bromide analysis of SV40 DNA from the drug-treated infected cultures demonstrated that SV40 DNA I banded at a higher buoyant density and therefore had a decreased ability to bind ethidium bromide. Sedimentation analysis in sucrose gradients containing various concentrations of ethidium bromide indicated that these molecules were deficient in superhelical turns. Treatment of infected cultures with 5-AzaCR inhibited protein synthesis, which preceded the inhibition of DNA synthesis. It is concluded that inhibition of SV40 DNA synthesis and the conformational alterations in DNA I result from the inhibition of protein synthesis. (13 refs)

79-3881 Cell-free Translation of Simian Virus 40 16S and 19S L-Strand-specific mRNA Classes to Simian Virus 40 Major VP-1 and Minor VP-2 and VP-3 Capsid Proteins. (Eng) Prives, C. L. (Lab. DNA Tumor Viruses, NCI, Bethesda, MD 20014); Shure, H. *J Virol* 29(3): 1204-1212; 1979.

Simian virus 40 (SV40) capsid proteins VP-1, VP-2, and VP-3 were identified among the cell-free products of RNA from SV40-infected BSC-1 cells, and the approx size and subcellular distribution of their messenger RNA's (mRNA's) were assessed. The capsid proteins were synthesized in wheat germ and reticulocyte cell-free systems in response to either poly(A)-containing mRNA from the cytoplasm of infected cells or viral RNA purified by hybridization to SV40 DNA linked to Sepharose. All three viral polypeptides synthesized in vitro were specifically immunoprecipitated with anti-SV40 capsid serum. VP-2 and VP-3 were related by tryptic peptide mapping to each other but not to VP-1. The most abundant class of L-strand-specific viral mRNA, the 16S species, coded for the major capsid protein. The relatively minor 19S class directed the cell-free synthesis of VP-1, VP-2, and VP-3. Whether the 19S RNA represents more than one distinct species of mRNA is not yet clear. VP-1 mRNA could be isolated from the cytoplasm, detergent-washed nuclei, and the nuclear wash fraction. The mRNA from the nuclear wash fraction was enriched for VP-2 mRNA when compared with the other viral or cellular polypeptides. (36 refs)

79-3882 Characterization of a Fused Protein Specified by the Adenovirus Type 2-Simian Virus 40 Hybrid Ad2+ND1 dp2. (Eng) Fey, G. (Swiss Inst. Experimental Res. Cancer, CH 1066 Epalinges, Lausanne,

Switzerland); Lewis, J. B.; Grodzicker, T.; Bothwell, A. *J Virol* 30(1): 201-217; 1979.

The adenovirus type 2 (Ad2)-simian virus 40 (SV40) hybrid virus Ad2+ND1 dp2 specifies two proteins (mol wts, 24,000 and 23,000) that are, in part, products of the insertion of SV40 early DNA sequences. This was demonstrated by translation in vitro from viral messenger RNA (mRNA) that had been selected by hybridization to SV40 DNA. These two phosphorylated, nonvirion proteins were produced late in infection in amounts similar to those of AD2 structural proteins and were closely related to each other in tryptic peptide composition. The portion of SV40 DNA (map units 0.17 to 0.22 on the SV40 genome) coding for these proteins was joined to sequences coding for the amino-terminal part of the Ad2 structural protein IV (fiber). The Ad2+ND1 dp2 23,000- and 24,000-mol wt proteins were hybrid polypeptides, with about two-thirds of their tryptic peptides being contributed by the fiber protein and the remainder contributed by SV40 tumor (T) antigen. They shared with T antigen (mol wt 96,000) a carboxy-terminal proline-rich tryptic peptide. Together, the tryptic peptide composition of these proteins and the known SV40 DNA sequences suggested the reading frame for the translation of T antigen. The carboxy terminus for T antigen would then be located on the SV40 genome map next to the TAA terminator triplet at position 0.175, 910 bases away from the cleavage site of the restriction endonuclease *EcoRI*. Seven host range mutants from Ad2+ND1 dp2 were isolated that had lost the capacity to propagate on monkey cells. They did not induce detectable levels of the hybrid proteins. Three of these mutants had lost the SV40 DNA insertion that codes in part for these proteins. Thus, in analogy to the Ad2+ND1 30,000-mol wt protein, the presence of these proteins correlates with the presence of the helper function for adenovirus replication on monkey cells. (56 refs)

79-3883 Late Replicative Intermediates Are Accumulated During Simian Virus 40 DNA Replication In Vivo and In Vitro. (Eng) Seidman, M. M. (Lab. Biology Viruses, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Salzman, N. P. *J Virol* 30(2): 600-609; 1979.

The replicating molecules of simian virus 40 (SV40) DNA present in vivo and those in a nuclear extract containing replicating chromosomes (nucleoproteins) were characterized. In vitro, there was a conversion of previously labeled, replicating molecules to structures that sedimented in neutral sucrose gradients in the region of form I DNA. These molecules were actively replicating. Other products of incubation were molecules (after deproteinization) with sedimentation coefficients of 22S and 29S. A substantial portion of the 22S peak appeared to be an artifact of the in vitro replication system when it was operating under suboptimal conditions. Discrete classes of

intermediates were shown to accumulate during in vitro replication, and they were similar to the discrete classes of late replicative intermediates that accumulate in vivo. The major late form accumulated was 91% completed. The replicating chromosomes could be resolved into two distinct peaks on neutral sucrose gradients; the molecules in these peaks differed in extent of replication. The nuclear extraction procedure preferentially extracted early replicating chromosomes. (35 refs)

- 79-3884** The Initiation of Transcription of SV40 DNA at Late Time after Infection. (Eng) Laub, O. (Dept. Genetics, Weizmann Inst. Science, Rehovot, Israel); Bratosin, S.; Horowitz, M.; Aloni, Y. *Virology* 92(2): 310-323; 1979.

The initiation site for the transcription of simian virus 40 (SV40) DNA at late times after infection was localized. In vivo-labeled RNA was purified from productively infected BSC-1 cells, and in vitro-labeled RNA was purified from transcriptional complexes of SV40. The purified RNA's were denatured and fractionated by sedimentation through sucrose gradients. Labeled RNA's of various lengths were hybridized with restriction fragments of SV40 DNA of a known order. With both the in vivo- and in vitro-labeled RNA's, the shortest RNA's hybridized with a DNA fragment that spanned map units 0.67-0.76. Hybridization with this fragment decreased with successively longer RNA's, indicating that transcription initiates within this fragment or very close to it. Electron microscopic analysis of transcriptional complexes of SV40 revealed a substantial fraction (10%) with one short nascent RNA chain. The initiation site of the nascent chains was mapped at coordinate 0.67. The accumulation of transcriptional complexes with short nascent chains, initiated at coordinate 0.67, and the abundance of labeled nascent RNA's complementary to a fragment spanning map units 0.67-0.76 suggest the existence of an attenuator site in which RNA chain elongation is blocked, unless a stimulating factor is present that allows transcription to be completed. It is concluded that the joining of the 5' leader sequences to the coding region occurs by the looping out of intervening RNA sequences via intramolecular digestion and ligation. (32 refs)

- 79-3885** Simian Virus 40 Early mRNA's. I. Genomic Localization of 3' and 5' Termini and Two Major Splices in mRNA from Transformed and Lytically Infected Cells. (Eng) Reddy, V. B. (Dept. Human Genetics, Yale Univ. Sch. Medicine, New Haven, CT 06510); Ghosh, P. K.; Lebowitz, P.; Piatak, M.; Weissman, S. M. *J Virol* 30(1): 279-296; 1979.

A study was made of the structure of polyadenylated virus-specific cytoplasmic messenger RNA's (mRNA's) in mouse

and human cells transformed by simian virus 40 (SV40) and in monkey cells infected with SV40 in the presence of cytosine arabinoside by reverse transcriptase-catalyzed complementary DNA (cDNA) synthesis and cDNA sequencing. Abundant mRNA species containing splices from residues 4490 to 4557 [0.533 to 0.546 map units (mu)] and 4490 to 4837 (0.533 to 0.600 mu) were identified in both transformed and infected cells. Two principal reverse transcriptase stops were observed at the 5' termini of these mRNA's, and both occurred with approx equal frequency. The most distal of these stops was localized at residues 5152 to 5154 (0.660 mu), and the second was localized at residues 5147 to 5148 (0.659 mu). Several additional minor stops, between approx 0.62 and 0.65 mu, were also found on cDNA copied from transformed cell mRNA; in contrast, only one additional stop was present on cDNA copied from early lytic mRNA. These data suggest the presence of a principal 5' terminus of early lytic and transformed cell mRNA's at residues 5152 to 5154 and raise the possibility of additional 5' termini at one or more locations in the 0.62- to 0.659-mu region of these mRNA's. Transformed cell mRNA was also found to contain a single 3' terminus at positions 2504 and 2505 (0.153 mu); termini lying beyond this site were not detected. (63 refs)

- 79-3886** Evolutionary Relationships of the Primate Papovaviruses: Base Sequence Homology among the Genomes of Simian Virus 40, Stump-tailed Macaque Virus, and SA12 Virus. (Eng) Newell, N. (Dept. Genetics, Univ. Wisconsin, Madison, WI 53706); Shah, K. V.; Kelly, T. J. *J Virol* 30(2): 624-636; 1979.

Heteroduplex mapping was used to analyze the regions of homology between the DNA's of simian virus 40 (SV40), stump-tailed macaque virus (STMV), and SA12 virus from the chacma baboon. The SA12/SV40 heteroduplexes showed about 80% homology at an effective temperature of 33 C, whereas at 32 C, only a small amount of homology (20%) was detected between the genomes of SV40 and STMV. Results from a previous study showed about 90% homology between the genomes of SV40 and BK virus at 33 C. When the heteroduplexes were mounted for microscopy at higher effective temperatures, the fraction of duplex DNA decreased in each case, indicating the existence of considerable base mismatching in the homologous regions. When specific coding or noncoding regions of the viral genomes were compared, the extent of sequence divergence was shown to differ markedly from one region to another. In all heteroduplexes studied, there were two regions, located near the junctions between the early and late regions on the SV40 map, that were essentially nonhomologous. All of the heteroduplexes studied showed significantly greater homology in the late region than in the early region. Within the late region, the sequences coding for the major capsid polypeptide, VP1, were the most highly conserved. (50 refs)

- 79-3887 Functional Similarity Between the Early Antigens of Simian Virus 40 and Human Papovavirus BK.** (Eng) Lai, C. J. (Lab. Molecular Virology, NCI, NIH, Bethesda, MD 20014); Goldman, N. D.; Khoury, G. *J Virol* 30(1): 141-147; 1979.

The functional properties of the early antigens of simian virus 40 (SV40) and human papovavirus BK (BKV) were investigated. Infection of African green monkey kidney (AGMK) cells with BKV permitted the bidirectional replication of an early temperature-sensitive mutant (tsA) at the nonpermissive temperature (41°C). Conceivably, an early gene product [tumor (T) antigen] of BKV can substitute functionally for the defective SV40 T antigen. On the other hand, SV40 DNA replication remained undetectable in human embryonic kidney cells preinfected with BKV, suggesting that BKV early antigens alone are not sufficient to provide for the replication of SV40. Preinfection of AGMK cells with BKV restored the normal pattern of late lytic SV40 transcription, suppressing the overproduction of early RNA by an SV40 tsA mutant at the nonpermissive temperature. Furthermore, preinfection of AGMK cells with BKV supported the growth of adenovirus type 2, providing a "helper function" similar to that provided by SV40 for the growth of human adenovirus in monkey kidney cells. (32 refs)

- 79-3888 Single Strand DNA Binding of Simian Virus 40 Tumor Antigen.** (Eng) Spillman, T. (Dept. Biochemistry, Univ. Illinois, Urbana, IL 61801); Giacherio, D.; Hager, L. P. *J Biol Chem* 254(8): 3100-3104; 1979.

The affinity of tumor (T) antigen prepared from SV80 cell [human epithelial fibroblasts transformed by simian virus 40 (SV40)] for single-strand DNA was studied. T-antigen activity from crude nuclear extracts bound to native calf thymus DNA cellulose, with slight reduction of the salt concentration to 50 mM NaCl and the pH to 6.0 being required for quantitative retention of the antigen. MgCl₂ and the nonionic detergent NP-40 did not influence binding of the antigen or its elution by electrophoresis. Complement-fixing T-antigen activity from nuclear extracts was also retained by denatured calf DNA-cellulose columns under the conditions required for efficient double-strand DNA binding. The binding was not due to a minor effect caused by renaturation of a small amount of the DNA. In a filter binding assay using SV40 double-strand DNA, the addition of increasing amounts of highly purified T antigen resulted in increased binding up to saturation levels. At saturation, the ratio of antigen molecules added to DNA molecules bound was 5:1. There was a 17-fold increase in binding affinity for the single-strand calf DNA compared with the native double-strand calf DNA. (31 refs)

- 79-3889 Simian Virus 40 t Antigen Affects the Sensitivity of Cellular DNA Synthesis to**

Theophylline. (Eng) Rundell, K. (Dept. Microbiology-Immunology, Northwestern Univ., Medical and Dental Schs., Chicago, IL 60611); Cox, J. *J Virol* 30(1): 394-396; 1979.

Cellular DNA synthesis induced by simian virus 40 (SV40) mutants that lack small tumor (t) antigen was found to be sensitive to theophylline concentrations (1.0 and 1.5 mM) that did not affect wild-type SV40-induced DNA synthesis. (10 refs)

- 79-3890 Subcellular Localization of Simian Virus 40 Large Tumor Antigen.** (Eng) Soule, H. R. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Butel, J. S. *J Virol* 30(2): 523-532; 1979.

The distribution of simian virus 40 (SV40) large tumor (T) antigen in subcellular fractions from SV40-transformed hamster (H-50) and mouse (VLM) cells and from SV40-infected monkey cells was determined. The major polypeptide immunoprecipitated with anti-T serum from solubilized nuclei or from surface membranes of H-50 or VLM cells migrated at approx 96,000 daltons (96K) on polyacrylamide gels. Minor components of H-50 cells migrated at approx 68K and 56K, and VLM cells also contained anti-T-reactive polypeptides of approx 140K and 56K. The 56K component appeared to be greatly reduced in inorganic ³²P-labeled surface membrane fractions. Normal cells or cells transformed with a heterologous agent, such as polyoma virus or a chemical carcinogen, lacked immunoprecipitable T antigen. Cell fractionation was monitored by [³H]thymidine labeling, NADH-diaphorase activity, and Na⁺-K⁺-dependent ATPase activity. The results revealed only trace contamination of surface membranes by nuclei, extremely low levels of nuclear rupture during homogenization, and an approx 10-fold enrichment of surface membrane. Reconstruction experiments demonstrated that soluble T antigen failed to associate or copurify with surface membranes during fractionation procedures. These results indicate the presence of a protein in the plasma membrane of cells transformed or infected by SV40 that is immunologically indistinguishable from nuclear T antigen. (63 refs)

- 79-3891 Genetic Control of the Cytotoxic T Cell Response to SV40 Tumor-associated Specific Antigen.** (Eng) Knowles, B. B. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA 19104); Koncar, M.; Pfizenmaier, K.; Solter, D.; Aden, D. P.; Trinchieri, G. *J Immunol* 122(5): 1798-1806; 1979.

To study the genetic control of the cytotoxic T-lymphocyte (CTL) response to simian virus 40 (SV40) tumor-associated specific antigen (TASA), mice of six H-2 haplotypes were challenged in vivo with syngeneic SV40-transformed cells.

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Strains bearing the H-2b haplotype produced CTL specific for SV40 TASA, whereas those bearing the H-2d haplotype did not. F₁ hybrid mice could generate CTL only if one parent bore the H-2b haplotype. The cytotoxic response was always specific for SV40-transformed cells expressing H-2b antigens. When immune lymphocytes from both H-2b and H-2k mice were assayed after secondary in vitro restimulation, they lysed SV40-transformed target cells of the appropriate H-2 genotype. Popliteal lymph node cells from H-2b, H-2k, and H-2d mice, immunized by footpad injection of SV40 and then incubated for 14 days in vitro before assay, were cytotoxic for syngeneic SV40-transformed cells. With this immunization protocol, H-2b mice responded to SV40 TASA in association with both the H-2 K and D locus molecules, whereas H-2k mice responded to SV40 TASA in association only with the H-2 K molecule and H-2d responded to SV40 TASA in association only with the H-2 D gene product. SV40 TASA-immune lymphocytes from various F₁ mice were unable to lyse syngeneic H-2Kd or H-2Dk SV40-transformed target cells. Analysis of lymphocytes from SV40 TASA-immune F₁ hybrid (H-2b x H-2d) or H-2 congenic recombinant (H-2KbDd) mice showed inhibition of the response to SV40 TASA in association with H-2Dd. A dominant suppressor or recessive helper gene(s) is hypothesized to account for these findings. (25 refs)

- 79-3892 The Differential Effect of Interferon on T Antigen Production in Simian Virus 40-infected or Transformed Cells.** (Eng) Mozes, L. W. (Papanicolaou Cancer Res. Inst., 1155 N.W. 14th St., Miami, FL 33101); Defendi, V. *Virology* 93(2): 558-568; 1979.

The ability of interferon (IF) to inhibit tumor antigen (T Ag) production in simian virus 40 (SV40)-infected or -transformed cells was studied primarily through the use of immunoprecipitation followed by gel electrophoresis and autoradiography. Addition of IF to monkey cells prior to or subsequent to inoculation with SV40 inhibited the amount of T Ag that was synthesized late in infection. In contrast, when a similar experiment was performed with a temperature-sensitive mutant of SV40, tsA58, which does not replicate at the nonpermissive temperature, the amount of immunoprecipitable T Ag was not inhibited when IF was added at 30 hr postinfection at 40.5 C. The effect of IF on an integrated vs nonintegrated genome within the same cell population was studied in an SV40-transformed mouse cell line, H6-15, which is temperature-sensitive for the transformed phenotype and for the expression of T-antigen. In shift-down experiments, the reappearance of SV40 T Ag was insensitive to the addition of IF, whereas superinfection of H6-15 cells with polyoma virus resulted in a dose-dependent inhibition of polyoma T Ag infection. An SV40-transformed mouse cell line (nonpermissive) and two SV40-transformed human cell lines (semipermissive) were passaged in the presence of IF for four generations. Approx the same amount of labeled T Ag could be im-

munoprecipitated from IF-treated compared with control mouse cultures, whereas there was a marked decrease in the amount of newly synthesized T Ag in IF-treated human cultures. All these results are compatible with the hypothesis that IF affects differentially the expression of early viral genes whether the viral DNA is integrated or not integrated. (34 refs)

- 79-3893 Karyologically Identified Homozygous t(w18) Embryos: Extrauterine Growth Properties and Transformation by SV40.** (Eng) Kelly, F. (Service de Genetique Cellulaire, College de France et de l'Institut Pasteur, 25 rue du Docteur Roux, 75015 Paris, France); Guenet, J. L.; Condamine, H. *Cell* 16(4): 919-927; 1979.

A method was developed for the karyotypic identification of mouse embryos derived from a cross between parents heterozygous for a *t* haplotype using the marker chromosome Rb7. The method was used to examine the growth properties of embryos carrying the t(w18) mutation, both in vitro and in an ectopic site. When continued growth was observed, a contractile area appeared within the embryo. Six- and 8-day wild-type and heterozygous embryos demonstrated considerable growth during the 6-day in vitro period. The five homozygous embryos grew poorly or not at all. Two of them, however, showed a contractile area, indicating that cells of mesodermal origin in these embryos are capable of at least some multiplication and differentiation in vitro. Three-day blastocysts or 6- and 7-day embryos from crosses between heterozygous + Rb7/t(w18) + parents were injected into the testes of 6-wk-old mice. Testes were examined 17-45 days later, and the teratomas (2-10 mm in diameter) were removed, dissociated, and cultured in vitro. Of the 52 tumors obtained, 43 were heterozygous, 6 were wild-type, and 3 were homozygous mutants. These results suggest that homozygous mutants grow poorly in vivo and in an ectopic site compared with wild-type and heterozygous mutants. When the homozygous t(w18)/t(w18) teratomas were infected with simian virus 40, permanent cell lines of mesodermal origin that were capable of myoblastic or adipocytic differentiation were obtained. (17 refs)

- 79-3894 Sialic Acid Content and Growth Control of Mouse Cells Transformed by a Temperature-sensitive Mutant of SV40.** (Eng) Aoi, Y. (Inst. Medical Science, Univ. Tokyo, P.O. Takanawa 108, Tokyo, Japan). *J Med Clin Exp Theor* 9(6): 503-510; 1978.

Temperature-dependent alterations in plasma membrane glycoproteins observed in mouse cells transformed by a temperature-sensitive mutant of simian virus 40 were shown to be derived from changes in plasma membrane sialic acid levels. There was a positive correlation between sialic acid levels and loss of contact inhibition in the cells. (17 refs)

- 79-3895 Regulation of Viral Functions in Simian Virus 40-transformed Cells.** (Eng) Zouzas, D. (Dept. Pathology, New York Univ. Sch. Medicine, 550 First Ave., New York, NY 10016); Basilico, C. *Natl Cancer Inst Monogr* (48): 239-244; 1978.

A study was made of the expression of simian virus 40 (SV40)-specific tumor (T) antigen and viral transcription in SV40-transformed cells that were growing exponentially or arrested in G₁ in order to define the relationship between T antigen and cell growth and to determine the regulatory mechanisms that might control T-antigen synthesis in transformed cells. Advantage was taken of the behavior of two lines of SV40-transformed mouse 3T3 cells (ts SV3T3), which, although transformed by wild-type SV40, are temperature-sensitive for expression of the transformed phenotype. At 32 C, ts SV3T3 cells behave like standard transformants, whereas at 39 C, they become arrested in G₁ after reaching saturation density or under serum starvation. At 32 C or growing at 39 C, ts SV3T3 were 100% T-antigen positive and contained virus-specific messenger RNA. However, after G₁ arrest at 39 C, most of the cells became T-antigen negative. This seems to be caused by a lack of transcription of the integrated viral DNA, since these cells contain no appreciable amounts of SV40-specific RNA. Induction of proliferation in resting, T-antigen-negative ts SV3T3 cultures results in the reappearance of T antigen a few hours before the cells enter DNA synthesis. These results suggest that transcription of the viral genome and T-antigen expression in SV40-transformed cells are subjected to cell-cycle control. (34 refs)

- 79-3896 Simian Virus 40-Host Cell Interaction During Lytic Infection.** (Eng) Gershey, E. L. (Rockefeller Univ., New York, NY 10021). *J Virol* 30(1): 76-83; 1979.

It has been postulated that besides requiring the presence of tumor (T) antigen, the replication of simian virus 40 (SV40) DNA is dependent upon an event in the host cell cycle occurring in late G₁ or early S. This virus-host relationship was studied in exponentially growing and serum-arrested subcloned CV-1 cell cultures infected with SV40. By 24 hr postinfection (PI), 96% of the nuclei of these permissive cells contained SV40 T antigen. Analysis of the av DNA content per cell at various times PI indicated that by 24 hr, most of the cells contained amounts of DNA similar to those normally found in G₂ cells. Analysis of cell cycle distributions indicated that a G₂ DNA complement was maintained by >90% of the cells in the infected populations 24-48 hr PI. Cells continued to synthesize SV40 DNA during the first 50 hr PI, and a cytopathic effect was first observed 60 hr PI. After infection, the number of mitotic cells that could be recovered by selective detachment decreased precipitously and was drastically reduced by 24 hr. A study of the kinetics of decline in the number of mitotic cells suggests that this decline is related to an event

during the cell cycle at or near the G₁-S phase border upon which commencement of SV40 DNA replication apparently depends. It was concluded that after SV40 infection, stationary cells are induced to cycle, and the cycling cells complete one round of cellular DNA synthesis but do not divide. Although the infected cells continue to synthesize viral DNA, they do not appear able to reinitiate cellular DNA replication units. These results imply that the abundance of T antigen (produced independently of cell cycle phase) in the presence of the enzymes required for continued DNA synthesis is not sufficient for reinitiation of cellular DNA synthesis. (55 refs)

- 79-3897 Human Adenoviruses: Growth, Purification, and Transfection Assay.** (Eng) Green, M. (Inst. Molecular Virology, St. Louis Univ. Sch. Medicine, St. Louis, MO 63110); Wold, W. S. *Methods Enzymol* 48: 425-435; 1979.

Methods for growing of cells for the propagation and plaque assay of all human adenovirus serotypes are described in detail. The isolation of viral DNA and the transfection assay for determining adenovirus DNA infectivity and localizing adenovirus-transforming genes are also described. (24 refs)

- 79-3898 Nucleosome-like Structural Subunits of Intranuclear Parental Adenovirus Type 2 DNA.** (Eng) Sergeant, A. (U 102 de Virologie, Institut National de la Sante et de la Recherche Medicale, 59045 Lille Cedex, France); Tigges, M. A.; Raskas, H. J. *J Virol* 29(3): 888-898; 1979.

The intranuclear structure of parental adenovirus 2 DNA was studied using digestion with micrococcal nuclease as a probe, and the relationship of this structure to multiplicity of infection, the time course of infection, and viral transcription was examined. When cultures were infected with ³²P-labeled virions, at a multiplicity of 3,000 particles per cell, 14%-21% of parental DNA penetrated the cell and reached the nucleus. Of this parental DNA, 60% could be solubilized by extensive digestion with micrococcal nuclease. The nuclease-resistant fraction contained viral deoxyribonucleoprotein monomers and oligomers. These nucleosome-like structures contained DNA fragments that were integral multiples of a unit-length DNA of approx 185 base pairs. The monomeric DNA was similar in length to the unit-length DNA contained in cellular nucleosomes. However, the viral oligomers were slightly smaller than their cellular counterparts. DNA-DNA hybridization demonstrated that all segments of the viral genome, including those expressed as messenger RNA only at late times, were represented in the nucleosomal viral DNA. The amount of early intranuclear viral chromatin was proportional to the multiplicity of infection up to multiplicities of

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4,000 particles per cell. However, viral transcriptional activity did not increase in direct proportion to the amount of viral chromatin. Max accumulation of intranuclear viral chromatin was achieved by 3 hr after infection. The intranuclear parental viral chromatin remained resistant to nuclease digestion even at late times in infection, after viral DNA replication had begun. (40 refs)

- 79-3899 The Nucleotide Sequences at the Termini of Adenovirus-2 DNA.** (Eng) Arrand, J. R. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Roberts, R. J. *J Mol Biol* 128(4): 577-594; 1979.

The primary structure of the terminal regions of adenovirus 2 (Ad2) DNA through the inverted terminal repetition is reported, along with its significance for models of DNA replication. The nucleotide sequence of the first 156 residues from the left end and the first 134 residues from the right end of Ad2 DNA was determined by direct DNA sequencing techniques. The inverted terminal repetition was 102 nucleotide pairs long. The 5' ends of the intact DNA were resistant to the action of T4 polynucleotide kinase and the 5'→3' exonucleases from phages λ and T7. This resistance was most likely due to the covalent attachment of the 5'-terminal C residue to the terminal protein. No significant self-complementarity existed within the inverted terminal repetition, making terminal initiation of DNA replication via a self-priming mechanism unlikely. However, the terminal A + T-rich region followed immediately by a very G + C-rich region was consistent with other schemes for Ad2 replication. The left end of Ad2 DNA contained extensive sequence repetition. (35 refs)

- 79-3900 Sequence Analysis of Adenovirus DNA: Complete Nucleotide Sequence of the Spliced 5' Noncoding Region of Adenovirus 2 Hexon Messenger RNA.** (Eng) Akusjarvi, G. (Dept. Microbiology, Biomedical Center, Uppsala Univ., P.O. Box 581, Uppsala, Sweden); Pettersson, U. *Cell* 16(4): 841-850; 1979.

The complete nucleotide sequence of the 5' noncoding region of the adenovirus 2 hexon messenger RNA (mRNA) was established by sequence analysis of reverse transcripts. The transcripts were generated by extension of specific single-stranded DNA primers with reverse transcriptase after hybridization of the primers to purified hexon mRNA. The total length of the 5' noncoding region was determined to be 240 nucleotides, 202 of which constituted the spliced tripartite leader sequence, including the terminal 7m-G cap. The size of each leader segment was estimated by comparing the established mRNA sequence with the genomic sequences for the first and third leader segments. The first, second, and third segments were approx 42, 71, and 89 nucleotides long, respectively. The

leader sequence allows the occurrence of hydrogen-bonded interactions with the 3' end of 18S ribosomal RNA near the capped 5' end and close to the initiator AUG. (28 refs)

- 79-3901 Nucleotide Sequence Analysis of the Leader Segments in a Cloned Copy of Adenovirus 2 Fiber mRNA.** (Eng) Zain, S. (Cold Spring Harbor Lab., Cold Spring Harbor, NY 11724); Sambrook, J.; Roberts, R. J.; Keller, W.; Fried, M.; Dunn, A. R. *Cell* 16(4): 851-861; 1979.

Recombinant plasmids carrying reverse transcripts of adenovirus 2 (Ad2) fiber messenger RNA (mRNA) were prepared by a method involving synthesis of a complementary DNA (cDNA) copy of mRNA followed by the addition of homopolymeric tracts to the 3' termini of the resulting hybrid using terminal deoxynucleotidyl transferase. mRNA/cDNA hybrids were inserted directly at the Pst I site of the plasmid vector pBR322 after A:T tailing and established in *Escherichia coli*. One recombinant plasmid, pJAW 43, was characterized and shown to contain sequences from the main body of fiber mRNA, the three leaders common to most late Ad mRNA's and a fourth leader found in some species of fiber mRNA. The complete DNA sequence of the leader region did not contain the initiation codon AUG although this codon does occur immediately downstream from the junction between the fourth leader and the main body of the fiber mRNA. The first leader was 41 nucleotides long, the second 71, the third 88, and the fourth 181 nucleotides. The location of junctions between viral leaders and intervening sequences was determined by reference, when possible, to sequences of the Ad2 genome. All of the leader-intervening sequence junctions could be arranged so that the dinucleotides GT and AG were at the 5' and 3' ends, respectively, of the intervening sequences. This prototype sequence, which has also been recognized at or near the splice points in other eukaryotic systems, may be part of a larger unit that acts as a recognition site for specific excision-ligation events that lead to the production of mature mRNA's. (41 refs)

- 79-3902 A Maturation Protein in Adenovirus Morphogenesis.** (Eng) Persson, H. (Dept. Microbiology, Univ. Uppsala, Biomedical Center, Box 581, S-751 23 Uppsala, Sweden); Mathisen, B.; Philipson, L.; Pettersson, U. *Virology* 93(1): 198-208; 1979.

The polypeptide composition of adenovirus type 2 (Ad2) assembly intermediates was studied. A nuclear extract was prepared from HeLa S3 cells labeled with ³⁵S-methionine 15-20 hr after Ad2 infection. Assembly intermediates were separated from nuclear or young virions (NV) and mature virions by sucrose gradient centrifugation. A polypeptide with a mol wt of 50,000 daltons (50K) was present in the intermediate fraction and in trace amounts in the NV frac-

tion. Since the 50K polypeptide was not present in the mature virions and it is not known to be a precursor of any other polypeptide, it appears to be a maturation protein in virus assembly. In pulse-chase experiments with Ad2-infected HeLa cells, the labeled 50K polypeptide band was prominent after 1.5 hr but disappeared almost completely after a 6-hr chase. In vitro translation of messenger RNA selected on the l-strand of Ad2 DNA demonstrated that the viral genome specifies a polypeptide that has a similar electrophoretic mobility in sodium dodecyl sulfate-polyacrylamide gels as the 50K maturation protein. The identity between the in vitro-synthesized polypeptide and the maturation protein was further established by tryptic fingerprint analysis. Hybrid arrested cell-free translation experiments revealed that the major part of the gene for the 50K maturation protein is located within fragment *Sma*-F (map coordinates 11.3-18.1). (30 refs)

79-3903 Adenovirus Type 2 Early Polypeptides Immunoprecipitated by Antisera to Five Lines of Adenovirus-transformed Rat Cells. (Eng) Wold, W. S. (Inst. Molecular Virology, Saint Louis Univ. Sch. Medicine, St. Louis, MO 63110); Green, M. *J Virol* 30(1): 297-310; 1979.

Adenovirus type 2 (Ad2)-induced early polypeptides (EP's) were identified, and an attempt was made to determine which EP's are coded by each of the four early gene blocks. [³⁵S]methionine-labeled EP's were resolved by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Cycloheximide pretreatment followed by labeling in hypertonic medium (210-250 mM NaCl) facilitated the detection of EP's. Seven major (reproducible bands in autoradiograms) EP's were detected with mol wts of 74,000 (74K), 21K, 19K, 15K, 13.5K, 11.5K, and 11K. Minor (weaker bands) EP's of 55K, 52K, 42K, 18K, 12K, 8.8K, and 8.3K were also often seen. To identify and map the genes for virus-coded EP's, antisera were prepared against five lines of Ad-transformed cells that retain different fractions of the viral genome. The lines were F17, 8617, F4, and T2C4 transformed by Ad2 virions and 5RK (clone 1) transformed by transfection with the Ad5 *Hs*ul-G fragment (map position 0-8). The early gene blocks retained and expressed (in part) as RNA in these cells were: 5RK(I), block 1 (70% of left 8% of genome); F17, block 1; 8617, blocks 1 and 4; F4, blocks 1, 2, and 4; T2C4, blocks 1-4. The following major EP's were immunoprecipitated: 15K by all antisera; 53K and 14.5K by F17, T2C4, 8617, and F4 antisera; 11.5K by T2C4, 8617, and F4 antisera; 44K, 42K, 19K, and 13.5K by T2C4 antisera; 11K by 8617 antisera. Minor EP's of 28K, 18K, and 12K were precipitated by all antisera except 5RK(I). The 53K and 15K EP's were precipitated also from Ad2 early infected monkey cells by the F17 antiserum and by sera from hamsters bearing tumors induced by Ad1-simian virus 40. The relationships between some of the immunoprecipitated EP's were investigated by partial proteolysis. All 53K EP's are the

"same" (ie, highly related), as are all 15K EP's and all 11.5K EP's. The 15K EP is highly related to the 14.5K EP. Although less certain, all 28K EP's appear to be related, as do all 18K EP's. The T2C4-specific 44K EP is probably a dimer of the 21K glycopolyptide. The T2C4-specific 13.5K EP and the 8617-specific 11K EP appear unrelated to any other polypeptides. These immunoprecipitation data provide evidence that early gene block 1 (map position 1-11) may encode major 53K, 15K, and 14.5K polypeptides, and minor 28K, 18K, and 12K polypeptides and that all or some of the gene for 15K and 14.5K lies within map position 1-8. The surprisingly complex pattern of polypeptides coded by early gene block 1 raises the possibility that some polypeptides may be coded by overlapping "spliced" messenger RNA's. The possible block locations of the genes for the 21K, 13.5K, and 11.5K polypeptides are presented. (72 refs)

79-3904 Protein Synthesized Early after Infection Is Linked to the Termini of Adenovirus Type 2 DNA Synthesized In Vivo and In Vitro. (Eng) Yamashita, T. (Inst. Molecular Virology, St. Louis Univ. Sch. Medicine, St. Louis, MO 63110); Arens, M.; Green, M. *J Virol* 30(2): 497-507; 1979.

Studies were undertaken to determine whether the covalently bound protein (CBP) linked to the 5' termini of the human adenovirus (Ad) DNA genome is synthesized early in infection. Ad type 2-infected KB cells were incubated with hydroxyurea for 1-18 hr postinfection, the hydroxyurea was then removed, cycloheximide was added, and the viral DNA was labeled with ³H-thymidine from 18 to 23 hr postinfection. Under these conditions, only early proteins and viral DNA were synthesized, the synthesis of late viral proteins apparently being completely blocked. The DNA synthesized appeared to contain CBP attached to the terminal fragments. Through use of a soluble complex that synthesizes exclusively viral DNA as completed viral genomes in vitro, evidence was obtained that the CBP produced during the early stages of infection was attached to both termini of the in vitro-synthesized viral DNA. The data suggest that CBP is coded by an Ad type 2 early gene or by a cell gene, and that it is likely present in a viral DNA replication complex. The results are consistent with the idea that CBP functions in DNA replication. (23 refs)

79-3905 Adenovirus 5 DNA Sequences Present and RNA Sequences Transcribed in Transformed Human Embryo Kidney Cells (HEK-Ad-5 or 293). (Eng) Aiello, L. (Wistar Inst. Anatomy and Biology, 36th Street at Spruce, Philadelphia, PA 19104); Guilfoyle, R.; Huebner, K.; Weinmann, R. *Virology* 94(2): 460-469; 1979.

The viral DNA sequences present in a human embryo

kidney cell line (293) transformed from sheared adenovirus 5 (Ad5) fragments were analyzed. Using restriction endonuclease fragments as labeled probes, four copies of 12.5% of the left end of the genome and one copy of 8.7% of the right end were demonstrated in the transformed cells. Analysis of the RNA sequences transcribed in nuclei isolated from this cell line indicated that only transcripts from sequences of the left end were detected. The expression of these viral sequences was mediated by DNA-dependent RNA polymerase II. Analysis of the viral DNA from 293 cells using restriction enzymes revealed that the viral DNA was integrated and located at two sites on the cellular genome. This arrangement was stable through several cell passages. (26 refs)

- 79-3906 Hemagglutination Caused by Simian Adenovirus 7.** (Fre) Faucon-Biguet, N. (Unite de Virologie fondamentale et appliquee, C.N.R.S., 1, place Joseph-Renaut, 69371 Lyon Cedex 2, France); Samolyk, D.; Tournier, P. *CR Acad Sci [D] (Paris)* 288(5): 563-565; 1979.

Agglutination of Sprague-Dawley rat RBC by simian adenovirus 7 (SA 7: complete virion and capsid subunits) was observed at 4-32 C in the presence of heterotypic antiserum. The hemagglutination was prevented by homologous antiserum only. In view of these properties, SA 7 can be compared to the Subgroup III human adenovirus of Rosen. (7 refs)

- 79-3907 In Vitro Translation of Adenovirus Type 12-specific mRNA Isolated from Infected and Transformed Cells.** (Eng) Esche, H. (Inst. Genetics, Univ. Cologne, Cologne, W. Germany); Schilling, R.; Doerfler, W. *J Virol* 30(1): 21-31; 1979.

The early and late gene products of human adenovirus type 12 (Ad12) and the viral proteins synthesized in an Ad12-transformed Syrian hamster kidney cell line (HA12/7) were identified by translation of viral messenger RNA (mRNA) in an in vitro protein-synthesizing system. The products of the cell-free translation system were detected by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and characterized as virus-specific by immunoprecipitation. Cytoplasmic RNA was isolated from permissive KB or nonpermissive BHK cells infected with Ad12 and from the HA12/7 cells. Virus-specific RNA was selected by hybridization to Ad12 DNA covalently bound to cellulose. Viral RNA was then translated in a fractionated rabbit reticulocyte cell-free system or in wheat germ S-30 extracts. The proteins synthesized were then characterized by immunoprecipitation and electrophoresis. RNA prepared from KB cells late after infection elicited the synthesis of most of the structural polypeptides of the virion and at least two presumably nonstructural Ad12 proteins. When

viral RNA isolated early after infection of KB cells was translated in vitro, 10 polypeptides were observed: E-68K (68,000 daltons), E-50K, E-42K, E-39K, E-34K, E-21K, E-19K, E-13K, E-12K, and E-10K. Ad12-specific RNA was also isolated from the HA12/7 cells, which contain several copies of the Ad12 genome integrated in the host genome. The RNA codes for at least seven polypeptides with mol wts very similar to those of the early viral proteins. (39 refs)

- 79-3908 A Rapid Screening for the Specific DNA Sequence: Analysis of Transforming DNA Segments in Adenovirus-transformed Cells.** (Eng) Fujinaga, K. (Dept. Molecular Biology, Cancer Res. Inst., Sapporo Medical Coll., Minami-jo, Nishi-17-chome, Chuo-ku, Sapporo 060, Japan); Sawada, Y.; Uemizu, Y. *Gann* 70(2): 239-243; 1979.

Viral DNA sequences in rat cells transformed by the adenovirus type 12 (Ad12) DNA fragments *EcoRI*-C and *HindIII*-G were investigated by spot hybridization, an autoradiographic detection of nucleic acid hybrids formed between cell DNA's spotted on a membrane filter and various nick-translated Ad12 DNA fragments. In CY1 cells, a rat cell line transformed by the *EcoRI*-C fragment (left hand 16%), all of the *HindIII* fragments included in the *EcoRI*-C fragment were present. In GY1 cells, a rat cell line transformed by the *HindIII*-G fragment (left hand 7%), both *BpaI*-H and a part of *BpaI*-J, two components consisting of the *HindIII*-G fragment, were found. The Ad12 *BpaI*-H fragment (left hand 4.5%) of the Ad12 DNA molecule, which represented approx 60% of the Ad12 transforming DNA sequences (Ad12 *HindIII*-G), was also found in GY1. The spot hybridization technique will be useful for detecting viral nucleic acid sequences in cells and for investigating the viral etiology of tumors and transformed cells. (17 refs)

- 79-3909 Detection and Quantitation of Adenovirus Type 12 Transforming DNA Segments and Their Application in Etiological Studies of Human Neoplasia.** (Eng) Fujinaga, K. (Dept. Molecular Biology, Cancer Res. Inst Sapporo Medical Coll., Sapporo 060, Japan); Yano, S.; Ojima, S.; Sawada, Y. *IARC Sci Publ* 20: 403-412; 1978.

The detection and quantitation of the transforming DNA sequence of adenovirus type 12 (Ad12) are described. Rat cells transformed by the *EcoRI*-C (CY-1) fragment of Ad12 DNA contained most, if not all, of the *HindIII*-G fragment (1.69 copies/haploid cell DNA quantity) but only a part of the *HindIII*-I fragment. A portion of the *HindIII*-G sequence was demonstrated in DNA from rat cells transformed by the *HindIII*-G fragment (GY-1) of Ad12 DNA, and the *HindIII*-I fragment was absent from these cells. Thus,

the transforming gene(s) of Ad12 exists in the *Hind*III-G fragment of the viral DNA molecule, which represents only 7.2% of the left end of the molecule. Using DNA-DNA reassociation techniques the Ad12 transforming gene sequence was detected in Ad12-transformed rat cells (W-3), but not in Raji or P3HR-1 Burkitt's lymphoma cell lines, KB cells, cells from patients with acute myelogenous leukemia, normal cells (NC37), or in normal human spleen, hamster embryo, AKR mouse embryo, or Fischer rat embryo tissues. The absence of Ad12 transforming genes in the DNA of the human tumor cells suggests that neither Ad12 nor the closely related Ad31 is the major cause of these human neoplasias. (17 refs)

- 79-3910 Enhancement of Adenovirus Transformation by Pretreatment of Rat Embryo Cells with Estrogenic and Androgenic Hormones (40456).** (Eng) Vanderpool, E. A. (Dept. Microbiology, Coll. Medicine, Howard Univ., Washington, DC 20059); Roane, P.; Turner, W. *Proc Soc Exp Biol Med* 160(4): 389-395; 1979.

The influence of sex steroids on the in vitro transformation of rat embryo cells (REC) by adenovirus type 12 (Ad12) was studied. Pretreatment with 17 β -estradiol (ED), estrone, or testosterone increased the number of identifiable foci in Ad12-infected RFEC by six- to eightfold. Transformed foci also appeared earlier in the hormone-treated cultures than in the untreated cultures. Male and female REC responded similarly to the hormone treatments. The mean number of transformed foci decreased as the concentration of ED or testosterone increased from 5 to 200 μ g/ml. Female REC appeared to be more responsive to Ad12-induced transformation than male cultures. Treatment with ED or testosterone increased viral adsorption to REC. ED-treated Ad12-transformed cells produced tumors in 9/11 rats and 3/5 hamsters after inoculation, the tumors appeared 5-10 days earlier in animals injected with hormone-treated cells. Characteristic viral cytopathic effects were markedly diminished in cultures treated with 1-5 μ g/ml of hormone. It is concluded that sex steroids play an important and perhaps a determinant role in virus-induced transformation in vitro. (14 refs)

- 79-3911 Demonstration of Hepatitis B Virus Surface Component in Human Hepatocellular Cancer Cells.** (Eng) Nayak, N. C. (Dept. Pathology, All India Inst. Medical Sciences, New Delhi, India); Sachdeva, R.; Dhar, A.; Seth, H. N. *Indian J Med Res* 69: 161-167; 1979.

Paraffin sections of livers from autopsied patients with hepatocellular carcinoma (HCC) were examined for the presence of hepatitis B virus surface (HBsAg) and core (HBcAg) antigens. Of 18 livers, 16 were cirrhotic and 2 had features of chronic hepatitis. The nonneoplastic areas of all 18 livers contained HBsAg-positive cells, and in 5 livers,

HBsAg was also found in the cytoplasm of tumor cells. The tumors in which HBsAg was demonstrated were of the well- or moderately well-differentiated trabecular types; all were associated with cirrhosis. HBsAg was also observed in the nuclei of nontumorous hepatocytes in five livers. HBcAg was not found in any of the tumors, although some livers showed this antigen in nonneoplastic areas. These findings corroborate other data pointing to a possible etiologic role of hepatitis virus in human HCC. (27 refs)

- 79-3912 Isolation of a Non-Tumor-inducing Mutant of the Ti Plasmid of *Agrobacterium tumefaciens* Strain B₆.** (Eng) Rapp, B. J. (Dept. Biochemistry, Univ. Missouri, Columbia, MO 65201); Kemp, J. D.; White, F. *Can J Microbiol* 25(3): 291-297; 1979.

A nonpathogenic mutant of *Agrobacterium tumefaciens* strain B₆ was isolated, and its properties were compared with those of the parental strain in an effort to localize the mutation. Strain B₆ was treated with mitomycin C, and only 1/200 single-colony isolates (B₆-95) was avirulent when inoculated into sunflower plants, and contained plasmid DNA. Kinetic analysis of DNA reannealing showed that total DNA homology and plasmid DNA homology between B₆ and B₆-95 was at least 90%. The length of both plasmids was 58 μ m. Plasmid DNA from both B₆ and the mutant was digested with endonucleases, and the fragments were separated by agarose gel electrophoresis. In all cases, the pattern for B₆ was identical with that of B₆-95. The Ti plasmid from B₆ and the mutant was transferred to an avirulent, plasmidless strain of *A. tumefaciens* by in vitro conjugation and transformation. All of the B₆ transconjugants and transformants were virulent, whereas all of the mutant transconjugants and transformants were avirulent. Electrophoretic patterns of endonuclease-digested plasmid DNA from the transformants were identical to those of plasmid DNA from B₆. Therefore, it is concluded that the virulence mutation lies on the Ti plasmid. (21 refs)

- 79-3913 Electron Microscopical and Biochemical Investigations on Rebra Viruses in Spleen Tissue in Malignancy.** (Eng) Warnaar, S. O. (Pathological Lab., Univ. Medical Center, Wassenaarseweg 62, Postbus 9603, NL-2300 RC Leiden, Netherlands); te Velde, J.; den Otlander, G. J.; Prins, F.; Mooren, H.; van Muijen, G. N. *J Cancer Res Clin Oncol* 93(2): 137-147; 1979.

Spleen tissue from five patients (2 with hematological malignancies and 3 with hematological diseases) and one control subject was analyzed biochemically for the presence of reverse transcriptase (RT) and examined electron microscopically for viruslike particles. In two patients (1 with non-Hodgkin's lymphoma and 1 with a myeloproliferative disorder), RT activity was found in

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fractions of homogenized tissue with a density of 1.16 and 1.24 g/cm³ before and after treatment with nonionic detergents, respectively. No RT activity was detected in the other four patients, one of whom suffered from hairy cell leukemia. Viruslike particles were detected by electron microscopy in the two spleens that were positive for RT activity. No viruslike particles were detected in the other four spleens. Thus, although the presence of retra virus or RT could not be demonstrated in the spleens of both hematological malignancy patients, it was not found in the spleens of the normal subjects and the patients with reactive conditions. Therefore, tests for RT may be helpful in differentiating proliferative from reactive disorders. The role of retra viruses in the genesis of hematological malignancies in humans is unclear. (24 refs)

- 79-3914 **Cell Transformation.** (Eng) Pastan, I. (NIH, Room B27, Building 37, Bethesda, MD 20014). *Methods Enzymol* 48: 368-370; 1979.

The characteristics of transformed cells include the ability to grow in the absence of substratum, rapid glucose metabolism, abnormal morphology, increased ability to be agglutinated by plant lectins, and decreased adhesion to substratum. It is possible to transform almost all cells in a culture within a few days with RNA tumor viruses, whereas DNA tumor viruses generally transform only a small fraction of the cells they infect. (13 refs)

- 79-3915 **Possible Role of Mumps Virus in the Etiology of Ovarian Cancer.** (Eng) Menczer, J. (Dept. Obstetrics and Gynecology, Chaim Sheba Medical Center, Tel Hashomer, Israel); Modan, M.; Ranon, L.; Golan, A. *Cancer* 43(4): 1375-1379; 1979.

The possible role of mumps virus in the etiology of ovarian cancer (OCA) was studied using 84 OCA patients and 84 controls with nonmalignant conditions matched by age and ethnic origin. Significantly fewer OCA patients than controls reported a history of clinical mumps, and the geometric mean titers of complement-fixing (CF) mumps antibodies were significantly lower in the cancer patients than in the controls. The titers did not vary significantly with age or ethnic origin. The distribution of CF antibody titers was similar in OCA patients with negative and positive histories of clinical mumps; a similar situation was found among controls. Thus, most of the negative clinical histories among both OCA patients and controls represented subclinical infections and not lack of contact with the virus. CF antibody titers did not differ among OCA patients who had received radiotherapy and/or chemotherapy and those who had recently undergone surgery. The results suggest that an immunological competence enables the development of OCA possibly through a direct etiologic role of mumps virus. (33 refs)

- 79-3916 **Early Molecular Events During the Interaction of Enveloped Riboviruses with Cells. II. A Kinetic Study.** (Eng) Perram, J. W. (Dept. Mathematics, Odense Univ., DK-5000 Odense, Denmark); Reimann, B.; Klenk, H. D.; Nicolau, C.; Polansky, O. E. *Biophys Struct Mech* 5(1): 25-32; 1979.

A kinetic model was constructed to describe the migration of the fluorescence label 1,6-diphenylhexatriene (DPH) in both directions when enveloped viruses labeled with DPH in the envelopes are in contact with unlabeled cells or when cells labeled in their membranes are in contact with unlabeled envelope viruses. Two types of receptor sites were assumed to exist on the cell surface: P sites, where physical adsorption of viruses may take place, and B sites where binding of penetrating viruses only may occur. Differential equations for label migration for different ratios of the number of viruses: number of sites were solved for different fractions of P and B sites. It is likely that two transfer mechanisms occur. With nonpenetrating viruses, transfer takes place by reversible diffusion from the labeled lipid bilayer to the label-free component. Labels also move from the penetrating viruses by a similar process prior to penetration of these viruses into the host cells. The resulting curves do not match the experimental ones precisely, but they are in broad agreement with the actual complex system investigated. It is concluded that coupling between adsorption at the different types of sites and label transfer can account for the observed kinetic behavior. No simple first-order reaction could account for the different half-lives measured for label migration in the forward and reverse reactions. (4 refs)

- 79-3917 **Early Molecular Events in the Interaction of Enveloped Viruses with Cells. I. A Fluorescence and Radioactivity Study.** (Eng) Nicolau, C. (Institut für Strahlenchemie im Max-Planck-Institut für Kohlenforschung, Stiftstrasse 34, D-4330 Mulheim a.d. Ruhr, W. Germany); Klenk, H. D.; Hildenbrand, K.; Reimann, B.; Reimann, A.; Bauer, H. *Biophys Struct Mech* 5(1): 11-23; 1979.

Molecular transfer between viruses and cells was investigated using the fluorescence depolarization of 1,6-diphenylhexatriene (DPH). Viruses studied were active and inactive influenza A virus, Newcastle disease virus (NDV), and Rous sarcoma virus acting on chick embryo fibroblasts (CEF) and susceptible (C/E) and nonsusceptible (C/B) chicken cells. Suspensions of virus (10¹¹ particles/ml) or cells (1 x 10⁶ cells/ml) were incubated with a tetrahydrofuran soln of DPH for 30 min. The polarization degrees and av rotational correlation times of DPH were measured in labeled viruses mixed with unlabeled cells and vice versa. The virus envelopes were significantly more rigid than the host cell membranes, which may be a result of restriction of label motion by one of several factors: differences in degree of fatty acid saturation, differences in

the cholesterol:phospholipid molar ratio, or differences in protein composition. When labeled viruses were mixed with unlabeled CEF, the degree of polarization decreased markedly with time. When labeled cells were mixed with unlabeled virus, fluorescence polarization increased with time. This indicates a rapid transfer of label from the labeled to the unlabeled lipid bilayer, irrespective of fluidity differences of lipid composition. Studies with CEF exposed to fowl plaque virions containing ^{14}C -labeled cholesterol and ^3H -choline-labeled phospholipid indicated that the change in polarization was caused by migration of DPH while the viruses are adsorbed on the cells. Kinetic studies demonstrated that the rate of DPH transfer from virus to cells and cells to virus is similar for the penetrating viruses but that DPH transfer from virus to cells is much faster than transfer from cells to virus in mixtures of nonpenetrating virus and cells. (30 refs)

- 79-3918 Avian Acute Leukemia Viruses MC29 and MH2 Share Specific RNA Sequences: Evidence for a Second Class of Transforming Genes.** (Eng) Duesberg, P. H. (Dept. Molecular Biology, Univ. California, Berkeley, CA 94720); Vogt, P. K. *Proc Natl Acad Sci USA* 76(4): 1633-1637; 1979.

The genome of the defective avian tumor virus MH2 was identified as a RNA of 5.7 kilobases by its presence in different MH2-helper virus complexes and its absence from pure helper virus, by its unique fingerprint pattern of RNase T1-resistant (T1) oligonucleotides that differed from those of two helper virus RNAs, and by its structural analogy to the RNA of MC29, another avian acute leukemia virus. Two sets of sequences were distinguished in MH2 RNA: 66% hybridized with DNA complementary to helper-independent avian tumor viruses and were termed group-specific, and 34% were MH2-specific. The percentage of specific sequences is a minimal estimate because the MH2 RNA used was about 30% contaminated by helper virus RNA. No sequences related to the transforming *src* gene of avian sarcoma viruses were found in MH2. MH2 shared three large T1 oligonucleotides with MC29, two of which could also be isolated from a RNase A- and T1-resistant hybrid formed between MH2 RNA and MC29 specific cDNA. These oligonucleotides belong to a group of six that define the specific segment of MC29 RNA described previously. The group-specific sequences of MH2 and MC29 RNA shared only the two smallest of about 20 T1 oligonucleotides associated with MH2 RNA. It is concluded that the specific sequences of MH2 and MC29 are related, and it is proposed that they are necessary for, or identical with, the *onc* genes of these viruses. These sequences would define a related class of transforming genes in avian tumor viruses that differs from the *src* genes of avian sarcoma viruses. (28 refs)

- 79-3919 Integration of Different Sarcoma Virus Genomes into Host DNA: Evidence Against**

Tandem Arrangement and for Shared Integration Sites. (Eng) Akiyama, Y. (Dept. Microbiology, Univ. Southern California, Sch. Medicine, Los Angeles, CA 90033); Vogt, P. K. *Proc Natl Acad Sci USA* 76(5): 2465-2469; 1979.

Integration sites for avian leukosis and sarcoma viruses in host DNA were investigated via transfection of secondary chicken embryo fibroblast cultures with high-mol-wt DNA from doubly-infected chicken embryo cells. Most foci induced by DNA from doubly-infected cells produced only one type of virus, regardless of whether the transfecting DNA was unsheread (approx 50-54S) or sheared (approx 8-10S). The small percentage of doubly producing, DNA-induced foci could be accounted for by the frequency with which multiple DNA molecules established infection in the same cell. When the two infecting viruses were varied with respect to multiplicity or time of infection, the initial infecting virus or the virus of higher multiplicity of infection was recovered at higher frequency in the foci produced by the extracted DNA. Cells uniformly infected with avian leukosis virus could be transformed by superinfection with an avian sarcoma virus from a different envelope subgroup. Infectious DNA recovered from such cells contained 3-10 ID₅₀ (50% infectious dose) units of leukosis virus per μg but only 0.3-0.4 ID₅₀ of sarcoma virus. DNA from cells infected with sarcoma virus alone contained three sarcoma virus ID₅₀/ μg . These results suggest that, even though a second virus integrates with lower efficiency into preinfected cells, there is an incomplete block of integration sites by the first virus. (24 refs)

- 79-3920 Structural Studies on Oncornavirus-related Sequences in Chicken Genomic DNA: Two-Step Analyses of *EcoRI* and *BglI* Restriction Digests and Tentative Mapping of a Ubiquitous Endogenous Provirus.** (Eng) McClements, W. (Dept. Cell Biology, Roche Inst. Molecular Biology, Nutley, NJ 07110); Hanafusa, H.; Tilghman, S.; Skalka, A. *Proc Natl Acad Sci USA* 76(5): 2165-2169; 1979.

DNA from a variety of uninfected chicken cells was analyzed for endogenous retrovirus sequences using restriction endonuclease digestion, ion-exchange chromatography, agarose gel electrophoresis, and a ^{32}P -labeled avian leukosis viral RNA probe. Pooled RBC DNA from five mature chickens negative for group-specific antigens and chicken helper factor (gs- chf-) contained sequences homologous to Rous-associated virus type 2. One simple pattern was identified in a gs- chf- individual that was common to all individuals tested. A tentative restriction map was derived for this and one other gs- chf- endogenous provirus. Other gs- chf- individuals and individuals with other phenotypes showed more complicated patterns that often included additional bands and, probably, additional proviruses. RNA from an avian sarcoma virus was used to detect cellular sequences (*sarc*) homologous to the viral transforming gene (*sarc*). Results revealed that a single

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restriction endonuclease *EcoRI* fragment of 13×10^6 daltons contained the majority of these sequences, and they confirmed that they were not adjacent to the endogenous provirus. (43 refs)

in 70S RNA preparations of LA23, even when the virus was passaged successively 12 times at high and low multiplicity of infection and at both the permissive and nonpermissive temperatures. These fingerprint analyses do not exclude the possibility that small deletions or point mutations that would not affect the large T_1 oligonucleotides have occurred in the region of the *onc* gene. (18 refs)

79-3921 Protected Deoxyribonucleoside-3' Aryl Phosphodiester as Key Intermediates in Polynucleotide Synthesis. Construction of an Icosanucleotide Analogous to the Sequence at the Ends of Rous Sarcoma Virus 35S RNA. (Eng) Gough, G. R. (Dept. Biological Sciences, Purdue Univ., W. Lafayette, IN 47907); Singleton, C. K.; Weith, H. L.; Gilham, P. T. *Nucleic Acids Res* 6(4): 1557-1570; 1979.

79-3923 Inhibition of Nucleoside and Sugar Transport into Cells by an Oncostatic Methylase Inhibitor, 5'-Deoxy-5'-isobutylthioadenosine (SIBA). (Eng) Pierre, A. (Institut de Chimie des Substances Naturelles, C.N.R.S., 91190 Gif-sur-Yvette, France); Robert-Gero, M. *FEBS Lett* 101(2): 233-238; 1979.

Techniques for constructing the icosamer d(G-C-C-A-T-T-T-T-A-C-C-A-T-T-C-A-C-C-A)-rC, corresponding to a ribonucleotide sequence located at the 3' and 5' ends of Rous sarcoma virus 35S RNA, are described. Several modifications were incorporated into the phosphotriester strategy for chemical synthesis of oligodeoxyribonucleotides. These included high-yield methods for the preparation and isolation of $O^{5'}$,N-protected deoxyribonucleoside-3' p-chlorophenyl phosphates that serve as key intermediates and the elimination of some superfluous manipulation and purification steps commonly used in the synthesis of oligonucleotide blocks. Two new arylsulfonyl nitroimidazole derivatives were also prepared and found to be highly effective agents for internucleotide bond formation. The icosamer was constructed for use as a specific hybridization probe to determine the number of copies of this sequence present in circular proviral DNA and to study the mechanism of provirus integration. (22 refs)

The effect of 5'-deoxy-5'-S-isobutylthioadenosine (SIBA) on the uptake of nucleosides and 2-deoxy-D-glucose (2-DG) by normal and Rous sarcoma virus (RSV)-transformed chick embryo fibroblasts (CEF) was studied. Half maximal inhibition of uridine uptake by normal CEF was observed with 50 μ M SIBA, and half maximal inhibition of thymidine uptake by the trichloroacetic acid-soluble and -insoluble fractions of CEF was achieved with 100 and 75 μ M SIBA, respectively. SIBA at ≤ 500 μ M inhibited nucleotide transport in phosphorylating and non-phosphorylating (2-DG-treated) cells to the same extent. In the presence of 1 mM SIBA, 2-DG uptake by normal and transformed cells was reduced by approx 80%. Phosphorylation in vitro was not affected and the proportion of 2-DG-6-phosphate to 2-DG did not change. SIBA also inhibited RSV-induced transformation paralleled the inhibition of increased 2-DG uptake by transformed cells. Among several synthetic analogues, the best inhibitors of cell transformation had the lowest I_{50} (concentration producing 50% inhibition) for uridine and 2-DG. The data suggest an interaction between SIBA and the cell surface. (15 refs)

79-3922 Characterization of the Genomic RNA from a Rous Sarcoma Virus Mutant Temperature Sensitive for Cell Transformation. (Eng) Darlix, J. L. (Departement de Biologie Molculaire, Universite de Geneve, 30, quai Ernest-Ansermet, CH-1211, Geneva 4, Switzerland); Levray, M.; Bromley, P. A.; Spahr, P. F. *Nucleic Acids Res* 6(2): 471-485; 1979.

79-3924 Effect of Growth Conditions on the Content of the Major Groups of Carbohydrates in Chick Embryo Fibroblasts. (Eng) Roll, D. E. (Dept. Biochemistry, Univ. Illinois, Urbana, IL 61801); Weber, M. J.; Conrad, H. E. *Cancer Res* 39(7): 2550-2555; 1979.

The genome of the LA23 temperature-sensitive (ts) mutant of the Prague B (Pr-B) strain of Rous sarcoma virus (RSV) was characterized by fingerprint and sequence analyses of the large T_1 oligonucleotides. Thirty-eight large T_1 oligonucleotides were separated by two-dimensional gel electrophoresis and ordered relative to the 3' poly(A) end of the RNA subunit. Only three of the LA23 T_1 oligonucleotides that are markers for the transforming (*onc*) gene were similar to the T_1 oligonucleotide *onc* gene markers in the RNA of Pr-A and Pr-B strains of RSV. This indicates that the *onc* gene region of Pr-A or Pr-B has been heavily mutated to produce that of the mutant LA23. A remarkable stability was observed in all T_1 oligonucleotides

The levels of glycogen, hyaluronic acid, chondroitin sulfates, N-acetylneuraminic acid, all of the monosaccharide components of the glycoprotein and glycolipid fractions, and the monosaccharide phosphate pools were measured in cultured chick embryo fibroblasts. Under all growth conditions, the glycogen plus the glucose phosphate pool contained approx 50% of total monosaccharide content of the cells. However, marked qualitative and quantitative alterations were found in the glycoprotein, glycolipid, and mucopolysaccharide fractions when grow-

ing cells reached confluence, when the growth temperature was shifted from 36 to 41 C, or when the cells were transformed with Rous sarcoma virus. From 65% to 95% of the total monosaccharide residues in these complex carbohydrates were found in the glycoprotein fraction; the glycolipids contained 5%-10% of the residues and the mucopolysaccharides contained 5%-25%. Changes in the complex carbohydrates in normal cells following changes in cell density or growth temperature were so great that they obscured any transformation-dependent changes that might have occurred consistently in the virus-infected cells under different growth conditions. (49 refs)

- 79-3925 Reticuloendotheliosis Virus: Detection of Immunological Relationship to Mammalian Type C Retroviruses.** (Eng) Charman, H. P. (Biological Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Gilden, R. V.; Oroszlan, S. *J Virol* 29(3): 1221-1225; 1979.

To determine whether there is an immunological relationship between reticuloendotheliosis virus (REV) and mammalian C-type retroviruses, an interspecies competition radioimmunoassay was developed using iodinated REV p30 and a broadly reactive antiserum to mammalian virus p30's. Rauscher murine leukemia virus, hamster leukemia virus, rat leukemia virus, and feline leukemia virus all gave equivalent slopes in the assay, as did REV. Gibbon ape leukemia virus and simian sarcoma-associated virus competed efficiently but not completely. RD-114 and baboon viruses gave complete competition, but they had slightly reduced slopes compared with the murine leukemia virus group. These results are consistent with the previously shown existence of three subgroups of mammalian p30's. These findings provide evidence that the REV group should be closely aligned with the mammalian C-type viruses. Since REV is not represented in normal avian cell DNA, it may represent a mammalian C-type virus reintroduced in Aves. If this speculation is correct, this would be the first case of interclass transmission among the Retroviridae. (22 refs)

- 79-3926 Lymphomas Resembling Lymphoid Leukosis in Chickens Inoculated with Reticuloendotheliosis Virus.** (Eng) Witter, R. L. (Dept. Agr., Science Education Admin.--Agricultural Res., Regional Poultry Res. Lab., 3606 E. Mount Hope Rd., East Lansing, MI 48823); Crittenden, L. B. *Int J Cancer* 23(5): 673-678; 1979.

The F₁ progeny of isolator-reared line 151_s male chickens and line 7_s females were inoculated with the chick syncytial (CS) strain of reticuloendotheliosis virus (REV) as 6-day embryos by the yolk sac route or when 1 day old by the intraabdominal or iv routes and held for long-term evalua-

tion. Chickens in both groups died from lymphoid neoplasia soon after the 17th week. After 345 days of observation, tumors were found in 6/13 embryo-inoculated chickens and in 13/14 chickens inoculated at 1 day of age. One embryo-inoculated chicken developed a myxosarcoma; the other 18 tumors were identical to those of lymphoid leukosis. The neoplasms were characterized by nodular or diffuse involvement of the liver. Tumors were commonly observed in the bursa of Fabricius, the gonads, spleen, kidney, and lungs. Most tumors were composed of lymphoblastic cells with large nuclei and prominent nucleoli; mitotic figures were common. None of the nine uninoculated control chickens developed tumors. Embryo-inoculated chickens remained viremic with REV through 33 wk, but no REV antibodies were detected. REV antibody was detected at 10 wk in the chickens inoculated at age 1 day, but whole-blood samples were negative for REV at 10 and 33 wk. Membrane immunofluorescence tests detected no Marek's disease tumor-associated surface antigen on liver tumor cells, but most of the large lymphoblasts were strongly positive for surface IgM. These results suggest that the neoplasms were not caused by exogenous avian leukosis virus or Marek's disease virus, but were induced by an unknown mechanism by the REV inoculum. (47 refs)

- 79-3927 Minor DNA Homology Between Herpesvirus of Turkey and Marek's Disease Virus?** (Eng) Lee, Y. S. (Lab. Molecular Virology, Life Sciences, Inc., 2900 72nd St. N., St. Petersburg, FL 33710); Tanaka, A.; Silver, S.; Smith, M.; Nonoyama, M. *Virology* 93(1): 277-280; 1979.

The degree of homology between Marek's disease virus (MDV) and herpesvirus of turkeys (HVT) DNA was investigated by DNA-DNA reassociation kinetics and by hybridization using the Southern blotting technique. There was no detectable homology between HVT and MDV DNA by reassociation kinetics. In the blotting technique, MDV DNA (0.75 µg) or HVT DNA (0.25 µg) was digested with *EcoRI* endonuclease and electrophoresed in 0.5% agarose gel. The DNA fragments were transferred to a nitrocellulose filter paper, which was baked at 80 C for 2 hr and hybridized with ³²P-labeled viral DNA of high specific activity. Autoradiography of the hybridized fragments indicated that homologous sequences between MDV and HVT DNA are within 1%-4%. This degree of homology between MDV and HVT DNA of 10⁶ daltons is equivalent to 1.5-6 coding genes, unless sequences are scattered. The HVT used for the homology experiments was effective as a vaccine in chickens against MDV-induced tumors. It should be determined whether the common sequence in HVT DNA and MDV DNA is related to the antigenic similarity shared by the two viruses, a similarity that is responsible for the successful vaccination of chickens against Marek's disease. (16 refs)

- 79-3928 The Role of the Macrophage in Marek's Disease: In Vitro and In Vivo Studies.** (Eng)

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Haffer, K. (Dept. Foods and Nutrition, Purdue Univ., W. Lafayette, IN 47907); Sevoian, M.; Wilder, M. *Int J Cancer* 23(5): 648-656; 1979.

Macrophages (MP's) from S- and K-strain Leghorn chickens [susceptible and resistant to Marek's disease (MD), respectively] were studied to determine their contribution to the dynamics of MD infection, tumorigenesis, and genetic resistance to this disease. In vitro studies demonstrated that MP's from both strains had similar responses toward the JM strain MD virus and JM-1 tumor cells. The MP's phagocytized JM virus, but the enveloped virus did not replicate within the MP or induce antigenic changes in the cell membrane. JM-1 tumor cells were destroyed by both cytolytic and phagocytic mechanisms. The in vivo selective suppression of MP functions by anti-MP serum or trypan blue inoculations resulted in significantly elevated virus titers and increased tumorigenesis, compared with infected, nonsuppressed or with noninfected control birds. The results indicate that genetic susceptibility or resistance to MD, as exhibited by S- and K-strain chickens, respectively, is not controlled by MP's. The role of the MP in MD infection appears to be specifically one of surveillance. (51 refs)

79-3929 Infection With LDH Virus Alters Host Response to Tumours. (Eng) Henderson, D. C. (Dept. Pathology, Royal Coll. Surgeons of England, Lincoln's Inn Fields, London, England); Chang, R. W.; Turk, J. L. *Br J Cancer* 39(4): 453-456; 1979.

The effect of infection with lactic dehydrogenase-elevating virus (LDV) on the resistance of splenectomized and intact BALB/c mice to ip inoculation of methylcholanthrene-induced (Meth A) tumor cells was studied. Splenectomy (Sp-x) alone had no protective effect on the growth of LDV-free Meth A tumor cells in virus-free mice at any cell dose, and it did not alter the growth of virus-infected tumor cells in these mice. However, in mice infected with LDV, Sp-x exerted a protective effect, as indicated by an increase in survival time and an overall increase in the number of mice surviving a dose of 10^3 tumor cells. The prognosis was better when LDV infection occurred shortly (within 24 hr) after Sp-x. These findings emphasize the need to screen for the presence of the ubiquitous LDV virus, especially in experimental systems in which tumor lines are maintained by passage in mice. (12 refs)

79-3930 Characterisation of RNA-directed DNA Polymerase in the Milk of Strain ICRC Mice. (Eng) Dumaswala, R. U. (Biology Div., Cancer Res. Inst., Tata Memorial Centre, Parel, Bombay 400012, India); Talageri, V. R.; Karande, K. A.; Joshi, B. J.; Ranadive, K. J. *Indian J Biochem Biophys* 15(6): 493-496; 1978.

The reverse transcriptase (RT) of purified mouse mammary tumor virus (MuMTV) obtained from the milk of ICRC

and C3H (Jax) mice was studied. The RT of ICRC MuMTV was able to utilize various synthetic and natural template-primers for DNA synthesis, poly(rA)-oligo dT) 12-18 being the most efficient primer. Mg^{2+} was preferred to Mn^{2+} for the template-primers and polymerases studied. The $Mg^{2+}:Mn^{2+}$ ratio for the MuMTV RT from ICRC milk using rA-dT template-primer was approx 2, whereas it was approx 6.6 for C3H MuMTV RT. When inorganic phosphate (Pi) was added to the assay system, higher $Mg^{2+}:Mn^{2+}$ ratios were observed. Addition of 10 mM Pi inhibited the utilization of template-primers in the presence Mg^{2+} and, to a greater extent, in the presence of Mn^{2+} . (rA)-(dT)12-18 was more sensitive to Pi inhibition than was (rC)-(dG)12-18. The inhibition due to Pi was much less in the case of C3H MuMTV RT. (22 refs)

79-3931 Identification of the Messenger RNAs Coding for the gag and env Gene Products of the Murine Mammary Tumor Virus. (Eng) Sen, G. C. (Lab. Molecular Virology, Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Smith, S. W.; Marcus, S. L.; Sarkar, N. H. *Proc Natl Acad Sci USA* 76(4): 1736-1740; 1979.

In a reticulocyte lysate system, full-length (35S) genomic RNA from murine mammary tumor virus (MuMTV) was translated in vitro into proteins of 105,000, 75,000, 65,000, 35,000, and 27,000 daltons. These proteins were all immunoprecipitable with a monospecific antiserum to the major viral core protein, p27, but not with antiserum to the major viral envelope glycoprotein, gp47. Translation in vitro of RNA of about 24S size extracted from MuMTV yielded proteins similar in size and immunoreactivity to the products of the 25S RNA translation. Polyadenylated RNA isolated from an MuMTV-producing cell line was fractionated according to size by velocity sedimentation and subsequently hybridized to MuMTV complementary DNA probes. These studies identified at least three size classes (35S, 24S, and 14S-18S) of intracellular MuMTV-specific RNA. The 35S intracellular RNA was translated into MuMTV-specific proteins identical in size and immunoreactivity to the products of the virion-derived 35S RNA. On the other hand, translation of the intracellular 24S RNA fraction resulted in the synthesis of proteins, of which two (about 70,000 daltons) could be immunoprecipitated with anti-gp47 serum, but not with anti-p27 serum. These data suggest that MuMTV core and envelope proteins are synthesized from two different mRNA's with approximate sizes of 25S and 24S, respectively. The results also imply that the intracellular 24S messenger RNA is synthesized by a process more complex than simple cleavage of the 35S RNA. (20 refs)

79-3932 Idiopathic Mammary Tumors in BALB/c Mice. (Eng) Moore, D. H. (Dept. Microbiology and Immunology, Hahnemann Medical

Coll. and Hosp., Philadelphia, PA 19102); Sarkar, N. H.; Holben, J. A.; Sheffield, J. B. *Int J Cancer* 23(5): 713-717; 1979.

A colony of BALB/c mice consisting of two sublines with a high incidence of mammary tumors (35% and 18%) was examined for the presence of murine mammary tumor virus (MuMTV). The milk was examined at the third or later litters by microimmunodiffusion tests, and the milk and tumors were examined for the presence of B particles by electron microscopy. Neither antigen nor B particles were found. Milk and tumor extracts from the higher mammary tumor incidence line were also assayed for MuMTV activity by ip injection of these extracts into weanling C57BL, BALB/c, and RIIIf females. There was no response, except possibly in RIIIf mice. The MuMTV antigen and tumor incidences in inoculated RIIIf mice were slightly over control values. Among 53 3-mo-old mice given a single dose of x-rays (24 mice received 200 or 250 R and 29 received 450 R), only 1 developed a mammary tumor, an incidence lower than that reported for unirradiated mice. Immunization of BALB/c mice with inactivated MuMTV in Freund's complete adjuvant did not influence tumor incidence. BALB/c mice foster-nursed on C57BL mothers had an elevated mammary tumor incidence, but hybridization of these mice with C57BL males lowered their mammary tumor incidence. These results indicate that C57BL milk is mammary-tumor promoting for the BALB/c mice and that C57BL genes are not. (25 refs)

79-3933 Human Breast Carcinoma Antigen Is Immunologically Related to the Polypeptide of the Group-specific Glycoprotein of Mouse Mammary Tumor Virus. (Eng) Ohno, T. (Inst. Cancer Res., Coll. Physicians and Surgeons Columbia Univ., 701 W. 168th St., New York, NY 10032); Mesa-Tejada, R.; Keydar, I.; Ramanarayanan, M.; Bausch, J.; Spiegelman, S. *Proc Natl Acad Sci USA* 76(5): 2460-2464; 1979.

The nature of the cross-reactivity observed between gp52 [a 52,000-dalton glycoprotein of mouse mammary tumor virus (MMTV)] and a unique antigen found in human breast cancers was studied. The polysaccharide prepared from gp52 with proteinase K was unable to remove any immunohistochemical reactivity with cells from four human breast carcinomas. In contrast, the sugar-free gp52 resulting from treatment with a glycosidase enzyme mixture did absorb out those antibodies from the anti-MMTV IgG that were responsible for the reaction with the malignant cells. A radioimmunoassay titration curve also demonstrated that removal of the sugars from gp52 did not influence its ability to compete with intact gp52 for the relevant antibodies. At the very least, these results indicate that the immunological relationship between the human tumor antigen and gp52 is more than a chance correspondence of polysaccharide complexes. (23 refs)

79-3934 Humoral Antibodies to Mouse Mammary Tumor Virus in Sera from Breast Cancer Patients. (Eng) Mehta, S. P. (Tata Memorial Centre, Bombay 400 012, India); Sirsat, S. M.; Jussawalla, D. J. *Indian J Exp Biol* 16(11): 1126-1128; 1978.

The sera of 34 patients with breast cancer (22 nonoperated, 12 operated) were examined for the presence of blocking factors, eg, antibodies to mouse mammary tumor virus (MMTV). The sera of 76.4% of the patients gave positive immunofluorescence reactions with cryostat sections of C3H spontaneous mammary adenocarcinomas producing MMTV. Absorption studies demonstrated that antibodies in these sera were directed against heterophile, Forssman, and MMTV antigens. Sera from 4/10 normal controls showed the presence of antibodies against heterophile and Forssman antigens, but not against MMTV. The percentage of sera showing the presence of heterophile, Forssman, and MMTV antigens was similar in the operated and nonoperated patients. A higher percentage (82.8%) of positive immunofluorescence reactions with unabsorbed sera was observed in patients with malignant breast tumors than in those with benign tumors (50%). This pattern of distribution persisted even after absorption with heterophile and Forssman antigens. The data indicate the existence of mouse-human cross-reactivity and suggest that a virus putatively associated with human breast cancer is antigenically related to MMTV. (19 refs)

79-3935 Inhibition of Focus Formation of Rat Cells by Mouse Sarcoma Virus by Damavaricin Fc Derivatives. (Letter to Editor). (Eng) Onodera, K. (Inst. Virus Res., Kyoto Univ., Sakyo-ku, Kyoto 606, Japan); Hiragun, A.; Sato, M.; Mitsui, H.; Sasaki, K. *J Antibiot (Tokyo)* 32(5): 545-547; 1979.

The biologic activity of various derivatives of damavaricin Fc (DvFc) and damavaricin C (DvC) was studied. Focus formation in rat kidney cell cultures by the Moloney murine sarcoma-leukemia complex was inhibited by the methyl, ethyl, and n-pentyl ethers of DvFc, but not by the benzyl ether. The inhibitory activity for focus formation was related to ability to inhibit reverse transcriptase in vitro. In normal rat cells infected by mouse leukemia virus, cell growth was inhibited by the n-pentyl ether of DvFc, but not by the methyl, n-hexyl, or n-undecyl ethers or by DvFc itself. The introduction of hydrophobic groups into the C-19 position of DvFc appeared to confer the properties of penetrability and selectivity for virus-infected cells. The growth of uninfected cells was not inhibited. (12 refs)

79-3936 Expression of Xenotropic Murine Leukemia Viruses as Cell-Surface gp70 in Genetic Crosses Between Strains DBA/2 and C57BL/6. (Eng) Morse, H. C. (Lab. Microbial Immunity, Building 5,

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Room 224, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD 20014); Chused, T. M.; Hartley, J. W.; Mathieson, B. J.; Sharrow, S. O.; Taylor, B. A. *J Exp Med* 149(5): 1183-1196; 1979.

Levels of xenotropic murine leukemia virus (MuLV) envelope-related cell-surface antigens (XenCSA) in 24 BXD recombinant inbred (RI) strains derived from crosses and backcrosses between DBA/2 (high XenCSA expression) and C57BL/6 (low expression) mice were measured. It was possible to classify both thymocytes and spleen cells of 20/24 strains and one of the tissues of the remaining 4 strains as resembling those of one parent. However, strains designated as being like the progenitor strains did not form two homogeneous groups. The major locus affecting XenCSA expression appeared to be centromeric to *Gpd-I* and to segregate with *Fv-1*, both on chromosome 4. Although there were no confirmations by progeny testing, there were four dissociations between *Fv-1* and the XenCSA phenotype among the 24 BXD strains and two dissociations among 28 backcrosses between BXD and DBA/2 mice. Ecotropic MuLV was produced by 1/10 DBA/2 spleens, 8/9 F₁ spleens, and 1/9 C57BL/6 spleens tested. Seven of the 23 BXD strains studied also spontaneously produced ecotropic MuLV. There was no consistent relationship between XenCSA phenotype or alleles of *Fv-1* and production of ecotropic MuLV. In contrast, among mice resulting from backcrosses of BXD with DBA/2 mice, a relationship between high production of virus and expression of the *n* allele of *Fv-1* and high XenCSA expression was seen. (30 refs)

- 79-3937 Restriction Enzyme Analysis of Mouse Cellular Type C Viral DNA: Emergence of New Viral Sequences in Spontaneous AKR/J Lymphomas.** (Eng) Canaani, E. (Lab. Cellular and Molecular Biology, NCI, Bethesda, MD 20014); Aaronson, S. A. *Proc Natl Acad Sci USA* 76(4): 1677-1681; 1979.

The topography of endogenous C-type viral sequences in mouse cellular DNA was investigated by *EcoRI* nuclease restriction and application of the Southern blotting technique. The DNA's from one outbred and five inbred strains were resolved in 20-35 fragments containing viral sequences distributed in unique, though related, patterns for each mouse strain. Different normal tissues from the same animal were indistinguishable in their DNA patterns, suggesting that tissue differentiation is not associated with gross alteration in the topography of endogenous C-type virus sequences. Tumor tissues from spontaneous lymphomas of AKR/J mice were similarly analyzed. The emergence of one or two new virus-containing DNA fragments was detected in 4/7 individual tumors. The mass of these fragments varied, indicating different insertion sites of the new viral sequences. The detection of these new viral sequences suggests that each tumor was composed of descendants of only one or a few cells. (23 refs)

- 79-3938 Immunosuppression by Friend Leukemia Virus Is *H-2* Restricted by Alloreactive T Lymphocytes.** (Eng) Kumar, V. (Dept. Pathology, Boston Univ. Sch. Medicine, Boston, MA 02118); Bennett, M. *Proc Natl Acad Sci USA* 76(5): 2415-2419; 1979.

A three-cell protocol was used to determine the nature of the interfering mouse cells involved in the *H-2* restriction of immunosuppression by Friend leukemia virus (FV). The ability of B6 spleen cells to interfere with the suppression of B10.D2 mitogen-responsive cells by irradiated *H-2* compatible DBA/2 T suppressor cells in cultures infected with FV was eliminated by the lysis of T lymphocytes but not by the elimination of macrophages or granulocytes. The interfering cell function was shown to mature during the second week of postnatal life. Both mitomycin C and radiation eliminated the interfering cell function. Interfering cells needed only to recognize mitogen-responsive cells as *H-2* allogeneic to function; *H-2D* differences between interfering and T suppressor cells were unimportant. Induction of tolerance to *H-2* alloantigens in semiallogeneic radiation marrow chimeras resulted in the specific loss of interfering cell function. (20 refs)

- 79-3939 Effects of Toyocamycin on the Biological Activity of a Murine Oncornavirus Produced by a Chronically Infected Cell Line.** (Eng) Mauchauffe, M. (Laboratoire de Pharmacologie Experimentale, Hopital Saint-Louis, 2 place du Docteur A. Fournier, 75475 Paris Cedex 10, France); Hamelin, R.; Tavitian, A.; Michel, M. L.; Larsen, C. J. *Biomedicine [Express]* 31(1): 17-20; 1979.

The effect of Toyocamycin (TMC, 0.2 µg/ml) on virus production by a murine cell line chronically infected with the Friend virus (Friend-Eveline line) was studied. TMC blocked cell growth and reduced the virus yield by two- or threefold; the latter effect was increased with increasing TMC concentration. The biological infectivity of the TMC particles was one-fourth of the control, while virus production estimated by protein content of the purified virus still represented 69% of the control culture. The 70S RNA content of virus particles released early after drug treatment was not modified, but the infectivity of these virions was reduced by two logs. Virus from TMC-treated cells retained approx 40% of the endogenous reverse transcriptase activity of the controls, but the enzyme in the TMC-treated virions appeared to have retained its normal activity. After incubation with (³H)TMC, the radioactivity recovered in the viral particles was incorporated in the 70S viral RNA. (12 refs)

- 79-3940 Friend Erythroleukemia Antigen: A Viral Antigen Specified by Spleen Focus-forming Virus and Differentiation Antigen Controlled by the *Fv-2* Locus.**

(Eng) Risser, R. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI); 53706 *J Exp Med* 149(5): 1152-1167; 1979.

Serologic techniques were used in an attempt to detect a viral antigen specified by spleen focus-forming virus (SFFV). Serum from C57BL/6 (B6) mice hyperimmunized with NB-tropic Friend virus (FV) was cytotoxic for FV-induced erythroleukemic (EL) spleen cells and B6 Friend-murine leukemia virus (F-MuLV) lymphoma cells. Cytotoxic activity for EL cells remained after repeated absorption of B6 anti-FV antiserum with Friend-Moloney-Rauscher MuLV lymphoma cells, but it was removed by absorption with EL cells induced by FV or Rauscher virus. The previously unrecognized cell-surface antigen of mouse leukemia detected in this study was designated Friend EL (FE) antigen. FE antigen was not detected on 15 hematopoietic neoplasms nor on cells infected with ecotropic, xenotropic, or duotropic MuLV isolates in tissue culture. Two SFFV nonproducer cells of rats and one of mice expressed FE antigen in amounts comparable to those expressed by primary EL cells. FE antigen was expressed on bone marrow and spleen but not thymus, lymph node, or peripheral blood of uninfected AKR, BALB/c, DBA, and SWR mice; all five tissues from B6 and C57BL mice were negative. Quantitatively, FE antigen expression was greatest on fetal liver, less on bone marrow, and lowest on spleen from BALB and SWR mice, although all three were much less than that on EL cells. Expression of FE antigen cosegregated with inheritance of the *Fv-2S* allele. These results indicate that the FE antigenic system identifies a cell-surface determinant that has the properties of an SFFV-specified antigen and a hematopoietic differentiation alloantigen controlled by the *Fv-2S* locus. (56 refs)

79-3941 Evolution of Primary Tumors Induced in Adult Mice by MSV-H and MSV-H Producer Tumor Cells. (Eng) Branca, M. (Laboratorio di Malattie Batteriche e Virali, Istituto Superiore di Sanita, Viale Regina Elena 299, 00161 Rome, Italy); Nicoletti, L. *Tumori* 65(1): 1-8; 1979.

The oncogenic effect of the Harvey strain of murine sarcoma virus (MSV-H) and MSV-H-producer tumor cells (BALB/c 3T3 cells transformed by MSV-H, designated 3T3+MSV-H) was investigated in adult female BALB/c mice. Groups of 10 mice each were injected with 3T3+MSV-H cells ($10-10^6$ cells/1 ml medium) sc or with 1 ml of freshly filtered 3T3+MSV-H tissue culture fluid either sc or ip. Animals showing a swelling were autopsied; the rest were maintained for 6 mo. No tumors were detected in mice inoculated with 10 cells. Inoculation with higher numbers of cells resulted in numerous tumors in a high percentage of animals after a 12- to 14-day latent period for the early tumors and a 30- to 60-day period for the late ones (very few cases). The latent period was inversely proportional to the number of cells injected. Tumor incidence and

size were greatest with the lower cell inoculations (10^2 , 10^3). When mice were inoculated with undiluted MSV-H, tumors developed in half the treated mice, with the lowest incidence occurring in mice inoculated ip. The latent period was longer than that for mice inoculated with tumor cells. The tumors usually appeared at the injection site, and none regressed. The growth pattern was suggestive of a multicentric origin of tumors induced by MSV-H, which reacted with all the susceptible mesenchymal cells located at various sites. No simple dose-response correlation was observed with tumor cell inoculation, suggesting the existence of a balance between immunization and tumor growth. (13 refs)

79-3942 Germ Line Integration of Moloney Leukemia Virus: Identification of the Chromosomal Integration Site. (Genetic Transmission/Structural Gene/Segregation Analysis/Somatic Cell Hybrids/Backcross Animals). (Eng) Breindl, M. (Heinrich Pette-Institut für Experimentelle Virologie und Immunologie, Martinistrasse 52, 2000 Hamburg 20, W. Germany); Doehmer, J.; Willecke, K.; Dausman, J.; Jaenisch, R. *Proc Natl Acad Sci USA* 76(4): 1938-1942; 1979.

The chromosomal integration site of the structural gene of Moloney murine leukemia virus (M-MuLV) in the genome of BALB/Mo mice was mapped genetically. These mice transmit exogenous M-MuLV as an endogenous virus at a single Mendelian locus. In one series of experiments, non-virus-producing fibroblasts from homozygous BALB/Mo embryos were fused to Chinese hamster Wg3-h-o cells. The segregation of the viral genome in 30 independent mouse-Chinese hamster cell hybrid clones was measured by molecular hybridization and enzymes assigned to 16 different mouse chromosomes were compared. There was a highly concordant segregation of M-MuLV sequences and the mouse enzyme triosephosphate isomerase, whose gene has been assigned to chromosome 6. A further karyotype analysis of 9 clones, in which the chromosomes were identified cytochemically, supported this result. In a second experimental approach, the segregation of the viral genome was studied in backcrosses of BALB/Mo with ABP/J mice. A linkage of the M-MuLV genome to the morphological marker *wa-1* on mouse chromosome 6 was found in the backcross ABP/J x (ABP/J x BALB/Mo). This confirmed the conclusion that the M-MuLV genome is integrated in mouse chromosome 6. The genetic locus denoting the genetically transmitted structural gene of M-MuLV in BALB/Mo mice was designated *Mov-1*. (43 refs)

79-3943 Detection of Sequences in Human Leukemic Cell DNA Homologous with Moloney Mouse Leukemia Viral RNA. (Eng) Van Beveren, C. (Dept. Medicine, Sch. Medicine, Univ. California, San Diego, La Jolla, CA 92093); Gouliau, M. *Cancer Res* 39(7, part 1): 2532-2537; 1979.

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DNA complementary to Moloney murine leukemia virus (M-MuLV) RNA was annealed with DNA isolated from the peripheral leukocytes of 12 patients with leukemia and from spleen cells from two patients without malignant disease. The hydroxyapatite procedure used was able to detect one MuLV sequence per 10-40 cells. Leukocyte DNA from the normal patients and 11 leukemic patients lacked significant homology with cDNA prepared from MuLV. However, the DNA from one patient with acute myelocytic leukemia exhibited a limited degree (6%-10%) of homology with the M-MuLV cDNA. The significance of these results, especially with regard to a causal role for RNA tumor viruses in human leukemia, is unclear. (44 refs)

- 79-3944 Fusion of XC Cells by Murine Type-C Viruses: Cinematographic Observations.** (Eng) Ntake, K. (Dept. Microbiology, Aichi Medical Univ., Yazako, Nagakute-cho, Aichi-ken 480-11, Japan); Kobayashi, S.; Yoshikawa, K.; Ito, Y. *Gann* 70(2): 249-254; 1979.

Cinematographic studies of the cellular fusion of XC cells induced by two purified C-type virus preparations, one from a murine myeloma cell line and the other from a Rauscher leukemia virus (R-MuLV)-producing cell line, are described. A cell-free culture fluid and/or serially diluted purified virus preparation was mixed with trypsinized XC cells and transferred to vessels for cinematographic observation. Cellular fusion occurred within 5 hr after incubation of the cell-virus mixture. During the following 48-hr period, the number of nuclei in the fused cell and the cytoplasmic volume in the syncytia per se increased progressively. The process began by the fusion of two mononucleated cells. Additional cells continued to introduce their cytoplasm and nucleus to give rise to multinucleated giant cells. The simultaneous fusion of several multikaryons was frequently observed during the late incubation period. The final process was the termination of additional fusion, and it was followed by degeneration in most of the syncytia. None of the polykaryons underwent mitosis or cell division. These findings suggest that multikaryon formation of cells by murine C-type viruses is due to mutual cell fusion and not to endomitosis. (13 refs)

- 79-3945 Changes in the Fibroblastoid Colony Forming Unit Population from Mouse Bone Marrow in Early Stages of Soule Virus Induced Murine Leukemia.** (Eng) Zipori, D. (Dept. Cell Biology, Weizmann Inst. Science, Rehovot, Israel); van Bekkum, D. W. *Exp Hematol* 7(3): 137-144; 1979.

The incidence of bone marrow derived fibroblastoid cells was studied during the early stages of spontaneous and Soule virus-induced leukemogenesis in six strains of mice.

An age-dependent decline in the incidence of fibroblastoid colony-forming units recoverable from the bone marrow was demonstrated. The decline was steeper in the case of AKR mice (which show a high incidence of spontaneous leukemia) and BALB/c mice inoculated with Soule virus. Fibroblastoid colonies from the bone marrow of AKR and virus-inoculated BALB/c mice produced ecotropic virus, C-type particles having been observed budding from fibroblast-like cells obtained from such cultures. Fibroblastoid and granulocyte macrophage colonies from AKR and Soule virus-inoculated mice did not exhibit significant XC syncytia forming activity. The changes observed in the fibroblastoid colony-forming unit population during the first weeks following virus inoculation did not appear to be attributable to the activity of tumor cells invading the bone cavity. It is possible that the reduced incidence of hematopoietic stem cell forming colonies occurring at later stages of the disease was related to the activity of tumor cells rather than to infection with leukemia virus. (13 refs)

- 79-3946 Sera and Cerebrospinal Fluids from Normal Uninfected Sheep Contain a Visna Virus Inhibiting Factor.** (Eng) Thormar, H. (New York State Inst. Basic Res. in Mental Retardation, 1050 Forest Hill Road, Staten Island, NY 10314); Wisniewski, H. M.; Lin, F. H. *Nature* 279(5710): 245-246; 1979.

The presence of a visna virus (VV) inhibitory factor (VIF) in the sera and cerebrospinal fluid (CSF) of normal, uninfected sheep was studied. Sheep cell monolayers inoculated with VV were incubated with 90% lamb serum or CSF. Both the serum and CSF contained a visna VIF that, depending on the size of the inoculum, prevented or greatly limited VV infection. This factor had no effect on the proliferation of herpes simplex virus type I or vaccinia virus. VIF did not inhibit the production of virus particles, but it did directly inactivate the virus. The VIF did not appear to be an immunoglobulin with specific antibody against VV or a serologically related agent. Rather, it appeared to be a lipoprotein. Preliminary data suggests that VIF inhibits viral spread in inoculated sheep. It is hypothesized that the VIF is of importance in restraining persistent VV infection in sheep, at least in its early stages before the appearance of neutralizing antibodies. (24 refs)

- 79-3947 Adenovirus-induced Genetic Mutations in Mammalian Cells.** (Rus) Lukash, L. L. (Inst. Molecular Biology and Genetics, Kiev, USSR); Buzhievskaya, T. I.; Varshaver, N. B.; Shapiro, N. I. *Dokl Akad Nauk SSSR* 245(4): 970-973; 1979.

An attempt was made to determine whether oncogenic DNA-containing viruses other than simian virus 40, exhibit mutagenicity. Chinese hamster cells (line 237-8 Glu- ts;

modal chromosome number 18) were incubated with bovine adenovirus type 3 (BAV-3). The mutagenic effect was measured by the number of 6-mercaptopurine-resistant mutants. At a multiplicity of infection of 8 or 80 infectious units per cell, the frequency of resistant mutants was significantly greater than that in control cultures, but at 0.8 infectious unit per cell, the mutant frequency was similar to that in controls. (10 refs)

- 79-3948 UV-enhanced Virus Reactivation in Mammalian Cells: Effects of Metabolic Inhibitors.** (Eng) Lytle, C. D. (Bureau of Radiological Health, Food and Drug Admin., HEW, Rockville, MD 20857); Goddard, J. D. *Photochem Photobiol* 29(5): 959-962; 1979.

The induction process of UV-enhanced reactivation of UV-irradiated herpes simplex virus type 1 was investigated in CV-1 monkey kidney cells. The protein synthesis inhibitor cycloheximide (0.5-5 μ g/ml), when present in the culture medium for 24 hr between cell irradiation and virus infection, decreased the enhanced virus survival normally found in UV-irradiated cultures. The enhanced virus reactivation became essentially resistant to the addition of cycloheximide by 6-8 hr after cell irradiation, indicating that the cycloheximide-sensitive process necessary for enhanced reactivation was complete by that time. Since cycloheximide inhibits DNA as well as protein synthesis, the effect of a DNA synthesis inhibitor, hydroxyurea, was investigated. Hydroxyurea did not decrease UV-enhanced virus survival, but resulted in enhanced virus survival even in unirradiated cells. Therefore, the cycloheximide-caused inhibition of UV-enhanced reactivation did not arise from inhibition of DNA synthesis. The combined results indicated that (1) UV-enhanced virus reactivation in monkey kidney cells requires de novo protein synthesis during the first 6-8 hr after cell irradiation and that (2) DNA synthesis inhibition may be the initiating event. (19 refs)

- 79-3949 Interferon-sensitive Expression of Membrane-bound IgM on a Human Lymphoid B Cell Line Persistently Infected with Herpes Simplex Virus.** (Eng) Menezes, J. (Lab. Immunovirology, Dept. Microbiology and Immunology, Univ. Montreal, Montreal H3T 1C5, Canada); Bourkas, A. E. *Biomedicine [Express]* 31(1): 2-4; 1979.

The expression of Fc receptors and other surface immunologic markers by a B cell type lymphoid cell line persistently infected with herpes simplex virus (HSV) was compared with that of uninfected Raji cells and their primarily HSV-infected counterparts. Approx 50% of primarily and persistently HSV-infected cells expressed Fc receptors. However, unlike the primarily HSV-infected cells, about 80% of the persistently-infected cells were positive for surface IgM. No other classes of immunoglobulin were

detected. IgM induction could be suppressed by long-term treatment with human leukocyte interferon but not with phosphonoacetic acid (an inhibitor of herpesvirus DNA polymerase activity). Removal of interferon did not restore IgM expression within 90 days. When Raji-A44 cells were superinfected 15 days after the removal of interferon, about 40% expressed Fc receptors. (7 refs)

- 79-3950 Possible Pitfalls in the Study of IgG Receptors Produced by Herpesvirus-infected Cells. Brief Report.** (Eng) Forghani, B. (Viral and Rickettsial Disease Lab., State California Dept. Health, 2151 Berkeley Way, Berkeley, CA 94704); Schmidt, N. J.; Lennette, E. H. *Arch Virol* 60(2): 167-169; 1979.

A purified human IgG preparation was found to contain fairly high levels of antibodies to human viruses, including cytomegalovirus, herpes simplex virus type 1 (HSV-1), HSV-2, varicella-zoster, measles, rubella, mumps, adenovirus, and vaccinia. IgG binding to HSV-infected cells could be reduced to an insignificant level by preliminary absorption with concentrated and purified HSV-1 and HSV-2. Commercial IgG absorbed with purified HSV showed binding to infected cells similar to that of antibody-negative serum, indicating that most binding of unabsorbed material was due to specific HSV antibodies. (10 refs)

- 79-3951 Enhancement of Host Cell Reactivation of Ultraviolet-irradiated Herpes Simplex Virus by Caffeine, Hydroxyurea and 5-Bromodeoxyuridine.** (Eng) Fogel, M. (Dept. Genetics, Weizmann Inst. Science, Rehovoth, Israel); Yamanishi, K.; Rapp, F. *Int J Cancer* 23(5): 657-662; 1979.

The effects of caffeine, hydroxyurea, and 5-bromodeoxyuridine (BrdUrd) on the host cell reactivation (HCR) of UV-irradiated herpes simplex virus (HSV) was investigated. Primary rabbit kidney (RK) cell cultures and human embryo lung (HEL) cultures were treated with 10 mM caffeine for 24 hr and infected with virus immediately after drug removal or after 24 or 48 hr. In cultures infected with UV-irradiated HSV, enhancement of plaque formation was affected by the time of caffeine pretreatment and the UV dose used to irradiate the virus. In the RK cells, plaque formation was max when cultures were infected with HSV irradiated for 6 min and when caffeine treatment was terminated 24 hr before infection. UV-irradiated HSV exhibited a similar survival profile in HEL and RK cells, but the rate of virus inactivation was eight- to ninefold higher when it was assayed in HEL cells. Caffeine enhanced the HCR of irradiated virus about fivefold. In RK cultures treated with caffeine (2 mM) continuously after infection, the survival of the irradiated virus was reduced ninefold, but in cultures treated with drug 24 or 48 hr before infec-

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tion, virus survival was increased to a similar degree. In RK cells pretreated with hydroxyurea, the infectivity of virus irradiated for 6 min increased five times, but in BrdUrd-pretreated cells, infectivity increased three times. The results suggest that enhancement of the HCR of UV-irradiated HSV by DNA antimetabolites is primarily associated with DNA repair activated as a result of cell DNA damage. (16 refs)

- 79-3952 Long-Term T-Cell-mediated Immunity to Epstein-Barr Virus in Man. III. Activation of Cytotoxic T Cells in Virus-infected Leukocyte Cultures.** (Eng) Moss, D. J. (Queensland Inst. Medical Res., Bramston Terrace, Brisbane, Queensland 4006, Australia); Rickinson, A. B.; Pope, J. H. *Int J Cancer* 23(5): 618-625; 1979.

T-cell numbers were monitored in Epstein-Barr virus (EBV)-infected WBC cultures during a 4-wk period, and the cytotoxicity of T cells harvested from such cultures during the activation phase was assayed. Kinetic studies suggest that, in virus-infected cultures from seropositive (SP) donors, a population of T cells proliferates within the first 2 wk in response to the appearance of virus-infected B cells. This proliferation continues to some extent during the regression period. The addition of phytohemagglutinin to cultures 24 hr after infection did not affect the subsequent virus-induced transformation of seronegative (SN) donor cultures and protected the SP donor cultures from regression. T cells harvested from SP donor cultures 11-14 days after infection suppressed the outgrowth of 10- to 100-fold more autologous EBV-transformed target cells than did freshly prepared T cells or T cells from control cultures. T cells harvested from virus-infected cultures from SN donors showed little inhibitory effect upon the outgrowth of the autologous lymphoblastoid cell line. The results support the view that regression is mediated by cytotoxic T cells reactivated in vitro as part of a secondary immune response. This response, initiated from a pool of EBV-specific memory cells found in the blood of SP but not SN donors, can be divided into proliferative and cytotoxic components. (29 refs)

- 79-3953 Evidence for Antigenic Distinctness of the Epstein-Barr Virus-determined Nuclear Antigen and the Herpes-virus Papio-determined Nuclear Antigen.** (Eng) Ohno, S. (Dept. Tumor Biology, Karolinska Inst., S 104 01, Stockholm 60, Sweden); Luka, J.; Klein, G. *Cancer Lett* 6(6): 325-329; 1979.

Previous serological evidence suggested that Epstein-Barr virus-determined nuclear antigen (EBNA) has cross reactive components with the herpesvirus papio-determined nuclear antigen (HUPNA), in addition to distinct components of its own, not present in HUPNA. This

hypothesis was tested in absorption experiments by exposing human anti-EBNA-positive sera (EG) to EBNA or HUPNA and testing for residual activity against both antigens. Absorption of EG serum with EBNA abolished both anti-EBNA and anti-HUPNA reactivity. Absorption with HUPNA removed all anti-HUPNA reactivity but did not reduce anti-EBNA to any detectable degree. These results confirm the serological evidence and show that the difference in reactivity is due to the presence of EBNA-specific antigenic components, in addition to the cross-reactive components of EBNA and HUPNA. (11 refs)

- 79-3954 Long-Term T-Cell-mediated Immunity to Epstein-Barr Virus in Man. II. Components Necessary for Regression in Virus-infected Leukocyte Cultures.** (Eng) Rickinson, A. B. (Queensland Inst. Medical Res., Bramston Terrace, Brisbane, Queensland 4006, Australia); Moss, D. J.; Pope, J. H. *Int J Cancer* 23(5): 610-617; 1979.

The role played by T cells in the regression of Epstein-Barr virus (EBV)-induced transformation and additional components of the in vitro system that might be necessary to mediate the effect were investigated. In unfractionated mononuclear (UM) WBC cultures seeded at $\geq 10^6$ cells/ml, regression occurred irrespective of virus dose in cell cultures from seropositive (SP) donors but not in any of the cultures from seronegative (SN) donors. The effect with the SP donors was regularly reproduced in cultures reconstituted with equal numbers of T-cell-depleted (TD) and T-cell-enriched (T) populations. The SP donors whose UM cells regressed consistently only at initial cell concentrations $>10^6$ cells/ml required correspondingly higher numbers of T cells to display regression in the reconstituted cultures. The more densely seeded cultures of TD cells from SP donors occasionally (7/60 cultures) were transformed without the addition of EBV. Removal of residual viral envelope material with papain did not affect the successful transformation of cells in reconstituted cultures from SN donors nor the regression of SP donor cultures. The presence of monocytes was not required for regression. Regression was independent of cytotoxic mechanisms involving antiviral antibodies and was not mediated by soluble factors released into the culture medium. These results are consistent with the view that regression occurs through the direct recognition of EBV-infected B cells by a pool of memory T cells present in the circulation of individuals previously infected by the virus. (25 refs)

- 79-3955 Common Structural Features in DNA Around the Replication Origin of Papova Virus, Mouse Polyoma Virus, Simian Virus 40 and Human BKV.** (Eng) Soeda, E. (Nat. Inst. Genetics, Mishima 411, Japan); Miura, K. I. *FEBS Lett* 101(2): 359-363; 1979.

The nucleotide sequence of the mouse polyoma virus DNA fragment (*Hap11-3*) adjacent to the previously reported *Hap11-5* fragment was studied. The results were compared with those from studies of simian virus 40 (SV40) and human BKV DNA's. Remarkable similarities among these nucleotide sequences were found, the three DNA's containing several identical nucleotides at corresponding positions. An 8-T cluster terminated with A and a sequence AGAGGCCG were common to all three viral DNA's. The G-C rich regions containing AGAGGCCG had a dyad symmetry and were located near the origins of DNA replication. This may reveal an evolutionary relationship between the viruses. When the sequence around the DNA replication origin of polyoma virus was compared with that of rat mitochondria, the two DNA's showed similar features: there were A (or T) clusters on both sides of a G-C rich hairpin structure. These features were also common with the proposed sequence around the origin of replication of SV40 and BKV DNA's. (25 refs)

- 79-3956 Replication of Polyoma DNA Isolated Nuclei. VII. Initiator RNA Synthesis During Nucleotide Depletion. (Eng) Eliasson, R. (Medical Nobel Inst., Biochemistry Dept. I., Karolinska Inst., S-104 01 Stockholm, Sweden); Reichard, P. *J Mol Biol* 129(3): 393-409; 1979.

The effects on initiator RNA (iRNA) synthesis of limiting the concentrations of deoxyribonucleoside triphosphates (dNTP's) and ribonucleoside triphosphates (rNTP's) during incubation of nuclei were studied. As demonstrated by Ultrogel AcA34 chromatography, the length of iRNA decreased when the concentration of dNTP's became limiting. The chromatograms contained two peaks: one peak corresponded to a chain length for 30 nucleotides, suggesting a restriction of chain elongation to this size, and the other corresponded to a chain length of 50-100 nucleotides at limiting dNTP concentrations and 150-200 nucleotides in control incubations. Limiting conditions of dNTP also decreased the initiation of new strands of iRNA. In experiments in which nuclei were incubated at concentrations of dATP or deoxycytosine triphosphate that limited DNA synthesis to a varying degree, there was a decrease in iRNA synthesis concomitant with the inhibition of total DNA synthesis. When rNTP's other than ATP were deleted from the incubation medium, only a small decrease of DNA and iRNA synthesis occurred. Under those conditions, deoxyribonucleotides substituted for ribonucleotides and were incorporated internally into the iRNA. Thus, the iRNA-synthesizing enzyme can use either rNTP's or dNTP's. It is suggested that the switch from primer synthesis to DNA synthesis under conditions of rNTP limitation occurs after the primer has reached a length of 10 nucleotides, irrespective of whether the primer contains rNTP's or dNTP's. It is also suggested that a mammalian counterpart to primase, the *dnaG* gene product of *Escherichia coli*, catalyzes the synthesis of iRNA. (40 refs)

- 79-3957 Serological Detection of a Polyoma-Tumor-associated Membrane Antigen. (Eng) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S 104 01 Stockholm, Sweden); Ehlin, B.; Witz, I. *Int J Cancer* 23(5): 683-690; 1979.

The antigenic specificities of three polyoma virus-induced tumors, SEYF-a, SESO, and SEWE, of strain ABY, A, and ASW mice, respectively, were examined. Syngeneic antisera raised against the three tumors contained antibodies directed against multiple antigenic specificities. One antigen was cross-reactive for the three polyoma tumors, but it was absent from a large variety of other tumors tested (lymphomas, carcinomas, and sarcomas). Other antigens (common) were shared by a variety of other tumors of viral or nonviral origin. Five different common antigens were demonstrated. The SEWE tumor contained fewer common antigens than the more often transplanted SEYF-a and SESO tumors. This suggests that newly arising tumors may not express as many common antigens as often-transplanted tumors and/or they may express them in a lower concentration. It is likely that serial passage increases the chances for the expression of common antigens, possibly due to the acquisition of passenger viruses or as a result of secondary changes in the cytogenetics of the passaged tumor. (23 refs)

- 79-3958 Morphokinetic Aspects of Cell Transformation and Cancerization In Vitro. (Eng) Barski, G. (Laboratoire de Culture de Tissus et de Virologie, Institut Gustave-Roussy, 16 bis, Avenue Paul-Vaillant Couturier, 94800 Villejuif, France). *Natl Cancer Inst Monogr* (48): 263-267; 1978.

The morphokinetic aspects of cell transformation (tfn) and malignant tfn in vitro are summarized. In many instances, in vitro cell tfn, evidenced by adaptation to permanent growth in vitro, modification of cell morphology, and irreversible breakdown of the euploid karyotype, is not always accompanied by malignant tfn, evidenced by the ability to produce tumors in vivo. Even in mouse cells, in which in vitro tfn and chromosome variability are particularly precocious and intense, tumorigenicity may appear many months after generalized tfn of cultivated cells and, in some instances, may not occur at all. Some cells, such as human fibroblasts, do not acquire tumorigenicity in long-term cultures unless infected and transformed by DNA oncogenic viruses. On maintenance in long-term cell culture, female Akodon *Urichi venezuelensis* (a South American rodent) heart and kidney cells, which have an initial euploid karyotype of 18 chromosomes, show a progressive accumulation of hyperdiploid mitoses with wide dispersion of values and apparently random involvement of different chromosome pairs. These changes precede the development of tumorigenicity; even when the cells are subsequently exposed to simian virus 40, they may remain nontumorigenic. When neoplastic tfn occurs, it frequently

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involves a stepwise evolution, usually from low- to high-tumorigenic populations. A decrease in the intrinsic tumorigenicity of cells in vitro has also been observed with Chinese hamster and mouse cell lines. Highly tumorigenic cells generally show greater in vitro motility (cytoplasmic streaming) and loss of adherence to a substrate than those with low tumorigenicity. However, the capacity of these cells to penetrate and move into and through areas occupied by normal cells depends in part on the nature of the latter. (13 refs)

- 79-3959 A Simple Large-Scale Method for Separating Closed Circular Form DNA by Gel Electrophoresis.** (Eng) Tanabe, N. (Dept. Biochemistry, Cancer Inst., Okayama Univ. Medical Sch., Okayama 700, Japan); Hidaka, H.; Watanabe, S.; Oda, T. *Acta Med Okayama* 32(6): 379-385; 1978.

A simple method for isolating closed circular DNA by agarose gel electrophoresis is described. The covalently closed form of circular duplex simian virus 40 (SV40) DNA was separated from the open linear form by electrophoresis in a large-scale gel system. The gel containing the closed circular DNA was cut into small slices and placed in a glass pipette; a dialysis bag was slipped over the pipette and the DNA was electrophoresed out of the slices using electrophoresis buffer. DNA recovery was about 70%. Ultrastructural examination demonstrated that the recovered DNA had no double- or single-stranded breaks. This DNA can be used without further purification for ultrastructural studies, as a substrate for experiments using restriction endonuclease, and as a template for in vitro RNA synthesis. (15 refs)

- 79-3960 Two-dimensional Electrophoresis of Membrane Proteins from Normal and Transformed Cells.** (Eng) Litin, B. S. (Dept. Biochemistry, Coll. Medicine, Univ. Arizona, Tucson, AZ 85724); Grimes, W. J. *Cancer Res* 39(7): 2595-2603; 1979.

Two-dimensional electrophoresis was used to compare cell surface proteins and glycoproteins from normal and transformed BALB/c mouse cells. The tumor lines included spontaneous and viral transformants that caused tumors that regressed or grew progressively. The normal cell line (A31) and the nonimmunogenic progressor (3T12T) appeared very similar by (³⁵S)methionine labeling. Membrane proteins from murine sarcoma virus and simian virus 40 transformants (MSC and SVT₂) showed increased heterogeneity of the bands in the isoelectric focusing direction, compared with A31. The patterns of the two viral transformants were quite similar to each other. Many of the differences between A31 and the viral transformants were also seen between A31 and immunogenic spontaneous transformants (c5T and c5). Compared with the profiles of

the transformants, the (³⁵S)methionine gel pattern of A31 showed two changes related to missing spots; these changes could not be correlated with tumorigenicity. Glycoproteins were very heterogeneous in the isoelectric focusing dimension. This heterogeneity was not due entirely to variations in sialic acid content, although sialic acid had some effect on shifts in isoelectric points. A shift in isoelectric point was the only change that correlated with tumorigenicity. (29 refs)

- 79-3961 Dual Control of Cell Growth by Somatomedins and Platelet-derived Growth Factor.** (Eng) Stiles, C. D. (Lab. Tumor Biology, Group W, Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA 02115); Capone, G. T.; Scher, C. D.; Antoniades, H. N.; Van Wyk, J. J.; Pledger, W. J. *Proc Natl Acad Sci USA* 76(3): 1279-1283; 1979.

The role of somatomedins and various growth factors in cell-growth control was studied in BALB/c 3T3 mouse cells. Quiescent 3T3 cells were exposed briefly to human platelet-derived growth factor (PDGF) and then incubated with plasma and thymidine. Plasma from hypophysectomized rats had 1/20 of the potency of normal rat plasma for promoting DNA synthesis in PDGF-treated cells, indicating that progression through G₀/G₁ is under pituitary control. Pretreatment with PDGF was required for plasma to promote DNA synthesis, indicating that PDGF-treated cells become competent to replicate their DNA. Addition of somatomedin C (S-C) to hypophysectomized-rat plasma increased the fraction of the PDGF-treated cells that synthesized DNA, showing that S-C is required for progression through G₀/G₁. Various growth factors were tested for progression activity and competence activity by using BALB/c 3T3 tissue culture assays. The only agents that displayed potent progression activity were somatomedin A, S-C and the S-related factors multiplication-stimulating activity, insulinlike activity, and Buffalo rat liver cell-conditioned medium. Fibroblast growth factor and calcium phosphate precipitates were potent inducers of competence. Growth factors with potent progression activity had little or no competence activity and vice versa. Simian virus 40 (SV40), however, displayed both competence and progression activity. Thus, SV40 overrides the growth requirement for two functionally distinct sets of hormones. (44 refs)

- 79-3962 Tumorigenicity of Cells Transformed by Simian Virus 40 and of Hybrids Between Such Cells and Normal Diploid Cells.** (Eng) Gee, C. J. (Sir William Dunn Sch. Pathology, South Parks Road, Oxford OX1 3RE, England); Harris, H. *J Cell Sci* 36: 223-240; 1979.

The tumorigenicity of cells transformed by simian virus 40 (SV40) and of hybrids between these and normal cells was

studied. All of the SV40-transformed cell lines showed the transformed phenotype, and all expressed SV40 tumor (T) antigen in >95% of their nuclei. None of the cell lines produced progressively growing tumors following sc inoculation into irradiated genetically compatible newborn mice, although transient swellings were sometimes noted at the inoculation site. After treatment with ethyl methanesulfonate and a period of subcultivation, two transformed cell lines gave rise to progressively growing invasive fibrosarcomas in immunosuppressed hosts. Hybrids between tumor cells derived from SV40-transformed cells and normal diploid embryo cells showed the transformed phenotype in vitro, contained marker chromosomes from both parents, and expressed SV40 T antigen in 100% of their nuclei. The incidences of tumor take following sc inoculation of these hybrids into compatible hosts ranged from 0% to 28%, compared with 100% for the parental tumor lines. Thus, the malignant phenotype of the SV40-transformed cell was suppressed by the normal diploid cell. Morphologically transformed hybrids between PG19 melanoma cells and a nontumorigenic SV40-transformed cell line were nontumorigenic in syngeneic neonates; both lines expressed SV40 T antigen in 99%-100% of their nuclei. (43 refs)

- 79-3963 **Differentiation Between SV40 Large-T and U Antigenic Sites.** (Eng) Robb, J. A. (Dept. Pathology, Green Hosp. Scripps Clinic, La Jolla, CA 92037). *J Gen Virol* 42(2): 405-408; 1979.

Radioimmune precipitation, sodium dodecyl sulfate-polyacrylamide slab gel electrophoresis, and fluorography were used to investigate the simian virus 40 (SV40) large-T (tumor) and U antigenic sites on species of proteins synthesized during wild-type and tsA58 mutant infections in TC7 monkey cells. Advantage was taken of the instability of the mutant 94,000-mol wt large-T molecule produced during temperature-sensitive group A mutant infection in monkey cells at the restrictive temperature. Wild-type infection at 33 and 41.5 C and the A58 infection at 33 C produced similar profiles of three protein species ranging in mol wt from 84,000 to 94,000, all of which had both the large-T and U antigenic sites. At 41.5 C, however, the A58 infection produced an additional four discrete species ranging in mol wt from 60,000 to 74,000 that contained the large-T site(s), but not the U site(s). A subpopulation of the 74,000-mol wt species contained both sites. Therefore, the region of the A58 mutant 94,000-mol wt species containing the U antigenic site(s), presumably the COOH-terminal region, appears to be more sensitive to processing, probably proteolytic cleavage, than does the region containing the large-T antigenic site(s). (25 refs)

- 79-3964 **Augmentation of Specific Tumor Immunity Against a Syngeneic SV40-induced Sarcoma in**

Mice by Retinoic Acid. (Eng) Glaser, M. (Lab. Cell Biology, NCI, NIH, Bethesda, MD 20014); Lotan, R. *Cell Immunol* 45(1): 175-181; 1979.

The effect of retinoic acid (RA) on the specific tumor immunity against a syngeneic simian virus 40 (SV40)-induced sarcoma was studied using female BALB/c mice and the SV40-transformed BALB/c kidney cell line mKSA. In mice given 30 or 100 µg RA ip/day, protection against mKSA growth was observed at a spleen:tumor cell ratio of 25:1, whereas a ratio of 100:1 was required to produce a similar effect in the control group. Higher doses of RA had no enhancing effect, and no augmentation of the immune response was produced by RA in unimmunized mice. The augmentation of cell-mediated immunity caused by RA was specific for the syngeneic tumor cells. The immune response in the RA-treated mice was detected earlier and lasted longer than that in the control group. The data strongly suggest that T cells mediated the increase in the immune reactivity of the spleen cells of RA-treated animals. It is not known whether RA directly affects the same or different subpopulations of T cells than those activated by the tumor antigens, or whether RA first affects the macrophages which then interact directly or through a soluble factor to activate the effector T cells. (21 refs)

- 79-3965 **Augmentation of Specific Immune Response Against Syngeneic SV40-induced Tumor-associated Antigens by Splenectomy.** (Eng) Glaser, M. (Dept. Biochemistry, George Washington Univ. Medical Center, 2300 Eye St., N.W., Washington, DC 20037). *Cell Immunol* 45(1): 230-236; 1979.

The control mechanisms responsible for the regulation of cellular processes involved in the immune response against syngeneic simian virus 40 (SV40)-induced tumors were studied using SV40-transformed C57BL/6 (C57SV) cells. Splenectomy before immunization of mice with the syngeneic C57SV cells augmented the specific immune response against the corresponding tumor-associated antigens as measured by the in vitro release of ⁵¹Cr and the in vivo tumor-cell neutralization assay. The time of splenectomy before immunization was not critical. Thymus-derived lymphocytes in normal spleens suppressed the in vivo cell-mediated immune response against SV40-induced tumor-associated antigens. The resident population which normally operates in intact mice to suppress the specific immune response against SV40-induced tumor-associated antigens appeared to be T cell. (25 refs)

- 79-3966 **Changes in the Population of Lymphocytes and Their Response to Mitogens During the Growth of a Simian Virus 40-induced Fibrosarcoma in Hamsters.** (Eng) Chen, H. (Laboratoire d'Immunochimie, Institut de Recherches Scientifiques sur le Cancer, Chris-

tiane de Vaux St. Cyr, B. P. 8, F-94800, Villejuif, France); Quan, C. P.; Zuinghedau, J.; de Vaux Saint Cyr, C.; Lespinats, G. *Eur J Immunol* 9(4): 80-84; 1979.

A study was made of changes in the number of splenic and thymic lymphocytes and their response to mitogens that occurred during the growth of simian virus 40-induced fibrosarcomas in Syrian golden hamsters. Small fragments of dissected tumors from syngeneic animals were grafted onto the left flank of 2- to 3-mo-old hamsters. Splenomegaly was observed in tumor-bearing animals, and the number of splenic lymphocytes increased by 100% during tumor growth. Splenic lymphocytes showed a normal or slightly increased response to concanavalin A (Con A) when the tumors were small, but this response gradually decreased to 26%-42% of that of normal lymphocytes in animals bearing large tumors (14.6-27 g). When these cells were cultured with normal lymphocytes in the presence of Con A, they either had no effect on or slightly suppressed the normal cellular response to Con A. The response to lipopolysaccharide (LPS) decreased slightly. The thymus became smaller during tumor growth. However, the response of thymic lymphocytes to Con A was similar to that of splenic lymphocytes. Increased numbers of macrophages were observed in the spleens of tumor-bearing animals, and it is suggested that they may play a role in suppression of the lymphocyte response to mitogens. Elimination of macrophages from the cultures by the addition of carrageenan enhanced the lymphocyte response to Con A in tumor-bearing animals. Carrageenan acted as a mitogen, most probably for B cells, and when it was added to cultures containing LPS, it potentiated the mitogenic effect of LPS. The results indicate that significant changes occur in the immune system of hamsters with simian virus 40-induced fibrosarcomas. The changes that occurred immediately following the graft and in small tumors suggest an attempt by the host to mount a cellular immune response; later, the spleen cells became less and less responsive to mitogens, and the immune system appeared to become disorganized. (16 refs)

79-3967 Lipid Metabolism in Cultured Cells. XVIII. Comparative Uptake of Low Density and High Density Lipoproteins by Normal, Hypercholesterolemic and Tumor Virus-transformed Human Fibroblasts. (Eng) Wu, J. D. (Dept. Biochemistry, George Washington Univ. Sch. Medicine, Washington, DC 20037); Butler, J.; Bailey, J. M. *J Lipid Res* 20(4): 472-480; 1979.

The specific binding of high-density (HDL) and low-density (LDL) lipoproteins in normal and abnormal human fibroblasts (FB) was compared, along with the mechanism of uptake of the two lipoproteins in the various FB. The lipoproteins were doubly labeled with [^3H]cholesterol and [^{125}I] in the apoprotein moiety. In the binding assay for LDL, the absence of specific LDL receptors in type II

hypercholesterolemic FB was confirmed, whereas monolayers of simian virus 40-transformed human lung FB (VA-4) exhibited LDL binding characteristics similar to those of normal lung FB. In studies of HDL binding, specific HDL binding sites were demonstrated in normal and virus-transformed FB. In addition, type II hypercholesterolemic cells, despite the loss of LDL receptors, retained normal HDL binding sites. There was no significant competition between the two lipoprotein classes for their respective binding sites over a fivefold concentration range. In VA-4 cells, the amount of lipoprotein required to saturate half the receptor sites was $3.5 \mu\text{g/ml}$ ($9 \times 10^{-9} \text{ M}$) for LDL and $9.1 \mu\text{g/ml}$ ($9 \times 10^{-8} \text{ M}$) for HDL. Pronase treatment reduced LDL binding by more than half, but it had no effect on HDL binding. Chloroquine, a lysosomal enzyme inhibitor, stimulated net LDL uptake 3.5-fold by increasing internalized LDL, but it had essentially no effect on HDL uptake. Further experiments were conducted using doubly labeled lipoproteins to characterize the interaction of LDL and HDL with cells. The cholesterol and protein moieties of LDL were incorporated into cells at similar rates, but the uptake of the cholesterol moiety of HDL was 5-10 times more rapid than that of the protein component. Furthermore, the apoprotein component of LDL was extensively degraded following exposure, whereas the apoprotein moiety of HDL retained its macromolecular chromatographic characteristics. These results indicate that HDL and LDL bind to cultured cells at separate sites and that further processing of the two lipoprotein classes appears to take place by fundamentally different mechanisms. (26 refs)

79-3968 Adenovirus-associated Virus Polypeptides Synthesized in Cells Coinfected with Either Adenovirus or Herpesvirus. (Eng) Salo, R. J. (Dept. Biological Sciences, Univ. Texas at El Paso, El Paso, TX 79968); Mayor, H. D. *Virology* 93(1): 237-245; 1979.

Adenovirus-associated virus (AAV) protein synthesis was studied in HEp-2 cells (human epidermoid carcinoma of the larynx) coinfecting with adenovirus (Ad: a complete helper virus) or herpes simplex virus type 1 (HSV: a partial helper virus). Three AAV structural polypeptides (VP1, VP2, and VP3) were synthesized in AAV-infected cells coinfecting with Ad or HSV. The molar proportions of these polypeptides were the same as those found in the virion. In cells coinfecting with HSV, AAV protein synthesis began at 7-8 hr after infection, but in Ad-coinfecting cells, AAV protein synthesis started 9-10 hr after infection. The AAV polypeptides were neither phosphorylated nor glycosylated. Cell cultures coinfecting with Ad or HSV and AAV were pulse-labeled at various times, and the cytoplasmic and nuclear fractions were examined by electrophoresis to determine the site of AAV protein synthesis. The results suggest that AAV protein synthesis occurs in the cytoplasm and that the polypeptides are transported rapidly to the nucleus. Therefore, the failure of assembly

into complete virions to occur in cells coinfecting with HSV does not appear to be the result of a missing structural polypeptide or the inhibition of transport of a polypeptide. This failure may be expressed at the level of DNA strand segregation or encapsidation of DNA, or in faulty capsid assembly. (36 refs)

- 79-3969 Nucleotide Sequence at the Inverted Terminal Repetition of Adenovirus Type 2 DNA.** (Eng) Shinagawa, M. (Dept. Biological Chemistry, Univ. Maryland Sch. Medicine, Baltimore, MD 21201); Padmanabhan, R. *Biochem Biophys Res Commun* 87(3): 671-678; 1979.

The nucleotide sequence of the inverted repetition present at the termini of adenovirus type 2 (Ad2) DNA was determined. Sequence analysis demonstrated that Ad2 contains an inverted terminal repetition of 103 base pairs at each 3' end that is identical in length and sequence to that present in Ad5 DNA. Further experiments demonstrated the incorporation of dpG residues at the 3' termini, and it is possible that dpG residues are added to the 3' termini complementary to the 5' protruding dC residues by the polymerizing activity of DNA polymerase I. (29 refs)

- 79-3970 Studies of the Antigenic Composition of Adenovirus Hexones.** (Ger) Dohner, L. (Institut für Medizinische Mikrobiologie und Epidemiologie, Ernst-Moritz-Arndt-Universität, Martin-Luther-Strasse 6, DDR-22 Greifswald, E. Germany); Dieckmann, U. *Acta Biol Med Ger* 37(11/12): 1735-1740; 1978.

Quantitative relationships between the group-specific and type-specific components of the hexones of adenovirus types 2 and 5 (Ad2 and Ad5) were studied with the use of fluorescein isothiocyanate-conjugated antigen-binding fragments (Fab) of antibodies directed against Ad2 and Ad5 hexones. The hexones were recrystallized three times and were found to be homogeneous both immunologically and in the polyacrylamide test. The sedimentation constants of the hexones and Fab in the region of excess Fab indicated that there are at least 20 determinants on the hexone. Half were found to be type-specific, half group-specific. Both components of the Ad2 hexone consisted of equal parts of carbodiimide-sensitive and carbodiimide-resistant determinants. The carbodiimide-sensitive component was able to bind 7-10 Fabs, and the carbodiimide-resistant component was able to bind 3-4 Fabs. (16 refs)

- 79-3971 Hepatitis A Infection and Primary Hepatocellular Carcinoma.** (Eng) Drucker, J. A. (Institut Virologie, Facultes de Medecine et de Phar-

macie, 2 bis, Bd Tonnele, 37000 Tours, France); Cour-saget, P.; Maupas, P.; Goudeau, A.; Gerety, R. J.; Chiron, J. P.; Denis, F.; Mar, I. D. *Biomedicine [Express]* 31(1): 23-25; 1979.

The prevalence of antibody to the hepatitis A antigen (anti-HAV) was studied among 64 Senegalese patients with primary hepatocellular carcinoma (PHC) and 50 healthy blood donors. The two groups did not differ significantly in the prevalence of anti-HAV. However, 47 patients showed evidence of chronic infection with hepatitis B virus (HBV) as compared with only 15 controls ($p < 0.001$). Among PHC patients, anti-HAV was not correlated with the presence or absence of markers of active HBV. Anti-HAV was significantly more common among individuals aged <30 yr (81%) than among those aged >30 yr (45%). Anti-HAV also varied significantly with ethnic background: 2/21 Toucouleur and Serer subjects were anti-HAV positive as compared with 47/67 Peuhls and Woloffs of similar age ($p < 0.001$). HBV markers did not vary significantly with ethnic background and anti-HAV did not vary significantly with sex. The results suggest that infection with HAV was not related to the development of PHC. (13 refs)

- 79-3972 Cloning in *Escherichia coli* and Physical Structure of Hepatitis B Virion DNA.** (Eng) Char-nay, P. (Recombinaison et expression genetique, Institut National de la Sante Recherche Medicale U 163, Unite de Genie Genetique, Institut Pasteur, 28 rue du Docteur Roux, 75015 Paris, France); Pourcel, C.; Louise, A.; Fritsch, A.; Tiollais, P. *Proc Natl Acad Sci USA* 76(5): 2222-2226; 1979.

A restriction map of hepatitis B virion DNA was established after the entire viral genome was cloned in *Escherichia coli*. With the use of *EcoRI*, *XhoI*, *BglII*, *XbaI*, *BamHI*, *HincII*, and *HaeIII* endonucleases, a total of 28 restriction sites were mapped. The sum of the fragment sizes from each digest was 3,100 base pairs. The single-stranded region was localized on the restriction map and the 5' end of the short strand was mapped at a fixed position 1,580 base pairs from the *EcoRI* site, 1,700 base pairs from the *XhoI* site, and 1,310 base pairs from the *BamHI* A site. (16 refs)

- 79-3973 Serum Iron Levels and Response to Hepatitis B Virus.** (Eng) Felton, C. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111); Lustbader, E. D.; Merten, C.; Blumberg, B. S. *Proc Natl Acad Sci USA* 76(5): 2438-2441; 1979.

The hypothesis that the serum iron level is higher in chronic renal dialysis patients who have hepatitis B surface antigen (HBsAg) in their blood than those who have antibody to

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HBsAg (anti-HBs) or those who lack both HBsAg and anti-HBs (no evidence of infection) was tested. Response to hepatitis B virus (HBV) infection, serum Fe, total Fe-binding capacity, hematological status (RBC, Hb, and hematocrit), and evidence of liver damage (serum glutamic pyruvic transaminase; aspartate aminotransferase, L-aspartate:2-oxoglutarate aminotransferase) were determined for 201 patients on chronic renal dialysis. Four factors, serum Fe level, transaminase level, sex, and HBV response [HBsAg(+), anti-HBs(+), or no response], were analyzed simultaneously. Four independent, statistically significant, two-factor interactions were identified: (1) serum Fe is higher in HBsAg(+) patients; (2) serum Fe is higher in those with increased transaminase; (3) transaminase is higher in HBsAg(+) patients; and (4) men are more likely to be HBsAg(+) and women are more likely to be anti-HBs(+). Also, patients who are HBsAg(+) have a significantly higher percent Fe saturation (serum Fe/total Fe-binding capacity). Thus, the hypothesis was supported by the findings. Several additional biological hypotheses are suggested, including a possible role of increased Fe levels in susceptibility and response to HBV infection and the possible relationship between higher Fe levels and the likelihood of HBV infection progressing to primary hepatocellular carcinoma. (15 refs)

- 79-3974 **Alterations in Cell Characteristics in Relation to Malignant Transformation.** (Eng) Bachvaroff, R. J. (Dept. Surgery, State Univ. New York at Stony Brook, Stony Brook, NY 11794); Klein, G.; Rapaport, F. T. *Transplant Proc* 11(1): 1055-1059; 1979.

Proteins synthesized and released by mitogen-stimulated normal human T and B lymphocytes, by several Burkitt's lymphoma cell lines, by Epstein-Barr virus (EBV)-transformed normal human B lymphocytes, and by a cell line of infectious mononucleosis origin were detected by two-dimensional electrophoresis followed by fluorography of the second-dimension gels. The protein patterns of stimulated T and B cells on the third day of culture were almost identical. Approx 200 proteins were visualized, the most prominent ones being nonmuscle isoactins and tubulin. The presence of 5-bromodeoxyuridine (BrdU) did not affect the protein patterns. The 6-7 day protein maps of stimulated T cells were not significantly different from the 3-day maps, but the stimulated B lymphocytes had begun to synthesize large numbers of μ and γ chains. Burkitt's lymphoma cell lines produced approx 300 different proteins, which differed almost completely from those of normal stimulated B lymphocytes. The maps of proteins synthesized by the different Burkitt's lymphoma cell lines and by normal B lymphocytes immortalized in vitro with EBV were very similar. BrdU had no effect upon the number of proteins produced by the lymphoma cells, but it completely blocked the differentiation of normal B cells toward immunoglobulin production in response to mitogen stimula-

tion. The protein pattern of the infectious mononucleosis cell line was completely different from those of the other EBV-positive cells. These results provide clear evidence of the significant changes in gene expression produced by viral transformation. (24 refs)

- 79-3975 **Retrovirus-like Particles in EBV-negative Burkitt's Lymphoma Cell Line but not in EBV-DNA-Positive Lines from Patients with Ataxia Telangiectasia and Down's Syndrome.** (Eng) Kotler, M. (Lab. Molecular Virology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Balabanova, H.; Friedmann, A.; Becker, Y. *Br J Cancer* 39(4): 414-421; 1979.

The release of retroviruslike particles by Epstein-Barr virus (EBV)-negative and EBV-infected BJAB-1 cells (derived from a patient with Burkitt's lymphoma) and by lymphoblastoid cell lines derived from nonleukemic patients with ataxia telangiectasia (AT), Down's syndrome (DS), or infectious mononucleosis (IM) was studied. Viruslike particles released from EBV-negative BJAB-1 cells incubated in arginine-deficient medium contained reverse transcriptase activity and resembled the virus particles released from the P3HR-1, Raji, and 1301 lymphoblast cell lines. Similar particles were released from EBV-infected BJAB-1 cells and from a cell line resulting from the fusion of BJAB-1 cells with EBV DNA-positive Raji lymphoblasts. None of these cell lines released viruslike particles when grown in medium containing arginine. No retroviruslike particles were released by the EBV DNA- and EBV nuclear antigen-positive cell lines derived from AT, DS, and IM, whether grown in complete or arginine-deficient medium. (19 refs)

- 79-3976 **Different Responses to Drugs and Serum of Cells Transformed by Various Means.** (Eng) Dubrow, R. (Interdisciplinary Programs in Health, Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA 02115); Riddle, V. G.; Pardee, A. B. *Cancer Res* 39(7): 2718-2726; 1979.

Ten transformed cell lines (DNA and RNA tumor virus, chemically and spontaneously transformed) and their untransformed counterparts were examined for their growth responses with low serum and with 16 drugs. The response of seven BALB/3T3 transformed lines (two DNA virus, three RNA virus, and two chemical) to growth in low serum over a 3-day period was examined by flow microfluorimetry and autoradiography. Cells transformed by different means responded to low serum differently. The DNA virus-transformed lines were distributed around the cell cycle while the RNA virus and chemically transformed lines accumulated in G₁. The increase in cell number over a 2-day period was determined in the presence

of several concentrations of a drug. The drugs tested were rotenone, antimycin A, azide, oligomycin, arsenate, chloroquine, ouabain, tetracaine, actinomycin D, lucanthone, 6-azauridine, 3-acetylpyridine, picolinic acid, caffeine, methylglyoxal bis(guanyldrazone), and streptovitamin A. The most notable finding was the lack of consistency of drug response among transformed lines. For no drug were all transformed lines either more or less sensitive than were the corresponding untransformed lines. A particular mode of transformation did not exhibit a drug response which was unique to that mode of transformation. Thus, to provide evidence of a change universal for all tumor cells from studies of cells transformed by one agent appears impossible. The search for the most general traits of tumor-forming cells should be carried out with a variety of cells that are most closely related to cells of naturally occurring neoplasms. (69 refs)

- 79-3977 Mechanism of Rejection of Virus Persistently Infected Tumor Cells by Athymic Nude Mice.** (Eng) Minato, N. (Dept. Microbiology and Immunology, Albert Einstein Coll. Medicine, Bronx, NY 10461); Bloom, B. R.; Jones, C.; Holland, J.; Reid, L. M. *J Exp Med* 149(5): 1117-1133; 1979.

Studies were conducted to develop an appropriate model system for defining the mechanism of discrimination between different types of tumors and the possible mechanisms available for tumor rejection in the nude mouse. Cell lines known to be highly tumorigenic in the nude mouse [baby hamster kidney 21 (BHK21) and HeLa] were modified by rendering them persistently infected (PI) with a variety of RNA viruses. Although 10-100 HeLa and BHK21 cells produced tumors within 3 wk in 100% of nude mice, as many as 2×10^7 of the same cells PI with viruses failed to produce tumors. The finding of marked mononuclear cell infiltrates at the inoculation sites and the inability of irradiated nude mice to reject the PI cell lines suggest active thymus-independent mechanisms of rejection by the nude mouse. Studies of the in vitro cytotoxicity of spleen cells from normal nude mice indicated that PI cell lines, but not uninfected cell lines, were susceptible to spontaneous cytotoxicity, that in vivo inoculation of PI lines induced an enhanced cytotoxicity for a PI target in vitro, and that the effector cells had the characteristics of natural killer (NK) cells. Cold target competition experiments suggest specific recognition by NK cells. A variant subline of BHK cells PI with vesicular stomatitis virus, which could withstand the rejection process in nude mice, formed metastatic and invasive tumors. These cells were the most potent inducers of NK cell activity against various PI targets in vivo and the most resistant of the PI lines to NK cell cytotoxicity in vitro. There was a good correlation in this system between tumor rejection in vivo and susceptibility to NK cells in vitro. These studies suggest that

NK cells may play a significant role in rejection of tumor cells and in resistance to viruses. (38 refs)

- 79-3978 Virus Carrier State Suppresses Tumorigenicity of Tumor Cells in Athymic (Nude) Mice.** (Eng) Reid, L. M. (Dept. Molecular Pharmacology, Albert Einstein Coll. Medicine, 1300 Morris Park Ave., Bronx, NY 10461); Jones, C. L.; Holland, J. *J Gen Virol* 42(3): 609-614; 1979.

Nude mice injected sc with normal, uninfected BHK 21 cells or HeLa cells regularly develop large, rapidly growing tumors at the inoculation site. However, these same tumor cell lines when persistently infected with vesicular stomatitis virus (VSV) or other enveloped RNA viruses are either rejected or form small nodules in nude mice. This rejection phenomenon probably involves some type of immunocyte, since heavily irradiated nude mice (500 rads) cannot reject persistently infected cells but develop large, rapidly growing tumors that shed virus and defective interfering virus (DI) and do not exhibit the lymphocytic infiltration observed in the nodules of unirradiated mice given persistently infected cells. A subline of BHK 21-VSV carrier cells that regularly produces large, rapidly growing tumors in normal unirradiated nude mice was finally selected, although all these carrier cells express virus antigen and shed large amounts of mature infectious virus and DI both in vivo and in vitro. (17 refs)

- 79-3979 Ultrasonic Absorption Evidence of Structural Fluctuations in Viral Capsids.** (Eng) Cerf, R. (Laboratoire d'Acoustique Moléculaire, Centre National de la Recherche Scientifique, Université Louis Pasteur, 4, rue Blaise Pascal, 67000 Strasbourg, France); Michels, B.; Schulz, J. A.; Witz, J.; Pfeiffer, P.; Hirth, L. *Proc Natl Acad Sci USA* 76(4): 1780-1782; 1979.

Dissociated protein/capsid assembly equilibrium was investigated ultrasonically in two small icosahedral viruses, brome mosaic virus (BMV) and tomato bushy stunt virus (TBSV). When BMV protein polymerized from the dimer into empty capsids, the absorption of ultrasound at several 0.6, 0.9, 1.4, and 2.6 megahertz increased greatly. Ultrasonic absorption by BMV virions nearly equaled the sum of the contributions of the protein shell and of the RNA. It was concluded that there can be no large contribution to absorption resulting from RNA-protein interactions. The amount by which the absorption of the particles exceeded the absorption of the protein dimer was reduced to less than one-half upon swelling of the virion. Since protein subunits become less tightly packed when the virus

swells, these results suggest that the excess absorption is due to protein-protein interactions. This theory is supported by the finding of a reduction of excess absorption when TBSV virions were crosslinked with glutaraldehyde, which increased the rigidity of the shell. It was concluded that the excess ultrasonic absorption over the sum contributed by the components alone was specific to the self-assembled protein shell and represented spontaneous motions within an assembled system. Such motions could consist of cooperative structural changes involving either a few protein molecules or the whole shell. These spontaneous motions may play a role in liberating RNA and may be of

functional significance at an early stage of viral infection.
(8 refs)

See also:

*(Rev.): 79-3627, 79-3628, 79-3629, 79-3630, 79-3631,
79-3632, 79-3633, 79-3634, 79-3635, 79-3636,
79-3637, 79-3638, 79-3639, 79-3640.

*(Chem.): 79-3722.

*(Immun.): 79-3985, 79-3991, 79-3992, 79-4003, 79-4006,
79-4020, 79-4021, 79-4022, 79-4023.

*(Path.): 79-4047, 79-4063, 79-4066, 79-4067, 79-4073.

*(Epid.-Biom.): 79-4138, 79-4152.

- 79-3980 Radiorocket Electrophoresis Autography (RREA) Through Labelled Antigen for AFP Assay and Its Applications in Sero-epidemiologic Investigation of Primary Hepatocellular Carcinoma (PHC). (Eng) Tsung-tang, S. (Cancer Inst., Chinese Acad. Medical Sciences, China); Lai-chi, W.; Yu-lan, C.; Cheng-hung, L.; Chiu-chieh, H.; Feng-ming, L. *Chinese Med J* 92(1): 17-25; 1979.

A radiorocket electrophoresis autography (RREA) technique in which radiolabeled antigen is incorporated specifically into the immune precipitate was developed for measuring serum α -fetoprotein (AFP) concentrations. The technique was applied to a Chinese population with a high incidence of primary hepatocellular carcinoma (PHC). In 200 normal subjects, AFP levels were <25 nanograms (ng)/ml. In 30 patients with histologically proved PHC, 83% had AFP levels >400 ng/ml. An AFP positivity rate of 85% was observed for another PHC group. In a field survey of 10,000 people, 10 had AFP levels of 100-1,000 ng/ml. Four of these asymptomatic patients showed a steady increase of AFP over a 2-mo period. The AFP doubling time varied from 25 to 60 days. Two other apparently healthy persons had AFP values >600 ng/ml. These six patients were operated on when the AFP concentrations were 500-1,000 ng/ml. In three patients, localized, histologically proved PHC was found and resected. The AFP values fell rapidly and the patients are alive and well 4 yr after surgery. The other three patients died of PHC within 12 mo: in two no tumor could be found at surgery and in the third a localized but nonresectable tumor was detected. All three asymptomatic patients with early PHC had liver hyperplasia, and they were positive for serum hepatitis B surface antigen. The remaining four subjects with high AFP levels showed a fluctuating AFP pattern during follow-up; chronic hepatitis was diagnosed. If these results are confirmed in subsequent studies, it may be possible to develop an effective screening procedure for PHC following regular AFP surveys using RREA or other sensitive techniques. (19 refs)

- 79-3981 Fibrin Gel Investment Associated with Line 1 and Line 10 Solid Tumor Growth, Angiogenesis, and Fibroplasia in Guinea Pigs. Role of Cellular Immunity, Myofibroblasts, Microvascular Damage, and Infarction in Line 1 Tumor Regression. (Eng) Dvorak, H. F. (Immunopathology Unit, Dept. Pathology, Massachusetts General Hosp., Boston, MA 02114); Dvorak, A. M.; Manseau, E. J.; Wiberg, L.; Churchill, W. H. *J Natl Cancer Inst* 62(6): 1459-1472; 1979.

The morphologic events associated with the growth of line 1 and line 10 hepatocarcinoma in the sc spaces of nonim-

munized syngeneic Sewall Wright strain 2 guinea pigs were studied. Three distinct but overlapping phases characterized the growth of the line 1 tumors. During the first phase (days 1-3), the injection sites appeared as largely translucent gelatinous papules enveloping centrally placed, irregularly shaped clumps of tumor cells. The bulk of the mass was composed of a meshwork of fibrin strands oriented in three dimensions and interspersed in edema fluid. Microvascular proliferation accompanied the development of the fibrin gel. During the second phase (days 4-7), the tumor mass developed an increasingly rubbery consistency as it approached max size on days 6-8. The fibrin gel was replaced with granulation tissue, and dilated, tortuous small blood vessels at the tumor periphery became enveloped by extensive microhemorrhages. Phase 3 (days 8-13) was characterized by developing cellular immunity and tumor destruction. The tumor was replaced by a scar and there was widespread damage to the endothelium of the small vessels supplying it. There was little inflammatory reaction. The highly malignant line 10 carcinoma grew progressively while maintaining a relatively constant histologic pattern. Tumor papules were composed of rapidly dividing, pleomorphic tumor cells, including giant cells and glandular elements with minimal enveloping or intermingling stroma. There was very little inflammatory response, no vessel necrosis, and no tumor regression. The pattern of developing granulation tissue seen after injections of fibrinogen and thrombin was similar to that observed around line 1 tumors. (45 refs)

- 79-3982 Resting and Concanavalin-A Stimulated Levels of Cyclic Nucleotides in Splenic Cells of Aging Mice with Spontaneous Cancers. (Eng) Tam, C. F. (Dept. Pathology, Univ. California Sch. Medicine, Los Angeles, CA 90024); Smith, G. S.; Walford, R. L. *Life Sci* 24(4): 311-322; 1979.

Measurements were made of the resting levels of cyclic AMP (cAMP) and cyclic 3',5'-guanosine monophosphate (cGMP) in splenic lymphoid cells of 25 aged (C57BL/10 x C3H)F₁ hybrid mice with spontaneous tumors (5 with hepatoma, 10 with lung tumor, 2 with lymphoma, and 8 with several tumor varieties), 18 young mice, and 13 tumor-free aging mice. The alterations in spleen cell cyclic nucleotide levels that are characteristic of normal aging in tumor-free animals may be additionally influenced by the occurrence of spontaneous neoplasia. Furthermore, the levels may vary with different types of late-life tumors. For example, cAMP levels in the resting spleen cells of old mice with hepatomas and their age-matched controls were similar, whereas spleen cells from old mice with lung tumors showed exceedingly high levels of resting cAMP. Upon in vitro stimulation by concanavalin A (Con A), the

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splenic lymphoid cells from mice bearing spontaneous late-life lung and liver tumors displayed different kinetic patterns of percent changes in cAMP, cGMP, and cAMP/cGMP ratios, compared with young or age-matched tumor-free controls. Thus, both resting and Con-A-stimulated levels of cAMP, cGMP, and their ratios in splenic lymphoid cells may be affected by spontaneous cancer elsewhere in the body, including cancer of nonlymphoid type and origin. These findings plus the known functional decline in immune response capacity and the increase in spontaneous tumor incidence with age may suggest the existence of a complex relationship among cyclic nucleotide levels, immunity, aging, and cancer. (38 refs)

- 79-3983 Hybrid Resistance to BALB/c Plasmacytomas. I. Resistance to MPC-11 Controlled by a Locus Not Linked to H-2. (Eng) Walker, M. C. (Dept. Pathology, New York Univ. Medical Center, New York, NY 10016); Phillips-Quagliata, J. M. *J Immunol* 122(4): 1535-1543; 1979.

Genetic control of hybrid resistance to the BALB/c plasmacytoma MPC-11, an IgG2bx-secreting tumor, was investigated. The results indicate that F₁ hybrid resistance to this tumor is controlled by a single dominant autosomal gene or gene complex that segregates independently of H-2 and the coat color c and b loci. This gene has the same strain distribution pattern in the CXB Bailey recombinant inbred strains as three unlinked genes, H-2, Ly-4, and Ea-4. It is possible, therefore, that it could be linked to either of the latter two loci. Strains that carry a positive allele for resistance are C57BL/10 and all of its congenic resistant partners tested, C57BL/6, C57L, C57BL/Ks, AKR, and DBA/1. BALB/c and its congenic resistant partners are presumed to carry a negative allele of the gene for resistance to MPC-11. Strains such as SJL, DBA/2, and A and their congenic resistant partners, which form susceptible hybrids with BALB/c, could carry either the negative allele of the gene for resistance, like BALB/c, or a positive allele of the gene and some other gene conferring susceptibility on the hybrids. Heterozygosity within the H-2 complex increases resistance only in the presence of this non-H-2-linked gene for resistance, and the effect maps to the left of the H-2D region. (71 refs)

- 79-3984 Hybrid Resistance to BALB/c Plasmacytomas. II. Radiation Sensitivity and Silica Insensitivity of Resistance to MPC-11. (Eng) Walker, M. C. (Dept. Pathology, New York Univ. Medical Center, New York, NY 10016); Phillips-Quagliata, J. M. *J Immunol* 122(4): 1544-1547; 1979.

The effect of sublethal total-body irradiation or iv silica administration on the resistance of F₁ hybrids between BALB/c and four C57BL/10 congenic resistant strains to

the BALB/c plasmacytoma MPC-11 was investigated. Silica administration (3-4 mg) had no significant effect on the resistance of any of the hybrids to MPC-11, as determined from tumor incidence, median latency, median survival times, and numbers of survivors. However, 450 rads of total-body irradiation markedly reduced or abrogated the resistance of the hybrids to MPC-11. These results suggest that the mechanism of hybrid resistance to MPC-11 may depend on an active immune response that could be humoral and/or cellular and is different from Hh-1-controlled hybrid resistance. (34 refs)

- 79-3985 Reproducibility and Relation to Specific and Non-Specific Anti-Tumor Resistance of the Tumor 'Sneaking Through' Phenomenon. (Eng) Deichman, G. I. (Cancer Res. Center, Acad. Medical Sciences, Moscow, USSR); Kluchareva, T. E.; Kashkina, L. M.; Matveeva, V. A. *Int J Cancer* 23(4): 571-584; 1979.

The tumor 'sneaking-through' phenomenon, one of the possible ways in which single tumor cells can escape host control in syngeneic and allogeneic hosts, and enhancement of tumor growth were studied in syngeneic and random-bred Syrian hamsters. Effective immunization with large doses of irradiated tumor cells seemed to interfere with the sneaking-through phenomenon after primary challenge, but nonimmunizing doses did not regularly induce this effect. After secondary challenge, a striking enhancement of tumor growth and the appearance of sneaking through were observed in some immune animals that had resisted the primary challenge. Large doses of heat-inactivated tumor cells were highly efficient inducers of the sneaking-through phenomenon. Tumor growth was associated with sneaking through in the same animal, the period from 7 to 45 days after pretreatment being associated with an increased susceptibility to tumor cell transplantation. Normal hamster embryo (HE) cells did not induce the sneaking-through phenomenon, and spontaneously transformed HE cells were only somewhat effective. The induction of sneaking through was immunologically nonspecific. Normal animals were naturally resistant to the transplantation of 1 to 1×10^3 or more tumor cells, this resistance being totally abrogated by pretreatment with tumor cell preparations. The data are considered to demonstrate two antitumor defense systems; ie, nonspecific natural resistance and specific anti-tumor immunity. (43 refs)

- 79-3986 Mechanisms of "Cytostasis" of Tumours In Vitro by Syngeneic Lymphoid Cells of Tumour Bearers. (Eng) Farram, E. (Dept. Immunology, London Hosp. Medical Coll., Turner St., London E1, 2AD, England); Festenstein, H. *Cell Immunol* 44(2): 352-366; 1979.

The cytostasis assay, an in vivo-in vitro radioactive techni-

que for detecting antitumor responses in syngeneic tumor-bearing hosts, was studied. Cytostasis of the 3-methylcholanthrene-induced BALB/c sarcoma Meth A was shown to be a non-T-cell effector mechanism. Both macrophages and B cells appeared to be the effector cells, and antibody was required for cytostasis. Antibody-dependent cell-mediated cytotoxicity was ruled out as a likely mechanism for cytostasis, but Meth A cytostasis was able to occur in the presence of antibody and complement. Antibody-coated Meth A cells caused regression of some tumors when inoculated into BALB/c mice. When these regressor mice were rechallenged with tumor, they were found to be permanently immune to the tumor. (20 refs)

- 79-3987 Neutrophil Migration-inhibition Activity Produced by a Unique T Lymphoblast Cell Line.** (Eng) Weisbart, R. H. (Dept. Medicine, Veterans Admin. Hosp., Sepulveda, CA 91343); Billing, R.; Golde, D. W. *J Lab Clin Med* 93(4): 622-626; 1979.

Fifteen lymphoblast cell lines, including B-cell, T-cell, Null cell, and myeloblast cell types, were examined for the production of leukocyte migration-inhibition factor (LIF)-like activity. Only one T-lymphoblast line (Mo), established from a patient with hairy cell leukemia, produced LIF both spontaneously and in response to mitogen stimulation. As little as 4×10^4 Mo cells spontaneously produced detectable LIF-like activity by 24 hr. When Mo cells were cultured in the presence of concanavalin A, LIF-like activity became detectable by 3 hr (28% inhibition) and was max at 8 hr (45% inhibition). None of the other cell lines tested, including four other T-lymphoblast lines from patients with acute lymphoblastic leukemia, produced detectable LIF-like activity. Mo T cells also produced LIF-like activity in response to phytohemagglutinin. The LIF-like activity did not inhibit neutrophil migration as a result of chemotactic activity, and it was nondialyzable, stable at 56 C for 30 min, and resistant to diisopropylfluorophosphate treatment. The availability of the Mo T-lymphoblast line should allow the opportunity to isolate, purify, characterize, and compare this selective T-cell product with other known lymphocyte mediators. (20 refs)

- 79-3988 Impairment of Cell-mediated Immunity Functions by Dietary Zinc Deficiency in Mice.** (Eng) Fernandes, G. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Nair, M.; Onoe, K.; Tanaka, T.; Floyd, R.; Good, R. A. *Proc Natl Acad Sci USA* 76(1): 457-461; 1979.

The influence of zinc deficiency on cell-mediated immunity functions was studied in A/Jax, C57BL/Ks, and CBA/H mice. Several immunologic features were analyzed in mice on a Zn-deficient diet (Zn-), in mice pair-fed a diet containing 50 ppm Zn (Zn+), in mice fed a Zn+ diet ad lib, and in

mice fed laboratory chow ad lib. When placed on a Zn-diet, the 6- to 8-wk-old mice showed loss of body wt, low lymphoid tissue wt, and profound involution of the thymus within 4-8 wk after initiation of the regimen. Approx 50% of the mice on the Zn- diet developed severe acrodermatitis enteropathica (lesions on tail and paws) and diarrhea. Paired mice on the Zn+ diet did not show any of these symptoms. Mice on the Zn- diet showed the following immune deficiencies: (1) depressed plaque-forming cells against sheep RBC after in vivo immunization; (2) depressed T killer cell activity against EL-4 tumor cells after in vivo immunization; and (3) low natural killer cell activity. However, antibody-dependent cell-mediated cytotoxicity against chicken RBC was normal in the mice on the Zn-diet. Deficiency of T killer cell activity was not observed when immunization with EL-4 allogeneic lymphoma cells was carried out in vitro. A progressive loss of relative and absolute number of Thy 1.2+ cells and a proportionate relative increase in cells bearing Fc receptors were seen in spleen and lymph nodes of Zn- animals. Apparently, Zn is essential for maintaining normal T-cell and other immune functions in vivo. (41 refs)

- 79-3989 The Influence of Dietary Protein Concentration and Energy Intake on Mitogen Response and Tumor Growth in Melanoma-bearing Mice.** (Eng) Erickson, K. L. (Dept. Human Anatomy, Sch. Medicine, Univ. California, Davis, CA 95616); Gershwin, M. E.; Canolty, N. L.; Eckels, D. D. *J Nutr* 109(2): 353-359; 1979.

The effects of energy deprivation and low or high dietary protein levels upon lymphocyte transformation of spleen cells from syngeneic tumor-bearing and control mice were studied in C57BL/6J mice. Tumors were produced in 7-wk-old mice by sc injection of 5×10^6 P51 cells, a line derived from a transplantable B16 murine malignant melanoma. Animals were given diets that included 15% or 45% casein or a stock diet. In each of the three diet groups, experimental and control mice were provided daily, for 2 wk, with about 2, 3.3, or 4.6 g of feed, which represented 50%, 80%, or 100% of the ad libitum intake. Mitogen responses were evaluated after 19 days of tumor growth (14 days of dietary manipulation). The presence of a growing melanoma significantly stimulated both T- and B-lymphocyte transformation. T-cell responses were dependent on dietary protein concentrations, but not on the level of energy intake. The response elicited by the B-cell mitogen lipopolysaccharide was influenced by the level of energy intake but not by the amount of protein in the diet. The lowest level of lymphocyte transformation occurred with mice fed the stock diet. There was no significant correlation between changes in tumor wt and responses to mitogens. It is suggested that lymphocyte transformation may be depressed by relatively low phenylalanine or tyrosine levels in the diet when protein intake is limited by a low dietary concentration and/or restricted intake of a diet containing adequate protein. (29 refs)

79-3990 Studies on T and B Lymphocytes in Rats Bearing Methylcholanthrene-induced Tumor.

(Eng) Klobusicka, M. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia); Kalafut, F.; Novotna, L. *Neoplasma* 25(6): 667-677; 1978.

The influence of primary 3-methylcholanthrene (3-MC)-induced tumors on T and B lymphocyte levels in the spleen, thymus, lymph nodes, and peripheral blood was studied in 3- to 4-wk-old inbred Berlin Druckrey VIII (BD) and Lewis (LW) rats and their F₁ hybrids. Tumors started to appear 2 mo after the injection of MC (2 mg sc). The number of T and B cells in single lymphoid organs was influenced by tumor size. In rats bearing tumors weighing 2 g, T- and B-cell counts were increased in the spleen and lymph nodes, and the T-cell count was decreased in the peripheral blood. The number of T and B lymphocytes was reduced markedly in all lymphoid organs of animals bearing tumors weighing 20-80 g. However, there was still a higher percentage of T lymphocytes in the spleens of these rats than in the control spleens. T- and B-cell counts in the thymus apparently were not influenced by the presence of tumors. There was a high percentage of blasts, particularly in animals with tumors weighing 1-2 g. WBC counts increased with tumor size. The presence of tumor was also marked by reduced eosinophil counts. In the spleen and lymph nodes of animals with tumors weighing 10-80 g, there was a large percentage of cells that could not be identified as T or B cells; they are probably null (K) cells. It is concluded that tumor progression modulates the equilibrium between T-cell subpopulations with antagonistic functions, which leads to disturbances with kinetics of immunologically competent cells. (29 refs)

79-3991 A Role for Elevated H-2 Antigen Expression in Resistance to Neoplasia Caused by Radiation-induced Leukemia Virus. Enhancement of Effective Tumor Surveillance by Killer Lymphocytes. (Eng) Meruelo, D. (Irvington Hous. Inst., Dept. Pathology, New York Univ. Medical Center, New York, NY 10016). *J Exp Med* 149(4): 898-909; 1979.

Observations suggesting that elevated histocompatibility-2 complex (H-2) antigen expression enhances the effectiveness of the host's immune responses to virus-infected cells and may function in resistance to leukemogenesis are presented. First, cell-mediated immunity against radiation-induced leukemia virus (RadLV)-transformed or -infected cells can be detected with ease when H-2-positive target cells are used in the cell-mediated lympholysis (CML) assay. (Although RadLV-transformed cells obtained from overtly leukemic animals and maintained in tissue culture are H-2-negative, these cells can regain their H-2 phenotype by in vivo passage in normal animals. The H-2-negative cells are poor targets in a CML assay). Second, resistant mice develop greater numbers of effectors when infected

with RadLV than do susceptible mice. Third, injection of normal (uninfected) thymocytes into syngeneic recipients of resistant or susceptible H-2 type does not stimulate a CML response. However, injection of RadLV-infected thymocytes from resistant mice produces a vigorous CMI response, and such thymocytes elicit the strongest response at a time when both H-2 and viral antigen expression is elevated. By contrast, injection of infected thymocytes from susceptible mice, which express viral antigens but low levels of H-2 antigens, does not stimulate a CML reaction. These findings may explain the easier induction of leukemia found by many investigators when virus is inoculated into neonatal mice and the preferential thymus tropism of some oncogenic C-type RNA viruses. Cells expressing very low levels of H-2, such as thymocytes, may serve as permissive targets for virus infection because they lack an important component (H-2 antigens) of the dual or altered recognition signal required to trigger a defensive host immune response. (19 refs)

79-3992 Longterm Effects of Neonatal Anterior Hypophyseal Isografts on the Mammary Gland of Mammary Tumor Virus-expressed Mice. (Eng) Mori, T. (Zoological Inst., Faculty Science, Univ. Tokyo, Hongo, Tokyo 113, Japan); Bern, H. A. *Proc Soc Exp Biol Med* 161(1): 48-52; 1979.

The effects of neonatal anterior hypophysial (HP) implantation on the mammary glands were studied in female mice of the mammary tumor virus (MTV)-expressed BALB/cfC3H/Crgl (BALB/c) and SHN strains. Some of the mice received one-half of an anterior hypophysis sc on the back, and others received an entire anterior hypophysis in the right number 4 mammary fat pad (MFP). In some of the latter animals, the right MFP was removed at 21-23 days of age. Based on vaginal smears, 7/28 BALB/cfC3H mice given HP isografts showed prolonged estrus and 5 showed prolonged diestrus. Thus, HP implantation resulted in a significant occurrence of irregular estrous cycles. All females receiving HP isografts had a greatly increased incidence of hyperplastic alveolar nodules (HAN) and mammary tumors. In the BALB/c mice, 14/28 females had tumors by 6-12 mo of age and 16 animals had HAN at 1 yr of age, compared with 4/15 control mice with HAN and 1 with a small tumor. In the SHN mice, all experimental animals had HAN and 21/28 females had tumors by 4-7 mo of age. In the control group, 6/23 animals had tumors by 5-7 mo of age. When the right MFP bearing the isograft was removed at 21-23 days, development of mammary tumors and HAN was still greater in the experimental group than in the control group. This suggests that a relatively short-term exposure to neonatal HP grafts (presumably secreting prolactin) during early postnatal life results in permanent alterations in the development of mammary glands and in the early onset of mammary tumorigenesis. (19 refs)

- 79-3993 A Human Testicular Teratoma Serially Transplanted in Immune-deprived Mice.** (Eng) Selby, P. J. (Dept. Medicine, Royal Marsden Hosp., Sutton, Surrey, England); Heyderman, E.; Gibbs, J.; Peckham, M. J. *Br J Cancer* 39(5): 578-583; 1979.

The serial sc transplantation of a human chorionic gonadotropin (HCG)-producing human teratoma into thymectomized, irradiated CBA/lac mice is described. The xenograft was established from a metastatic sc nodule excised from a 21-yr-old man with a trophoblastic malignant teratoma of the testis. The xenograft (HX36) grew as a cystic mass containing blood-stained fluid. The volume-double time, 11-12 days, was the same as that for the patient's pulmonary metastases. The original tumor tissue and cells from passages 1 through 5 contained abundant HCG-positive cells, whereas cells from later passages contained only one HCG-positive cell. Autoradiography and immunoperoxidase staining gave similar results for both the xenograft and original tumor. β -HCG levels in the sera of xenograft-bearing mice were 500, 180, and 2.6 μ g/liter during passages 3, 4, and 6, respectively; HCG levels in the fluid from the center of the xenografted tumor were 36,000, 22,500, and 19,000 μ g/liter during passages 2, 3, and 4, respectively; and the B-HCG level in the patient's serum was 7.5 μ g/liter after unsuccessful chemotherapy. (15 refs)

- 79-3994 Helper and Suppressor T-Lymphocyte Leukemia in Ataxia Telangiectasia.** (Eng) Saxton, A. (Dept. Medicine, Div. Clinical Immunology/Allergy, Univ. California, Los Angeles, Sch. Medicine, Los Angeles, CA 90024); Stevens, R. H.; Golde, D. W. *N Engl J Med* 300(13): 700-704; 1979.

The nature of the lymphoid leukemia that developed in one of two siblings with ataxia telangiectasia was investigated. The leukemia cells were shown to be T lymphocytes, and they carried a characteristic 14q+ chromosome tandem translocation. This chromosome abnormality had been identified 11 years earlier among the patient's 'normal' lymphocytes. The patient's neoplastic T lymphocytes in vitro provided helper and suppressor T-lymphocyte activity equivalent to that of normal T lymphocytes. Some neoplastic T lymphocytes bore a receptor for the Fc portion of IgM (45% T θ), whereas others carried receptors for the Fc portion of IgG (10% T γ). All the T μ and T γ lymphocytes possessed the chromosome 14 abnormality. These data suggest that neoplastic transformation occurred in an uncommitted T lymphocyte that was capable of further differentiation into the distinct pathways for help and suppression, in a lymphoid analogy of chronic myelogenous leukemia. (44 refs)

- tients with Various Stages. (Eng) Ueda, K. (Dept. Obstetrics and Gynecology, Osaka City Univ. Medical Sch., 1-5-7, Asahi-machi, Abeno-ku, Osaka, Japan); Umesaki, N.; Nakamori, H.; Sako, H.; Kinoshita, Y.; Sugawa, T. *Gynecol Oncol* 7(1): 66-70; 1979.

The in vitro effect of human fetal thymic extract (hFTE) on the concanavalin A (Con A) responsiveness of peripheral blood lymphocytes from 28 patients with cervical cancer and 9 healthy volunteers was determined. The results were expressed as the percentage increase of the Con A response of hFTE-treated cells over that of nontreated cells. The increase was 14% in healthy volunteers, 34% in patients with Stage 0 or I cervical cancer, 12% in those with Stage II cancer, and 0% in those with Stage III or IV cancer. The increase among patients with carcinoma in situ or Stage 0 or I cancers was significantly greater than that in the normal subjects. Both the Con A response and the T-cell count per unit vol of the peripheral blood decreased as malignancy proceeded to Stages III and IV. The results support the hypothesis that disordered lymphocyte differentiation in the thymus contributes to carcinogenesis. (18 refs)

- 79-3996 Changes in T-Cell Subsets and Their Clinical Significance in Cancer Patients.** (Eng) Yata, J. (Dept. Pediatrics, Toho Univ., Omorinishi, Otaku, Tokyo, Japan); Shimbo, T.; Sawaki, S. *IARC Sci Publ* 20: 511-521; 1978.

Changes in T-cell subsets in patients with different types of cancer were studied. The proportion of T cells with receptors for the Fc portion of IgG (IgG-Fc R+ T cells) among circulating T cells was <10% in normal subjects and markedly increased in certain cancer patients, particularly those with stomach, nasopharyngeal, or uterine cancer. The percentage of IgG-Fc R+ T cells was higher in stomach cancer patients whose tumors had progressed too far for resection than in those whose tumors could be removed completely. The percentage fell to within normal ranges 1-2 wk after complete tumor removal in the latter group. Responsiveness to phytohemagglutinin, but not to concanavalin A, was decreased in lymphocyte populations with high percentages of IgG-Fc R+ T cells. A reverse relationship was observed between helper T-cell function and increased proportions of IgG-Fc R+ T cells. Among patients with nasopharyngeal carcinoma, the percentages of IgG-Fc R+ T cells were highest in those with advanced, relapsed, or metastasizing tumors and lowest in those who had been treated with radiation and had been free of tumor for >6 mo. The data suggest that IgG-Fc R+ T cells may be suppressor T cells and that the percentage of these cells among circulating T cells is related to the presence of tumor and reflects the status of host-tumor interactions. (7 refs)

- 79-3995 Effect of Fetal Thymic Extract on Maturation of Precursor Lymphocytes from Cancer Pa-**

- 79-3997 Immunosurveillance in Pre-Malignant Occupational Bladder Disease.** (Eng) Taylor, G.

(Appalachian Lab. Occupational Safety and Health, US Public Health Service, 944 Chestnut Ridge Road, Morgantown, W. VA 26505); Kumar, S.; Brenchley, P.; Wilson, P.; Costello, B.; Shaw, G. H. *Int J Cancer* 23(4): 487-493; 1979.

Lymphocyte reactivity against bladder cells from a patient with Grade II transitional cell carcinoma of the bladder and osteogenic sarcoma cells was studied in 93 clinically normal male workers exposed to bladder carcinogens, 23 untreated patients with transitional cell carcinoma of the bladder, and 25 healthy male subjects. Compared with the healthy subjects, the carcinogen-exposed, clinically normal workers and the bladder cancer patients had similar increases in lymphocyte reactivity toward the bladder cancer cells. This increase in reactivity was not demonstrable in either group when the osteogenic sarcoma cells were used, indicating specificity. The increased immunoreactivity resided in a subgroup of workers who experienced higher and more continuous carcinogen exposure than the others. Further, there was a correlation between increased specific reactivity toward the bladder cancer cells and exfoliative cytology results suggestive of early malignant change of the urothelium, in the small proportion of workers who exhibited such changes. Thus, the immune recognition of tumor antigen appears to occur before the development of overt malignancy. (17 refs)

79-3998 Direct Evidence that Natural Killer Cells in Nonimmune Spleen Cell Populations Prevent Tumor Growth In Vivo. (Eng) Kasai, M. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA 02115); Leclerc, J. C.; McVay-Boudreau, L.; Shen, F. W.; Cantor, H. *J Exp Med* 149(5): 1260-1264; 1979.

The ability of positively selected natural killer (NK) spleen cells from nonimmune mice (B6, BALB/c, and A/J) to prevent the in vivo growth of lymphomas was studied. Spleen cell populations depleted of Ly5⁺ cells exerted virtually no detectable NK activity, whereas populations with the cell-surface phenotype Ig-Thy1⁺ and containing approx 80% Ly5⁺ cells showed a four- to sixfold greater NK activity than unselected populations. The growth of sc injected BALB/c radiation-induced leukemia cells or YAC Moloney virus-induced lymphoma cells in BALB/c and A/J mice, respectively, was not affected by coinjection of the following syngeneic lymphocyte populations: nonimmune spleen cells, nylon-filtered spleen cells, Thy1⁺ spleen cells, Ig⁺ spleen cells, or Thy1⁺ spleen cells. In contrast, spleen cells expressing the Ig-Thy1-Ly5⁺ surface phenotype (which account for <5% of the total spleen cell population) conferred complete protection against tumor growth. (19 refs)

79-3999 Target-Effector Interaction in the Natural Killer Cell System: Isolation of Target

Structures. (Eng) Roder, J. C. (Dept. Zoology, Univ. Coll. London, Gower St., London WC1E 6BT, England); Rosen, A.; Fenyo, E. M.; Troy, F. A. *Proc Natl Acad Sci USA* 76(3): 1405-1409; 1979.

The target molecules for natural killer (NK) cells were isolated from several mouse and human tumor cell lines in a target-binding cell assay. Preincubation of NK cells with detergent-solubilized cell-surface proteins in mouse YAC lymphoma cells prevented subsequent binding to intact YAC targets. The NK target structures (NK-TS) consisted of three molecular species of mol wt 130,000, 160,000, and 240,000, based on their electrophoretic mobility in sodium dodecyl sulfate polyacrylamide gels. Moloney cell surface antigen (MCSA), gp71, p39, H-2, and NK-TS were localized in distinct fractions of gels. The NK-TS bound to concanavalin A-Sepharose columns and could be eluted with α -methyl-D-mannoside, suggesting that the NK-TS may be glycoprotein. NK-TS molecules could not be detected in gels of NK-insensitive target cells. The quantity of NK-TS obtained from the gels varied directly with the NK sensitivity of YAC, which is more sensitive when grown in vitro than in vivo. The NK-TS molecules specifically inhibited the binding of NK cells, but not alloimmune T cells, to their appropriate targets. NK-sensitive targets of diverse origin exhibited similarities in their target structure, sharing some but not all of the target molecules. In cross-inhibition experiments, the two human lines K562 and Molt-4 shared specificities, as did YAC and MPC-11, another mouse cell line, but neither of these two groups cross-reacted with each other. These results suggest that NK cells recognize more than one specificity. (13 refs)

79-4000 Defective Tumoricidal Capacity of Macrophages from A/J Mice. I. Characterization of the Macrophage Cytotoxic Defect after In Vivo and In Vitro Activation Stimuli. (Eng) Boraschi, D. (Immunopathology Section, Lab. Immunobiology, Immunology Program, Div. Cancer Biology and Diagnosis, NCI, NIH, Bethesda, MD 20014); Meltzer, M. S. *J Immunol* 122(4): 1587-1591; 1979.

The characteristics of the tumoricidal defect of macrophages from A-derived mouse strains were examined using A/J mice as the reference strain. Macrophages from A/J mice failed to develop tumoricidal activity after any of several in vivo or in vitro treatments that activate cells from C3H/HeN mice. Peritoneal macrophages from A/J mice treated ip with viable *Mycobacterium bovis*, strain BCG, killed *Corynebacterium parvum*, or pyran copolymer failed to develop in vitro tumoricidal activity. Varying the number of macrophages from treated mice added to target cells, the dose and time of treatment, or the treatment schedule of these in vivo activation stimuli did not evoke cytotoxic activity. Moreover, cytotoxic activity by macrophages from A/J mice was not observed with any of four target cell lines derived from three different mouse

strains. In vitro treatment of peritoneal exudate macrophages from A/J mice with lymphokine-rich supernatants, bacterial endotoxins, or T-cell mitogens was also ineffective. Varying the numbers of treated macrophages added to target cells, the dose of in vitro activation stimuli, or the time of treatment did not evoke cytotoxic activity. Thus, A/J mice exhibit a profound defect in macrophage tumoricidal capacity to both in vivo and in vitro activation stimuli over a wide range of experimental conditions. (21 refs)

- 79-4001 Defective Tumoricidal Capacity of Macrophages from A/J Mice. II. Comparison of the Macrophage Cytotoxic Defect of A/J Mice with That of Lipid A-Unresponsive C3H/HeJ Mice.** (Eng) Boraschi, D. (Immunopathology Sect., Lab. Immunobiology, Immunology Program, Div. Cancer Biology and Diagnosis, NCI, NIH, Bethesda, MD 20014); Meltzer, M. S. *J Immunol* 122(4): 1592-1597; 1979.

The macrophage (MP) cytotoxic defect of A/J mice, which fail to develop tumoricidal activity after any of several in vivo or in vitro treatments that activate cells from other mouse strains, was compared with that of lipid A-unresponsive C3H/HeJ mice. MP's from *Mycobacterium bovis* strain BCG-infected C3H/HeN mice were highly cytotoxic to tumor cells in vitro, but cells from BCG-infected A/J mice were not. Inflammatory responses to BCG infection in A/J mice were relatively normal, and production of MP activation factor activity by tuberculin-stimulated BCG-immune spleen cell cultures was also intact. MP's from BCG-infected but not control A/J mice developed tumoricidal activity after further in vitro treatment with lymphokines, bacterial lipopolysaccharides (LPS), or certain plant lectins. Peritoneal exudate MP's first treated with lymphokines also developed cytotoxic activity after further treatment with LPS or lectins. The phenotypic expression of the A/J genetic defect in MP cytotoxic activity was very similar to the defect of lipid A-unresponsive C3H/HeJ mice, except for the concentration of LPS required for cytotoxic activity by lymphokine-treated cells. Responses of A/J mice to the lethal toxicity of LPS or of A/J MP's to the direct toxicity of LPS in vitro, responses influenced by the *Lps* gene, were normal. However, MP cytotoxic activity and spleen cell proliferative responses to LPS in vitro, also controlled by the *Lps* gene, were abnormal. It is concluded that the genetic basis for the MP tumoricidal defect of A/J mice is probably a gene mutation(s) at a locus other than the *Lps* gene. (27 refs)

- 79-4002 Genetics of the Immune Response to Tumor-specific Cell-surface Antigens.** (Eng) Abeyounis, C. J. (Dept. Microbiology, State Univ. New

York at Buffalo, Buffalo, NY 14214); Zaleski, M. B.; Milgrom, F. *Transplant Proc* 11(1): 1066-1068; 1979.

The influence of genetics on the humoral response to tumor-specific cell-surface antigens (TSCSA) was studied in C57BL/6 (B6) mice and several of their F₁ hybrids. B6 mice and their male and female F₁ hybrids were injected ip with 10⁷ irradiated EL-4 tumor cells 1x/wk for 6 wk. Sera from these animals were tested against EL-4 cells by the cytotoxicity in agar gel procedure. B6 hybrids bearing the H-2b and H-2d haplotype responded to EL-4 cells in a manner similar to that of the B6 parental strain, but hybrids bearing the H-2k haplotype showed little or no response. These results suggest that the humoral response to TSCSA may be under genetic control. Other experiments indicated that genes at the H-2 complex may influence responsiveness to EL-4 cells. [B6 x B10.A(3R)]F₁ hybrids, which have the recombinant haplotype H-2³, showed much greater response than the B6 parent or hybrids carrying either the H-2b or H-2d haplotypes. This increased responsiveness may be due to complementation between genes within the same haplotype coupled with the presence of the proper background genes. These results support the argument that the humoral response to TSCSA is under genetic control, but they do not exclude the possibility that the observed response differences result from environmental factors. (7 refs)

- 79-4003 H-2 Antigen Variants in a Cultured Heterozygous Mouse Leukemia Cell Line. IV. Cell-mediated Cytotoxicity of Wild Type and Variant Cells.** (Eng) Rajan, T. V. (Dept. Pathology and Genetics, Albert Einstein Coll. Medicine, Bronx, NY 10461). *Immunogenetics* 7(5): 457-464; 1978.

Histocompatibility (H-2) antigen variants, derived from a heterozygous mouse Friend leukemia cell line by selection with anti-H-antisera and complement, were tested for susceptibility to cell-mediated cytotoxicity (CML) using T lymphocytes directed against individual H-2 antigens. For most of the cell lines tested, the pattern of susceptibility or resistance to lysis by cytotoxic T lymphocytes was consistent with the phenotypes assigned to them as a result of selection with antisera. The exception was -4+31 clone 1. This variant was selected for its resistance to lysis by an anti-H-2Dd antiserum and complement. Quantitative absorption assays showed that it did not express detectable H-2Dd; however, the cell line was susceptible to lysis by anti-H-2Dd killer cells. The reason for this disparity between serological and CML data is not clear. (15 refs)

- 79-4004 Shared Chemical Properties of Different Murine Thymus-Leukemia Antigens.** (Eng) Pischel, K. D. (Div. Biology and Biomedical Science,

Washington Univ. Sch. Medicine, St. Louis, MO 63110); Little, J. R. *J Immunol* 122(5): 1821-1827; 1979.

Structural similarities between different thymus-leukemia antigens (TLA) coded for by genes of the murine *Tla* locus were studied. On polyacrylamide gels, TLA components of thymocytes of B/6-*Tlaa* mice showed major bands corresponding to the heavy chain (mol wt, 45,000) and β 2-microglobulin light chain (β 2m) of H-2Kb. Similar TLA subunit patterns were obtained from cells of the *Tlac*, *Tlad*, and *Tlab* (derepressed) haplotypes. The TLA did not exhibit Fc receptor properties, and they did not adsorb to murine leukemia virus antigens after isolation and separation on polyacrylamide gels. Trypsin digestion resulted in the conversion of TLA heavy chains to a relatively trypsin-resistant 37,000-dalton core that remained associated with β 2m and reacted with anti-TLA. The findings strongly support the hypothesis that TLA comprise a family of chemically similar antigens belonging to a structurally related group that includes H-2D, H-2K, and Qa-2,3, all of which are genetically linked on chromosome 17. (36 refs)

79-4005 Lung Tumor-reactive Cytotoxic Lymphocytes Generated in Mixed Lymphocyte Reaction Between C3HfB/HeN (H-2kb) and C3H/HeN (H-2k) Strain Mice. (Eng) Imamura, M. (Dept. Health, Education and Welfare, Rockville Pike, Bethesda, MD 20014); Gipsen, T. G.; Bensky, N.; Justice, R.; Martin, W. J. *J Immunol* 122(5): 1863-1866; 1979.

Mixed lymphocyte reactions (MLR) were used to characterize the distinction between C3HfB/HeN mice and other strains of mice of known H-2k haplotype. Significant reciprocal stimulation was observed in MLR containing spleen cells from C3H and C3Hf mice, MLR containing C3Hf and CBA spleen cells, and MLR containing C3H or C3Hf and BALB/c or B10 spleen cells. No significant stimulation was observed between C3H and CBA spleen cells. C3Hf-derived spleen cells stimulated in MLR with C3H-derived spleen cells were cytotoxic for C3H and B10.A(4R)-derived target cells. Spleen cells from C3H anti-C3Hf MLR were cytotoxic for C3Hf, but not C3H, B10, or B10.A(4R) target cells. Cytotoxic activity developed in C3Hf anti-C3H MLR but not in C3H anti-C3Hf MLR. Cytotoxic activity against C3H and lung tumor 85 target cells was markedly reduced by pretreatment of C3H-stimulated C3Hf cells with anti- θ serum plus rabbit complement. The cytotoxic cells were nonadherent to nylon wool. At an effector:tumor cell ratio of 10:1, cells from C3Hf anti-C3H MLR and C3Hf anti-A MLR completely protected against the outgrowth of 5×10^4 lung tumor 85 cells in x-irradiated C3Hf mice. (15 refs)

79-4006 Cell-mediated Reactivity to Antigens Shared by Moloney-Virus-induced Lymphomas

(LSTRA) and Certain 3-Methylcholanthrene-induced Mouse Sarcomas. (Eng) Hellstrom, I. (Div. Tumor Immunology and Pediatric Oncology, Fred Hutchinson Cancer Res. Center, Seattle, WA); Hellstrom, K. E.; Zeidman, L.; Bernstein, I. D.; Brown, J. P. *Int J Cancer* 23(4): 555-564; 1979.

Spleen cells (SC) from BALB/c mice whose primary Moloney sarcoma virus (MSV)-induced sarcomas had spontaneously regressed and from normal, untreated BALB/c mice were cocultivated for 5 days with mitomycin-C-treated LSTRA cells; LSTRA is a BALB/c Moloney lymphoma that shares cell-surface antigens with MSV-induced sarcomas. These SC, referred to as cocultivated Moloney regressor (CMR) and cocultivated untreated (CU) cells, respectively, were cytotoxic to LSTRA cells in 3-hr ^{51}Cr -release assays; CMR cells showed, in most cases, the greatest lytic activity against LSTRA targets. The same SC were also reactive, in 20-hr microcytotoxicity and ^{51}Cr -release assays, against target cells from a variety of transplanted sarcomas induced by 3-MC in BALB/c mice. The highest reactivity was seen when CMR or CU cells were tested against target cells from sarcoma lines that expressed an NB-ecotropic MuLV cross-reacting serologically with Moloney virus. Reactivity against isotope-labeled tumor cells expressing MuLV-associated cell-surface antigens could be competitively inhibited by adding unlabeled tumor cells expressing such antigens. Finally, Winn assays were performed in which CMR cells strongly inhibited the outgrowth of cells from three sarcoma lines that express NB-ecotropic MuLV. There was less but significant inhibition of cells from some other 3-MC-induced sarcomas, either negative for the expression of MuLV-associated antigens or expressing the N-ecotropic endogenous BALB/c MuLV. CU cells enhanced tumor outgrowth in Winn assays at least as often as they inhibited it. (22 refs)

79-4007 Opposing Effects of Cryostat Sections of Preneoplastic and Neoplastic Mouse Mammary Lesions on In Vitro Migration of Peritoneal Exudate Cells. (Eng) Wei, W. Z. (Dept. Pathology, Univ. Connecticut Health Center, Farmington, CT 06032); Miller, F. R.; Blazar, B. A.; Medina, D.; Heppner, G. *J Immunol* 122(5): 2059-2067; 1979.

Cryostat sections of normal tissue and of preneoplastic hyperplastic alveolar nodules (HAN's) and neoplastic mammary tumors from BALB/c Crgl mice were used as "antigens" in macrophage inhibition tests. HAN cryostat sections including those grown in nude mice, exerted nonspecific migration inhibition effects on peritoneal exudate cells (PEC). A variety of normal, syngeneic tissue cryostat sections, particularly those of mammary gland and neonatal liver, also inhibited mouse PEC. The inhibition of macrophage migration was not mediated by a lymphocyte-released lymphokine. When serum from HAN-sensitized mice was used in place of normal mouse serum in the tests,

the migration inhibitory effect of the cryostat sections was abolished. Cryostat sections of mammary tumors did not inhibit and, occasionally, enhanced PEC migration. Also, the presence of tumor cryostat sections and eluates interfered with the inhibition induced by HAN cryostat sections and by purified protein derivatives with PEC from donors sensitized to that antigen. Histologic examination of HAN and mammary tumor tissue revealed inflammatory cells distributed diffusely in the former and localized peripherally around the latter type of lesion. (27 refs)

- 79-4008 Expression of Skin Antigen on Cell-Surface of Rat Urinary Bladder Epithelial Cells in Carcinogenesis.** (Eng) Hashimoto, Y. (Dept. Hygienic Chemistry, Pharmaceutical Inst., Tohoku Univ., Aobayama, Sendai 980, Japan); Masuko, T. *Gann* 70(2): 259-260; 1979.

Cell-surface antigens were detected in normal and malignant rat urinary bladder epithelial cells with the use of antisera raised in rabbits immunized with normal rat urinary bladder epithelial (NBE) cells and with bladder cancer (BC-47) cells established as a transplantation line from a tumor induced by N-butyl-N-(4-hydroxybutyl) nitrosamine (BBN). Both NBE and BC cells had an antigen common to lymphoid cells. Both NBE and BC cells also had an antigen common to cells of the lung, intestine, kidney, and skin but not common to muscle, liver, heart, and brain cells. NBE cells had an antigen that was absent in other normal cells but was present in preneoplastic (nodular hyperplasia and papilloma) and BC cells. BC cells had an antigen commonly present in in vivo- and in vitro-induced bladder cancer cells and common to epidermal cells but not present in NBE or other normal rat tissues. This antigen was detected in the bladder epithelial cells of ACI/N rats with BBN-induced nodular hyperplasia or papillomas (induced by 8 wk of treatment), papillomas (12 wk), and cancer (16-25 wk). (3 refs)

- 79-4009 Immunogenicity of N-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide-induced Bladder Cancer.** (Eng) Soloway, M. S. (Dept. Urology, Univ. Tennessee Center Health Sciences, Memphis, TN 38163); Martino, C.; Hyatt, C.; Marrone, J. C. *Natl Cancer Inst Monogr* (49): 293-300; 1978.

Five transplantable transitional cell carcinomas induced by the feeding of 0.1% N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) to female C3H/HeJ mice were tested for individual immunogenicity and the presence of cross-reacting tumor antigens. Three tumors (MBT-2, MBT-3, and MBT-1123) were sufficiently immunogenic to decrease the incidence of tumor growth in syngeneic mice immunized to the same tumor relative to nonimmunized controls. However, mice immunized to MBT-1123 by the

growth and amputation of this tumor exhibited no evidence of protection against the induction of primary FANFT tumors. The lack of cross-reacting tumor antigens has important implications for the use of allogeneic tumor cells as an antigen source in immunotherapy. (23 refs)

- 79-4010 Genetic Control of In Vivo Immunity to Tumor-specific Transplantation Antigens of Chemically Induced Murine Fibrosarcomas.** (Eng) Parmiani, G. (Div. Experimental Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Via G. Venezian 1, 20133 Milan, Italy); Ballinari, D. *Int J Cancer* 23(5): 697-705; 1979.

The possible genetic control of in vivo immunity to the tumor-specific transplantation antigens (TSTA) of two methylcholanthrene-induced BALB/c fibrosarcomas, C-1 and ST2, was studied in F₁ compatible hosts. The tumors had been shown to share their TSTA. Both tumors lost their immunogenicity in (BALB/c x C3Hf)F₁ mice, and C-1 also lost its immunogenicity in (BALB/c x BALB.K)F₁ mice. The immunogenicity of C-1 and ST2 remained unchanged in other combinations of BALB/c with H-2b or H-2q mice. The primary and secondary immune response to C-1 and the secondary immunity to ST2 were evaluated in BALB/c x (BALB/c x C3Hf) backcross mice. Results for both tumors were compatible with the presence of a histocompatibility (H-2) dominant gene whose products suppress immunity to TSTA; the linkage of low antitumor responsiveness with H-2k was demonstrated for C-1 and ST2. An attempt was made to map gene(s) governing the low immune response to C-1 and ST2 by studying the antitumor immunity of several hybrids. The Kk and Dk regions were not necessary for expression of the low immunity to C-1; therefore, the relevant gene(s) were mapped in the Ik region. The low immune response of (BALB/c x C3H.OH)F₁ mice to C-1 suggests that Dk genes are also important. For ST2, only BALB/c x C3H.OH animals showed a low antitumor response; thus, genes coding for suppression of anti-ST2 immunity were mapped in the Dk region. The results also indicate that non-H-2 factors may counteract the suppressive activity of major histocompatibility complex genes. (27 refs)

- 79-4011 Isolation and Characterization of Detergent-solubilized Human HLA-DR Transplantation Antigens.** (Eng) Klareskog, L. (Dept. Cell Res., Biomedical Center, Univ. Uppsala, Uppsala, Sweden); Tragardh, L.; Rask, L.; Peterson, P. A. *Biochemistry* 18(8): 1481-1489; 1979.

The isolation of HLA-DR antigens from surgically removed spleens, in vitro grown lymphoblastoid (Daudi) cells, and WBC obtained from chronic lymphatic leukemia pa-

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tients is described, along with the chemical and physicochemical characteristics of the antigens. Highly purified detergent-solubilized HLA-DR antigens were obtained by affinity chromatography on a *Lens culinaris* lectin column, two gel chromatography procedures, immunosorbent purification, and diethylaminoethyl-Sephadex chromatography. In the two gel chromatography steps, the HLA-DR antigens were separated into two fractions. The larger size material, fraction I, was converted to the smaller form, fraction II, upon storage. Fractions I and II were shown to be highly purified by chemical, physicochemical, and immunological criteria. Material in both fractions appeared equally reactive with xenoantisera and alloantisera, suggesting that antigenic differences did not account for the size separation. HLA-DR antigens in fractions I and II displayed identical profiles upon isoelectric focusing, and both were heterogeneous with regard to charge. Mol wt determinations by gel chromatography in 6 M guanidine hydrochloride and by sodium dodecyl sulfate-polyacrylamide gel electrophoresis indicated that the HLA-DR antigens in fractions I and II were composed of two types of polypeptide chains with apparent mol wts of 28,000 and 34,000. The two HLA-DR antigen subunits could be dissociated and separated by isoelectric focusing in 9 M urea. The separated chains bound detergents in micellar form, as revealed by charge shift electrophoresis and gel chromatography separations. This result strongly suggests that both HLA-DR antigen chains are integrated into the hydrocarbon matrix of the cell membrane. Some physicochemical properties of the isolated HLA-DR antigens in fractions I and II were determined. (49 refs)

- 79-4012 HLA in Populations: An Approach for Genetical Susceptibility to Cancer.** (Eng) Feingold, N. (Laboratoire de Genetique Epidemiologique, U 155 de l'INSERM, Chateau de Longchamp, Bois de Boulogne, 75016 Paris, France); Degos, L.; Feingold, J. *J Immunogenet* 6(1): 29-35; 1979.

Geographic correlations between the incidence of various cancers and the major histocompatibility complex (HLA system) and the ABO blood group system were studied. Twenty-six populations were studied, 22 Caucasian and 4 comprising Japanese, Mexicans, black Americans, and Venezuelans. The principal positive associations were between breast and colorectal carcinoma and the HLA A1, B8, and B12 antigens. These two cancers were negatively correlated with the HLA BW35 antigen. There was also a negative correlation between prostate carcinoma and B12. The well-known association of the ABO blood group with stomach carcinomas was confirmed. In the European populations, there was a correlation of O antigen with carcinoma of the breast and intestine. The geographic distributions do not exclude a true association between the HLA complex and cancer, but they may also be explained by a third factor related to HLA and cancer. This factor is probably a genetic background of susceptibility (or

resistance) to cancer. The alternative hypothesis of an environmental factor to explain these associations is not likely, since there is no evidence of environmental influences on HLA gene frequencies. (13 refs)

- 79-4013 Possible Relationship Between Antibody Responsive Capacity of the Host and Growth of Transplantable Tumour.** (Eng) Sarkar, R. K. (Chittaranjan Natl. Cancer Res. Center, 37 S.P. Mookerjee Road, Calcutta-700026, India); Mallik, R. K.; Dutta, S. R.; Mitra, S. *Indian J Med Res* 69: 482-488; 1979.

An attempt was made to correlate the growth of sarcoma 180 (S180) solid tumor with the antibody-response capacity of the host. Spleens were removed from inbred female albino Swiss mice at 48 hr or 5 wk of age. The animals were then inoculated with 2×10^7 viable S180 cells, and tumor volume was measured at different times posttransplantation (PT). Tumor growth was suppressed in adult splenectomized (ASP) mice during the first 3 wk PT compared with that in neonatally splenectomized (NSP) mice and nonsplenectomized controls. By the fourth week, however, the mean tumor volume in all three groups was almost equal, suggesting that the factor that induced tumor growth retardation at the earlier stages was not effective when the tumor load increased. There was no significant difference in mean survival time among the different groups. Immunoglobulins (Ig) were estimated from the serum pooled from six mice from each group on day 25 PT. There was a significant increase of serum Ig levels in all the groups, and Ig levels in the tumor-bearing mice were similar to those in the splenectomized and control mice. S180 growth was also examined in NSP and normal animals that were treated with cyclophosphamide (CP: 300 mg/kg in normal saline, ip) 24 hr before transplantation. Tumor growth was significantly reduced in CP-treated normal mice but not in CP-treated NSP mice, compared with the untreated group. The increased growth rate of S180 in ASP mice, after the initial retardation, may be due to the increased production of enhancing antibodies as a result of the stimulation of lymphoreticular tissues by progressive tumor growth. (17 refs)

- 79-4014 Production of Xenoantiserum to a Chemically Induced Murine Tumor: Analysis of Tumor Specificity in Different Immune Tests.** (Eng) Pugliese, O. (Istituto Superiore Sanita, Rome, Italy); Centis, D.; Nicotra, M. R.; Natali, P. G. *Immunol Commun* 8(1): 107-118; 1979.

Rabbit xenoantiserum to a methylcholanthrene-induced murine (C3H) sarcoma (MCIM) was absorbed in vivo in syngeneic normal host animals, and its specificity for the immunizing tumor was investigated. Before absorption, the anti-MCIM serum exhibited complement-dependent

cytotoxicity with MCIM cells and C₃H splenocytes. After 5 min in vivo absorption, all anti-C₃H activity was abolished, and after 10 min the level of anti-MCIM activity was 10% of the initial value. A lack of tissue and organ-specific antibodies in the absorbed antiserum was demonstrated, but the antiserum maintained its tumor specificity according to the antibody-dependent cell cytotoxicity test. A second chemically induced C₃H sarcoma showed approx 50% cross-reactivity with the serum and two murine leukemia virus-induced tumor cell lines showed 25% cross-reactivity; other unrelated tumors and syngeneic and allogeneic normal cells showed very little. Thus, in vivo absorbed antisera appear to be valuable reagents for the immunochemical characterization of chemically induced tumors. (21 refs)

- 79-4015 Characterization of the Spontaneous Murine B Cell Leukemia (BCL₁). III. Evidence for Monoclonality by Using an Anti-idiotypic Antibody.** (Eng) Vitetta, E. S. (Dept. Microbiology and Cancer Center, Univ. Texas Southwestern Medical Sch., Dallas, TX); Yuan, D.; Krolick, K.; Isakson, P.; Knapp, M.; Slavin, S.; Strober, S. *J Immunol* 122(5): 1649-1654; 1979.

An anti-idiotypic (anti-Id) antibody was raised against the cell-surface IgM of murine B-cell leukemia (BCL₁) cells. The anti-Id serum recognized the cell-surface IgM molecules on BCL₁ splenocytes but not on normal BALB/c splenocytes. Neither of two myeloma proteins, MOPC-104E (μ , λ_1) and MOPC-315 (α , λ_2), was bound by anti-Id, but both were bound by anti- λ . Also, anti-Id bound no molecules from normal BALB/c serum and only a small number of molecules from the serum of BCL₁-bearing mice. The anti-Id serum recognized the secreted IgM from lipopolysaccharide (LPS)-stimulated BCL₁ cells but not that from LPS-stimulated normal BALB/c splenocytes. The anti-Id serum also recognized a population of large cells in BCL₁-bearing mice that was not present in normal animals. (10 refs)

- 79-4016 Immunological Analysis of A Strain Mice Bearing the A-10 Mammary Adenocarcinoma.** (Eng) Tax, A. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA 19104); Manson, L. A. *Cancer Res* 39(5): 1739-1747; 1979.

An attempt was made to determine whether antitumor antibodies are produced by A strain mice during the growth of a transplantable mammary adenocarcinoma (A-10). A strain mice have a high mammary tumor incidence. The antibody response was monitored by a sensitive radioimmunoassay that can detect 1 nanogram of antibody. No evidence of a humoral antitumor response was observed in animals given ip or sc injections of A-10 ascites cells. Control experiments showed that a humoral response was detectable 1 wk after the inoculation of an allogeneic

tumor. Immunoglobulin (Ig) binds nonspecifically to cells via an Fc portion of the Ig molecule, and this was seen with a tumor-bearer serum pool and with Ig preparations eluted from A-10 ascites cells. No specific antitumor antibody was found in these sources. The A strain mice could not be immunized to reject a challenge of live A-10 cells with mitomycin C-treated A-10 cells, with neuraminidase-treated A-10 cells, or with A-10 membrane preparations. It was concluded that the A-10 tumor is not immunogenic in its host of origin. (30 refs)

- 79-4017 Galactosyltransferase Activities in Subcellular Fractions of Two YC8 Lymphoma Cell Variants: Does a Relationship Exist Between Antigenic Determinants and Acceptor Sites for Galactose?** (Eng) Barel, M. (Institut d'Immuno-Biologie, Hopital Broussais, 96 rue Didot, 75674 Paris Cedex 14, France); Dahan, A.; Morel, A. *Biochimie* 60(11/12): 1273-1282; 1978.

The physiological differences between two variants of the YC8 lymphoma cell line (P and L) were correlated with some of their immunological and enzymatic properties. In cytotoxicity tests, L cells are much less sensitive toward antilymphocyte serum and anti-Moloney murine leukemia virus serum. In addition, L cells have lost their specificity toward transplantation into allogeneic mice, and they do not provoke renal metastasis. Among the subcellular fractions of both variants, fraction A showed the highest ouabain-inhibited, Mg²⁺-stimulated, (Na⁺/K⁺)-dependent ATPase and galactosyltransferase (GT) activities. The pH optima and Mn²⁺ requirements for GT activity on endogenous acceptors were the same for both variants, but the apparent Km for uridine diphosphate (UDP)-galactose (Gal) was 1.7 x 10⁻⁶ M for P cells and 3.3 x 10⁻⁶ M for L cells, and the P-cell Vmax < the L-cell Vmax. On exogenous (ovomucoid) acceptors, the Km was 0.61 x 10⁻⁶ M for both variants. These results suggest that there are more endogenous acceptor sites on L cells, with the higher affinity of P cells for UDP-Gal being balanced by less endogenous acceptor sites for Gal. When an exogenous acceptor was added, Gal transfer on endogenous acceptor sites of both cells was negligible. The apparent Km for exogenous acceptors was 8.6 x 10⁻⁵ M for P cells and 4.3 x 10⁻⁵ M for L cells. These data suggest that there is a relationship between antigenic determinants and acceptor sites for Gal. L-cell enzymes are more rapidly saturated than P-cell enzymes because of the higher number of endogenous sites on the L cells. This fact plus other physiological differences may be related to the modification of P cells into L cells. (30 refs)

- 79-4018 'Hybrid Resistance' Against Parental Tumors: One or Several Genetic Patterns?** (Eng) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S-104 01

Stockholm, Sweden); Klein, G. O.; Karre, K.; Kiessling, R. *Immunogenetics* 7(5): 391-404; 1978.

A spectrum of lymphomas, sarcomas, and carcinomas was tested for F₁ hybrid resistance after sc inoculation of small numbers of tumor cells into syngeneic and F₁ hybrid mice. Significant F₁ hybrid resistance was found against all lymphomas and leukemias tested except the Moloney virus-induced YAD lymphoma. Resistance was under the influence of a strong histocompatibility (*H-2*)-linked factor in each case in which linkage was tested by backcross or congenic analysis. YAD showed no hybrid resistance pattern, and it was virtually completely resistant to natural killer cell-mediated lysis. Comparison of several hybrid resistance patterns showed that different *H-2*-linked genetic factors can influence resistance against lymphomas of different origin. The data suggest the existence of a polymorphic system, probably pseudoallelic, rather than a simply allelic system. The S3A carcinomas and the MC57X and MSWBS sarcomas failed to show any *H-2* linkage of hybrid resistance. The A.BY genome, which was unable to introduce resistance against C57BL lymphomas, introduced a relatively strong non-*H-2*-linked resistance against the MC57X sarcoma, C57L, a Moloney virus-induced lymphoma that was also *H-2b* but more closely related to C57BL, was unable to do so. The nature of the non-*H-2*-linked resistance factors that can act against sarcomas and carcinomas, their linkage relationships, and their mechanism of action are unknown. (13 refs)

79-4019 Stewart-Treves Syndrome (Angioplasic Sarcoma in Chronic Lymphedema). (Ger) Kostler, E. (Hautklinik des Bezirkskrankenhauses, Dresden-Friedrichstadt, Friedrichstrasse 41, DDR-801 Dresden, E. Germany); Roitzsch, E.; Kuntze, I. *Dermatol Monatsschr* 164(12): 882-888; 1978.

A 56-yr-old woman underwent radical left mastectomy with postoperative irradiation for breast carcinoma. She developed lymphedema of the left arm about 1 yr later, and a tumor was found in the lymphedema 10 yr after surgery. Histological examination revealed an angioablasic sarcoma with lymphocyte infiltration in the corium and subcutis and with spindle cell areas. The infiltration was bound predominantly to capillaries. (51 refs)

79-4020 Autoantibodies (Cold Lymphocytotoxins, Antiactin Antibodies and Antinuclear Factors) in Nasopharyngeal Carcinoma Patients. (Eng) Lamelin, J. P. (Unit Biological Carcinogenesis, International Agency Res. Cancer, Lyon, France); de-The, G.; Revillard, J. P.; Gabbiani, G. *IARC Sci Publ* 20: 523-536; 1978.

The occurrence of cold lymphocytotoxic antibodies (LTA), antiactin antibodies (SMA), and antinuclear antibodies

(ANA) in sera from patients with nasopharyngeal carcinoma (NPC) was investigated. Ninety-eight sera from NPC patients diagnosed in Hong Kong (43 cases), Tunis (42 cases), and Paris (13 cases), selected for even distribution of the various stages of the disease, were assayed for LTA. The SMA study was limited to 30 of the Tunisian patients, the ANA study to 29 Tunisian patients. All three antibodies were found at higher frequencies in sera from NPC patients than in those from matched controls. The frequency and geometric mean titers (GMT's) of LTA-positive sera varied with the origin of the patient (Chinese > North African > Caucasian), paralleling the risk for NPC in each ethnic group, and the stage of the disease (Stage IV > Stage I). A positive correlation was found between LTA and anti-Epstein-Barr virus (EBV) titers with regard to anti-viral capsid antigen (VCA) and anti-Epstein-Barr nuclear antigen (EBNA) antibodies. SMA were specific to actin, and their titers did not correlate with anti-EBV [VCA, EBNA, and early antigen (EA)] titers. There was a complete lack of correlation between LTA and SMA or ANA. The existence of a link, in a given geographical area, between the incidence of NPC and the proportion of NPC patients with high LTA titers indirectly supports the hypothesis that EBV plays a role in NPC. The origin and biological significance of the autoantibodies remain to be explained. (37 refs)

79-4021 Clinical Evaluation of EBV Serology in American Patients with Nasopharyngeal Carcinoma. (Eng) Pearson, G. R. (Dept. Microbiology, Mayo Clinic/Foundation, Rochester, MN 55901); Coates, H. L.; Neel, H. B.; Levine, P.; Ablashi, D.; Easton, J. *IARC Sci Publ* 20: 439-448; 1978.

The sera of American patients with nasopharyngeal carcinoma (NPC) were examined for the presence of antibodies to Epstein-Barr virus (EBV)-associated antigens to determine whether they might be of clinical importance in the diagnosis and prognosis of NPC. All sera from 69 NPC patients were positive for antibodies to viral capsid antigen (VCA), compared with 76%-90% of sera from 85 patients with other head and neck cancers, 80 patients with lymphoma, and 47 normal subjects. Ninety-seven percent of the sera from NPC patients were positive for antibodies to early antigen (EA), compared with 36%-59% of the sera from the control groups. Seventy-seven percent of the NPC sera were positive for IgA antibodies to VCA, and all of the negative sera were from treated patients with no clinical evidence of disease. Only 6%-10% of the sera from the three control groups were positive for IgA antibodies to VCA. VCA geometric mean titers were approx fourfold lower in patients in remission than in patients with clinical NPC; however, there was a substantial difference in EA and IgA-VCA titers between these two groups. The results suggest that high antibody titers to EBV-induced EA and the presence of antibody to EBV antigens in the IgA fraction were the two most specific discriminating parameters in NPC, although neither was infallible. (17 refs)

- 79-4022** Presence of Epstein-Barr Virus Specific IgA in Saliva of Nasopharyngeal Carcinoma Patients: Their Activity, Origin and Possible Clinical Value. (Eng) Desgranges, C. (Unit Biological Carcinogenesis, International Agency Res. Cancer, Lyon, France); De-The, G. *IARC Sci Publ* 20: 459-469; 1978.

The secretory nature, origin, and clinical significance of Epstein-Barr virus (EBV)-specific IgA serum antibodies in the saliva of patients with nasopharyngeal carcinoma (NPC) were investigated. Saliva specimens were collected from 10 NPC patients in Tunisia, 13 NPC patients in Hong Kong, 21 East African patients with Burkitt's lymphoma, 18 French patients with infectious mononucleosis, patients with other head and neck cancers, and controls. Of the 23 NPC salivas, 14 were positive for EBV-specific IgA(α). Of 12 NPC salivas positive for EBV IgA(α), 9 were also positive for the secretory portion (SP) of IgA, as determined by immunofluorescence using a fluorescein-labeled anti-human IgA specific for α chains or anti-human SP of IgA. EBV IgA(α)-negative salivas were also negative for the SP. Immunofluorescence examination of NPC biopsies demonstrated that the origin of EBV-specific IgA was the tumor tissue itself. Fifty-four percent of the throat washings from NPC patients contained IgA directed to EBV viral capsid antigen (VCA) and 27% contained IgA directed to early antigen (EA). Seventy-three percent and 54% of the throat washings contained IgG directed to VCA and EA, respectively. Throat washings from non-NPC patients and controls were devoid of detectable IgA antibodies, but some were positive for IgG. Sera from patients with Burkitt's lymphoma and other head and neck cancers contained IgA VCA at titers ranging from 1/10 to 1/40, whereas 76% of the NPC sera showed high titers (1/40 to <1,280). Testing for EBV-specific IgA could be used as a screening tool for NPC in high-risk areas. (5 refs)

- 79-4023** Epstein-Barr Virus-related Serology in Nasopharyngeal Carcinoma and Controls. (Eng) Henle, W. (Div. Virology, Joseph Stokes, Jr., Res. Inst., Children's Hosp., Philadelphia, PA 19104); Henle, G.; Ho, J. H. *IARC Sci Publ* 20: 427-437; 1978.

Studies of antibodies (ab's) to Epstein-Barr virus (EBV)-

related antigens in nasopharyngeal cancer (NPC) patients are reviewed. The geometric mean titers (GMT's) of IgG ab's to viral capsid antigen (VCA) and to the diffuse component of early antigen (EA-D) increase strikingly with the stage of the disease (Stage V > Stage I). The GMT's of ab's to Epstein Barr nuclear antigen (EBNA) increase in parallel with anti-VCA titers in the early stages of NPC, but level off in later stages. Before initiation of therapy, nearly all patients show IgA ab's to VCA, and many also to EA-D, at titers that occasionally match those of the corresponding IgG ab's. The incidence and GMT's of IgA ab's also increase with stage of the disease. VCA-specific IgM ab's have been detected in a small proportion of patients with Hodgkin's disease and in a few NPC patients. The VCA- and D-specific IgG ab titers were compared in two groups of patients: untreated patients and long-term (≥ 5 yr) survivors. The GMT's of anti-VCA and anti-D in the untreated patients showed the expected stepwise increases from Stage I to Stages IV or V, but the long-term survivors showed a downward trend in ab titers. Reversals from a downward to an upward trend could occur after several years, and they could occur months in advance of the detection of recurrent tumors or metastases. Close serological monitoring of NPC patients at frequent intervals is recommended. (29 refs)

See also:

- * (Rev.): 79-3614, 79-3632, 79-3633, 79-3635, 79-3637.
- * (Chem.): 79-3705, 79-3747, 79-3753.
- * (Phys.): 79-3768, 79-3772.
- * (Viral): 79-3798, 79-3806, 79-3807, 79-3811, 79-3823, 79-3827, 79-3828, 79-3834, 79-3835, 79-3843, 79-3844, 79-3864, 79-3891, 79-3892, 79-3915, 79-3928, 79-3933, 79-3934, 79-3938, 79-3940, 79-3949, 79-3950, 79-3952, 79-3954, 79-3957, 79-3962, 79-3964, 79-3965, 79-3966, 79-3974, 79-3975, 79-3977, 79-3978.
- * (Path.): 79-4026, 79-4044, 79-4047, 79-4067, 79-4071, 79-4072, 79-4085, 79-4098.
- * (Epid.-Biom.): 79-4128, 79-4129, 79-4148, 79-4152.

PATHOGENESIS

- 79-4024 A Diagnosis of Pleural Mesothelioma.** (Eng) Caravelli, J. F. (Dept. Diagnostic Radiology, Sloan-Kettering Memorial Cancer Center, New York, NY); Zaman, M. B. *Clin Bull* 8(4): 161-163; 1978.

The case report of a 58-yr-old woman with pleural mesothelioma is presented. The evaluation of this patient was difficult because of a childhood history of 'pleurisy,' a negative biopsy, and apparent symptomatic improvement following treatment with antituberculosis drugs. The development of a lobulated pleural thickening on a radiograph suggested a malignant process, which was confirmed on needle aspiration. (5 refs)

- 79-4025 Clinical and Radiographic Signs in Primary and Metastatic Esophageal Neoplasms of the Dog.** (Eng) Ridgway, R. L. (Dept. Medicine, Sch. Veterinary Medicine, Univ. California, Davis, CA 95616); Suter, P. F. *J Am Vet Med Assoc* 174(7): 700-704; 1979.

The clinical and radiographic evaluation of 2 primary and 6 metastatic esophageal neoplasms diagnosed in 8/49,229 dogs seen over the last 11 yr at a veterinary medicine teaching hospital is reviewed. The two primary esophageal tumors were a leiomyoma and a squamous cell carcinoma. The six invasive tumors originated from the thyroid gland (3 dogs), respiratory tract (2), and stomach (1). The most common clinical signs were regurgitation, dysphagia, wt loss, development of neck masses, and respiratory difficulties. It was concluded that the clinical signs often can be misleading. The interpretation of survey radiographs, barium contrast studies, or fluoroscopic studies often provides the initial data base. The final diagnosis requires histologic examination. Retention of air in the esophagus (with or without esophageal displacement) and motor dysfunction (with or without gross morphologic changes) are the most important criteria for radiographic diagnosis. (12 refs)

- 79-4026 Gastric Morphology, Function, and Immunology in First-Degree Relatives of Proband with Pernicious Anemia and Controls.** (Eng) Varis, K. (Gastroenterological Unit, Second Dept. Medicine, Meilahti Hosp., Haartmaninkatu 4, 00290 Helsinki 29, Finland); Ihmaki, T.; Harkonen, M.; Samloff, I. M.; Siurala, M. *Scand J Gastroenterol* 14(2): 129-139; 1979.

Morphologic, functional, and immunologic observations of 183 first-degree relatives of 68 patients with pernicious anemia indicated that one population of relatives had a

high and the other little or no proneness to severe atrophic gastritis of the body of the stomach (AGB). This bimodal distribution suggests the participation of a single major factor, probably genetic, in the pathogenesis of severe AGB in relatives of patients with pernicious anemia. (35 refs)

- 79-4027 Vascular Leiomyoblastoma of the Stomach. Observations Supporting its Origin from Zimmermann's Pericyte.** (Eng) Eimoto, T. (Clinical Lab., Div. Gastroenterology, Koshigaya City Hosp., 95 Higashikobayashi, Koshigaya City, Saitama-ken, 343 Japan); Miyake, M.; Sasaki, T. *Acta Pathol Jpn* 29(2): 277-288; 1979.

A gastric leiomyoblastoma and a small mesenteric metastasis in a 44-yr-old Japanese man were studied histologically. The primary tumor parenchyma was moderately cellular, and it consisted of large polygonal cells mixed with spindle cells. In small foci there were tumor cells with marked vacuolization and peripheral nuclei masquerading as signet ring cells. The tumor cells were closely associated with numerous small vessels, mostly capillaries. Chronic inflammatory cells were scattered throughout the tumor. Interwoven cytoplasmic processes were prominent, and many of the small vessels were intimately surrounded by these processes. The small mesenteric nodule contained a tumor with a myxoid stroma and, similar to the primary, abundant hyaluronic acid. The tumor cells were bizarre and hyperchromatic and their cytoplasm was faintly positive for myofibrils; there were multinucleate giant cells and many vacuolated cells. The stroma was also rich in blood vessels. The observations support the view that some leiomyoblastomas originate from the pericyte of Zimmermann. Leiomyoblastoma may be placed between hemangiopericytoma and glomus tumor in the spectrum of pericytic tumors. (34 refs)

- 79-4028 Comparative Studies on the Histology of Gastric Carcinoma in the Polish and Japanese Populations.** (Eng) Matusik, J. (Dept. Pathomorphology, Inst. Pathology, Medical Acad., Cracow, Poland). *Patol Pol* 29(3): 289-302; 1978.

The distribution of gastric carcinoma was compared among 676 inhabitants of southeastern Poland and 752 inhabitants of Kyushu Island, Japan. There was no significant difference in mean age between the Japanese and Polish male patients, but the Japanese women were younger than the Polish women. The intestinal type of car-

cinoma was more common than the diffuse type among men and women of both nationalities, the ratio of intestinal to diffuse types being greater in the Polish women than in the Japanese women. The diffuse type was more common in patients <50 yr old, the intestinal type in older patients. The mortality rate was higher among patients <50 yr old with the diffuse type of carcinoma and in older patients with the intestinal type. Standardized death rates were nearly twofold higher among the Japanese, the difference being due primarily to the greater number of intestinal-type tumors among the Poles. (47 refs)

- 79-4029 **Periampullary Carcinoma.** (Eng) Oehler, J. R. (Dept. Surgery, Univ. Michigan Medical Center, Ann Arbor, MI); Turcotte, J. G. *New Physician* 28(3): 40-42; 1979.

A 64-yr-old woman was diagnosed with a well-differentiated papillary adenocarcinoma involving the duodenum and the head of the pancreas. The symptoms included wt loss, vomiting, pruritis, and acholic stools, later followed by anemia and guaiac-positive stools. The extent of the neoplasm was demonstrated by gastroscopy, computerized axial tomography, ultrasound, iv cholangiography, and endoscopic retrograde cannulation of the pancreatic duct. (6 refs)

- 79-4030 **Relation of Pancreatic Duct Hyperplasia to Carcinoma.** (Eng) Kozuka, S. (2nd Dept. Pathology, Nagoya Univ. Sch. Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466, Japan); Sassa, R.; Taki, T.; Masamoto, K.; Nagasawa, S.; Saga, S.; Hasegawa, K.; Takeuchi, M. *Cancer* 43(4): 1418-1428; 1979.

To determine the relationship between pancreatic duct hyperplasia (PDH) and carcinoma of the pancreas, pancreases from 1,174 autopsy cases were examined histologically and histochemically. PDH was characterized by the presence of large epithelial cells, and it was found in ducts throughout the pancreas. Papillary proliferation of tall columnar epithelium was usually seen in organs affected by nonpapillary hyperplasia. Atypical hyperplasia was characterized by papillary projections of epithelium lacking a fibrous core. It was occasionally seen in organs with papillary and nonpapillary hyperplasia. Transition of nonpapillary into papillary hyperplasia was observed frequently, and transition between papillary and atypical hyperplasia was seen occasionally. All infiltrating carcinomas but one were of ductal origin, and all types of PDH, especially atypical hyperplasia, were significantly more common in organs with infiltrating carcinoma than in those without it. The amount of mucus gradually decreased in the order nonpapillary, papillary, and atypical hyperplasia, and carcinoma. The mucus was primarily neutral mucin or sialomucin, compared with sulfated

mucin in normal epithelium. The mucous-histochemical changes were associated with enlargement of the epithelial cells. The incidence of PDH increased steadily from 0% in patients under 19 yr to 54.5% in patients aged 80 yr or more. The incidence was 19.9% among men and 16.8% among women. The data suggest a sequential or multistep evolution of pancreatic carcinoma. (12 refs)

- 79-4031 **Cancer of the Pancreas in Young Adults.** (Eng) Kune, G. A. (Univ. Dept. Surgery, Repatriation General Hosp., Heidelberg, Victoria 3081, Australia); Hobbs, J. B.; Butterfield, D.; Sali, A. *Med J Aust* 65(2): 626-628; 1978.

The case reports of two women, aged 24 and 25 yr, with pancreatic cancer (PC) are presented. One presented with a 4-wk history of intermittent severe upper abdominal pain, increasing jaundice, dark urine, and pale stools, and the other with a 6-wk history of increasing jaundice and a mild ache under the right costal margin. There was no history of drug intake, contact with hepatitis, or of high alcohol intake. Literature data suggest that the incidence of PC is increasing in Western populations and that there is an increased number of young adults with this cancer. The use of cigarettes, certain types of diet, and psychosocial stresses of the Western lifestyle have been implicated. (21 refs)

- 79-4032 **Primary Lymphomas of the Small Intestine in Iraq: A Pathological Study of 145 Cases.** (Eng) Al-Saleem, T. (Dept. of Pathology, Coll. Medicine, Baghdad, Iraq); Zardawi, I. M. *Histopathology* 3(2): 89-106; 1979.

The histopathology of 145 malignant lymphomas of the small intestine in Iraq was studied, and the results were compared with clinical and immunological findings. The most common pathology was an intense mucosal lymphoplasmacytic proliferation that effaced the villi and crypts partially or completely. The proliferation was composed of mature plasma cells and limited to the lamina propria or it was associated with a full-blown lymphoplasmacytic lymphoma, almost always of the upper small intestine. The syndrome presented as abdominal pain, chronic diarrhea, and, sometimes, the serological demonstration of α heavy chains. Other types of lymphomas were associated with nonspecific mucosal inflammation or follicular lymphoid hyperplasia. They were lymphocytic, plasmacytic, or lymphoblastic with a 'starry sky' histiocytic reaction and representing distinct clinicopathological entities unrelated to α heavy chain disease. There was only one case of Hodgkin's disease in this series. (21 refs)

- 79-4033 Mucosal Hyperplasia in Colonic Diverticula.** (Eng) Rhatigan, R. M. (Univ. Hosp. Jacksonville, 655 W. Eighth St., Jacksonville, FL 32209); Saffos, R. O. *Histopathology* 3(2): 153-160; 1979.

Eight colectomy specimens resected for diverticulosis with or without diverticulitis, a disease generally accepted as not causally related to malignancy, were examined for non-polypoid mucosal hyperplasia (NPMH), which has consistently been observed adjacent to colonic carcinomas. None of the patients (5 men, 3 women 51-76 yr old) had any signs of neoplasm. Five patients had a total colectomy, one had a subtotal colectomy, and two had left hemicolectomies. A few small hyperplastic and adenomatous polyps were present in several of the specimens, but no other abnormalities were found. Significant microscopic changes were not noted in any section of the grossly normal bowel. NPMH was seen frequently within the diverticula in five specimens. In all five, the diverticula showing the mucosal change were in the descending or sigmoid colon, and the bowel wall was thickened due to hypertrophy of the muscle layer and/or inflammatory changes. In one case, mild NPMH was noted adjacent to diverticula. These findings suggest that NPMH is nonspecific, having no particular relationship to the development of malignancy. (24 refs)

- 79-4034 Hereditary Colon Cancer: Family Studies (Meeting Abstract).** (Eng) Lynch, H. T. (Creighton Univ., Omaha, NE 68131); Lynch, P. M.; Go, R. C.; Lynch, J. F.; Elston, R. C.; Follett, K. L.; Fain, P. *Proc Am Assoc Cancer Res* 20: 108; 1979 (2 refs)

- 79-4035 Isolation and Use of Phenotypically Different Subpopulations from a Mammary Cell Line to Study Metastasis (Meeting Abstract).** (Eng) Danielson, K. G. (Dept. Zoology, Washington State Univ., Pullman, WA 99164); Anderson, L. W.; Hosick, H. L. *In Vitro* 15(3): 175; 1979 (no refs)

- 79-4036 Adenocarcinoma of the Colon in an Adolescent with the Family Cancer Syndrome.** (Eng) Aiges, H. W. (Div. Pediatric Gastroenterology, Dept. Pediatrics, North Shore Univ. Hosp., 300 Community Drive, Manhasset, NY 11030); Kahn, E.; Silverberg, M.; Daum, F. *J Pediatr* 94(4): 632-633; 1979.

The case report of a 14-yr-old girl with colonic adenocarcinoma is presented. The patient had originally presented at age 10 with bright red rectal bleeding; barium enema demonstrated a polypoid lesion in the rectosigmoid area, but proctosigmoidoscopy was not performed. On second presentation with severe constipation, tenesmus, and hematochezia, biopsies revealed a mucinous invasive

adenocarcinoma. Both the patient's father and a paternal uncle had colonic adenocarcinoma. (10 refs)

- 79-4037 Localized Scleroderma as a Symptom of Rectal Cancer.** (Fre) Joliot, A. Y. (Service de Medecine Interne, Hopital des Charpennes, F 69603 Villeurbanne Cedex, France); Vittori, F.; Allard, C.; Levrat, R. *Nouv Presse Med* 8(9): 703; 1979.

Localized scleroderma of the legs was one of the symptoms of a differentiated rectal adenocarcinoma in a 65-yr-old man. Spontaneous regression of the scleroderma occurred about 2 mo after radical tumor resection. (5 refs)

- 79-4038 Angioimmunoblastic Lymphadenopathy: Termination as Diffuse Lymphosarcoma with Plasmacytoid Features.** (Eng) Fayemi, A. O. (Dept. Pathology, Holy Name Hosp., Teaneck, NJ 07666); Ali, M.; Braun, E. V.; DeCecio, T. *Mt Sinai J Med (NY)* 46(1): 39-43; 1979.

The evolution of angioimmunoblastic lymphadenopathy (AILD) into a diffuse lymphoplasmacytic proliferation disease indistinguishable from a malignant lymphoma is reported. The patient, a 77-yr-old woman, initially responded to corticosteroid treatment with temporary remission of her symptoms. The disease progressed rapidly, however, and death occurred about 3 mo after diagnosis. Massive generalized lymph node enlargement; nodular infiltration of the liver; infiltration of the vessels, bronchi, and pulmonary alveolar septa; destruction of lymph node architecture with extensive perinodal invasion; and focal infiltration of the spleen, skin, and kidneys by mature and immature lymphoid and plasmacytic cells were found at autopsy. Of 118 patients with AILD reported in the English literature, only 53 were alive after follow-up periods of 1 mo to 9 yr. Autopsies of 36 patients revealed multiple organ involvement by an infiltrate composed of plasmacytoid and lymphoid cells with scattered eosinophils. AILD and immunoblastic sarcoma may belong to a spectrum of neoplastic lymphoreticular lesions, the former appearing morphologically more benign than the latter. (25 refs)

- 79-4039 Establishment and Characterization of a New Cell Line (NRC-12) Derived from a Human Renal Cell Carcinoma.** (Jpn) Komatsubara, S. (Dept. Urology, Niigata Univ. Sch. Medicine, Niigata, Japan). *Jpn J Urol* 69(12): 1535-1542; 1978.

A new cell line (NRC-12) was derived from a 50-yr-old man with a clear cell-type renal cell carcinoma. NRC-12 has been maintained for 2.5 yr and for >140 in vitro passages.

Growth of these epithelial cells was characterized by an absence of contact inhibition and a 48-hr doubling time. Macroscopically, large, polymorphous cells in a stone wall arrangement were observed in generation 98, and glandular structures and huge polynuclear cells were seen in generation 6. Electron microscopy showed disorderly, small nuclei and chromosomes distributed on one side of the cells and prominent microvilli resembling the brush border-like structures of renal cell carcinoma on the opposite side. Chromosome analysis revealed the presence of hypodiploid and hypotetraploid distributions and a secondary constriction of chromosome A3 in all cells. When golden hamsters were inoculated in the cheek pouch with G88 stemline cells, tumors were formed that contained cells identical to those of the original tumor. (32 refs)

- 79-4040 Ureterosigmoidostomy in Rats: A Model for the Study of Bladder Tumour Carcinogenesis and Cocarcinogenesis.** (Eng) Rowland, R. G. (Dept. Urology, Northwestern Univ. Medical Sch., 1100 W. Michigan Ave., Indianapolis, IN 46223); Grayhack, J. T.; Oyasu, R. *Urol Res* 7(1): 23-26; 1979.

A modified Coffey I ureterosigmoidostomy in rats was developed as a model of urinary diversion for studying bladder carcinogenesis and the cocarcinogenic role of urine. Diverted and sham-operated animals were killed at 1, 3 and 6 mo. Excretory urograms revealed minimal hydronephrosis in most diverted animals. Upper tract bacterial colonization was 9 times more frequent in diverted animals. Approx one-third of the diverted animals had focal cortical scarring; however, renal function was normal in all groups, as assessed by serum creatinine and electrolytes. These studies indicate that ureterosigmoidostomy in rats is a satisfactory model of urinary diversion for studying bladder carcinogenesis. (15 refs)

- 79-4041 Thrombocytopathy in Preleukemia: Association with a Defect of Thromboxane A₂ Activity.** (Eng) Russell, N. H. (Dept. Haematology, Nuffield Unit Medical Genetics, Univ. Liverpool, Liverpool, England); Keenan, J. P.; Bellingham, A. J. *Br J Haematol* 41(3): 417-425; 1979.

Platelet aggregation and the platelet prostaglandin pathway were studied in two preleukemic patients with hemorrhagic tendencies but normal platelet counts. In both patients there was no secondary aggregation with ADP, no aggregation with collagen, and little or no aggregation with arachidonic acid (AA). The release of nucleotides in response to thrombin was diminished. The production of malonyldialdehyde in response to AA was normal in both cases, excluding a cyclooxygenase deficiency. The platelet-aggregating activity and rabbit aorta-contracting activity of

thromboxane A₂ (TxA₂) were low in both patients, but production of thromboxane B₂ from exogenous AA was normal. The abnormalities in platelet function in these patients appear to be due to the production of TxA₂ with a low biological activity. (29 refs)

- 79-4042 Medullary Sponge Kidney and Renal-Leak Hypercalciuria: A Link to the Development of Parathyroid Adenoma?** (Eng) Diabal, P. W. (Endocrine Service, Wilford Hall USAF Medical Center, Lackland AFB, San Antonio, TX 78236); Jordan, R. M.; Dorfman, S. G. *JAMA* 24(14): 1490-1491; 1979.

The case of a patient (20-yr-old man) with medullary sponge kidney and idiopathic hypercalciuria of the renal-leak type is presented. The patient had nephrocalcinosis, intermittent hypercalciuria, hyperparathyroidism, and normocalcemia. The hyperparathyroidism was resolved, with diminution in renal calcium excretion, during hydrochlorothiazide treatment. This indicates that the hyperparathyroidism was secondary to renal calcium wasting. Such persistent stimulation of the parathyroid glands could lead to unsuppressible hyperparathyroidism. It is hypothesized that parathyroid adenoma could develop in some patients with medullary sponge kidney after a long period of secondary hyperparathyroidism due to renal calcium wasting. Thus, many cases of associated medullary sponge kidney and parathyroid adenoma may be a consequence of disordered renal calcium excretion rather than a primary abnormality. (7 refs)

- 79-4043 Classification of Mouse Mammary Tumors in Dunn's Miscellaneous Group Including Recently Reported Types.** (Eng) Sass, B. (Registry Experimental Cancers, Div. Cancer Cause and Prevention, NCI, NIH, Bethesda, MD 20014); Dunn, T. B. *J Natl Cancer Inst* 62(5): 1287-1293; 1979.

Some of the mouse mammary tumor types in Dunn's miscellaneous category are subclassified, and other unusual types reported in the literature after the publication of Dunn's classification are reviewed. Adenocarcinoma type Y, first reported in (C3H x Y)F₁ mice, is characterized by tubules that branch at acute angles and are lined by cuboidal epithelium. Adenocarcinoma type L is characterized by a lacelike appearance and has projections of tumor cells with cytoplasmic vacuoles into lumina. Undifferentiated carcinomas in strains SWR/J and BALB/c contain sheetlike masses of epithelial cells that have very little tendency to form glands but may contain foci of cells with keratohyaline granules. Pale cell carcinomas, which occur in strain GR mice, are hormonally responsive and are characterized by the presence of pale cells and cystic spaces lined by basophilic cells. Foci of cells with acidophilic hyaline bodies and keratohyaline granules and with in-

tercellular bridges distinguish this neoplasm from adenocarcinoma type B. A second hormonally responsive mammary tumor in strain GR mice is adenocarcinoma type P, which consists mainly of acini lined by a single layer of polyhedral cells. Carcinosarcomas of BALB/c mice have a myxoid stroma and also containing glandlike structures; they resemble mixed mammary tumors of the bitch. Criteria for differentiating trichoepitheliomas from mammary tumors are presented. Neoplasms originating in hair follicles can resemble mammary tumors and are confusing because they have a tubular type of structure. (11 refs)

- 79-4044 Interactions of Normal and Neoplastic Parenchymal Cells During Metastasis of Mammary Carcinomas (Meeting Abstract).** (Eng) Slemmer, G. (Dept. Pathology, Univ. British Columbia, Vancouver, B.C., Canada). *Proc Am Assoc Cancer Res* 20: 163; 1979 (no refs)

- 79-4045 Ultrastructure of Myoepithelial Cells in the Development of Breast Cancer.** (Rus) Gachechiladze, I. A. (A. N. Natishvili Inst. Experimental Morphology, Tbilisi, USSR). *Soobshch Akad Nauk Gruz SSR* 92(2): 473-476; 1978.

To evaluate the role of myoepithelial cells in the histogenesis of the breast cancer, mammary gland specimens from 15 patients with benign dysplasias and 17 patients with breast cancer were examined electron microscopically. The characteristic feature of lobular mastopathy was a significantly increased number of myoepithelial cells (the myoepithelial:epithelial cell ratio was 1:2 to 1:1, compared with 1:6 in normal tissue). In regions of nonproliferative mastopathy, the myoepithelial cells remained in the characteristic basal location. In regions of proliferative mastopathy, the myoepithelial cells showed transition from a regular tangential to an irregular radial position. Analysis of various histological variants of lobular carcinoma revealed the presence of different cell types. In scirrhous carcinoma, cells that retained the typical ultrastructural organization of myoepithelial cells were dominant. (4 refs)

- 79-4046 Breast Fibroadenoma and Cardiac Anomaly Associated with EMG (Beckwith-Wiedemann) Syndrome.** (Eng) Raine, P. A. (Royal Hosp. Sick Children, Yorkhill, Glasgow, Scotland); Noblett, H. R.; Houghton-Allen, B. W.; Campbell, P. E. *J Pediatr* 94(4): 633-634; 1979.

A case of EMG (exomphalos, macroglossia, and gigantism: Beckwith-Wiedemann) syndrome associated with a rare occurrence of fibroadenoma of the breast, atrial septal

defect, pulmonary artery stenosis, and an unusual iv pyelogram occurred in a female infant. The benign fibroadenoma appeared at 7 mo, but all the other defects were noted at birth. An iv pyelogram showed enlargement of the right kidney with a mildly unusual pattern of both calyces. The infundibula appeared wide and short, and there was opacification of areas of the renal medulla. At age 17 mo, a recurrent breast nodule (a juvenile intracanalicular fibroadenoma) was excised. (4 refs)

- 79-4047 High Incidence of Mammary Tumors in Mice with Inherited Asplenia Carriers for the Nude Gene.** (Eng) Lozzio, B. B. (Dept. Medical Biology, Center Health Sciences, Univ. Tennessee Memorial Res. Center, Knoxville, TN 37920); Lopez, D. M.; Coulson, P.; Lair, S. V. *Cancer Res* 39(5): 1529-1533; 1979.

The occurrence of spontaneous mammary tumors (SMT) in an immunodeficient mouse model useful in elucidation of the role of genetic, viral, immunological, and hormone interactions in the development of mammary carcinomas is reported. A colony of mice suffering from dominant hemimelia associated with agenesis of the spleen has been developed and characterized during the past 7 yr. The hereditarily asplenic (*Dh/+*) mice have a very low incidence (9%) of SMT. Asplenic (*Dh/+*) females were mated with mice homozygous (*nu/nu*) for hereditary athymia (nude) having a BALB/c background. BALB/c females heterozygous for the *nu* gene and with spleen (*nu/+*, *+/+*) have a moderate incidence (12%) of SMT, whereas *nu/+*, *Dh/+* breeders have a drastic increase in the incidence of SMT to 46% when bred under identical conditions. Since all parent strains have a very low incidence of SMT, it appears that the spleen agenesis is a major factor accounting for an earlier and higher incidence of SMT in hereditarily asplenic (*nu/+*, *Dh/+*) mice than in normal (*nu/+*, *+/+*) siblings. The SMT express mammary tumor virus antigen(s) and possess estrogen, progesterone, and glucocorticoid receptors. The SMT rapidly metastasize and kill the host within 30-45 days. The BALB/c asplenic mice with SMT represent a unique model relevant to human breast cancer and for study of the function of the spleen in the development of solid tumors in general and of SMT in particular. (34 refs)

- 79-4048 Preliminary Notes on a Case of Male XX Chromosomes Associated with Breast Adenocarcinoma.** (Ita) Giammarini-Barsanti, A. (I Divisione di Medicina, Ospedali Riuniti di Trieste, Trieste, Italy); Zennaro, W. *Minerva Med* 70(9): 679-684; 1979.

Gynecomasty was diagnosed in a 66-yr-old man who had undergone a left mastectomy for breast adenocarcinoma 5 yr earlier. He was born with hypospadias, and hypogonadism was seen. His psychosexual behavior was

masculine. Examination of testicular biopsy samples revealed tubular fibrosis and germinal aplasia. Follicle-stimulating hormone, luteinizing hormone, testosterone, and 17-ketosteroid levels were close to those seen normally in women. The XX chromosomes were found in peripheral blood lymphocytes, and the sex chromatin test was always positive. Statistical evidence is cited for the proposition that breast tumors have an underlying genetic and endocrine component in their etiology. (23 refs)

- 79-4049 Breast Parenchymal Patterns: Prevalent and Incident Carcinomas.** (Eng) Wolfe, J. N. (Dept. Radiology, Hutzel Hosp., Detroit, MI 48201). *Radiology* 131(1): 267-268; 1979.

Mammographic parenchymal patterns in breasts in which cancer was apparent on first examination were compared with those in breast cancers that developed after a first negative observation. In a consecutive series (prevalent breast cancer), every breast biopsy done at one hospital over a 2-yr period was reviewed. The other series (incident breast cancer) comprised patients in whom no suggestion of cancer was found radiographically. In the 2-yr consecutive series, 139 new breast cancers were histologically proved. The incident (interval) series consisted of 169 cases. The two groups were divided into low- and high-risk categories. The low-risk group exhibited the N1-P1 parenchymal patterns and the high-risk group exhibited the P2-DY patterns. In the prevalent breast cancer series, 30% of the cases were in the N1-P1 groups, vs 8% in the incident series. The interval series had a lower rate of positive axillary nodes at the time of surgery than the consecutive series. The number of women having more than one metastatic lymph node was significantly higher in the consecutive series than in the interval one. In a discussion of the difference between prevalent and incident breast cancers, it is proposed that examination of 100,000 women on day 1 would reveal perhaps 10 carcinomas/1,000. This would reflect breast cancer prevalence as limited by the ability to diagnose it. If these same women were reexamined 1 yr later, 5 carcinomas/1,000 would be discovered. Finally, a plateau would be reached where essentially the same number of cancers would be found at yearly intervals, ie, 2 cases/1,000. This would be a reflection of breast cancer incidence. In a purely incident series, there should be a gradual decrease in the number of N1-P1 cancer cases and a corresponding increase in the number of P2-DY cases. (10 refs)

- 79-4050 Immunocytochemical Localization of Calcitonin-producing Cells in a Strumal Carcinoid with Amyloid Stroma.** (Eng) Dayal, Y. (Dept. Pathology, New England Medical Center Hosp., 171 Harrison Ave., Boston, MA 02111); Tashjian, A. H.; Wolfe, H. J. *Cancer* 43(4): 1331-1338; 1979.

A nonfunctioning strumal carcinoid arising in a 48-yr-old Caucasian woman was studied histochemically and immunocytochemically. At surgery, the uterus was found to contain two subserosal leiomyomas, the left ovary a hemorrhagic corpus luteum; the right ovary was enlarged and showed a small cyst along one pole. The right ovary was almost completely replaced by a tumor that had a variegated histologic pattern of cells arranged in solid islands, trabeculae, small glands, acini, and folliclelike structures dispersed in a fibrovascular stroma. The tumor cells were all intensely argyrophilic, and there were several clusters of calcitonin-containing C cells. The C cells were present as small aggregates of four to eight cells within the connective tissue stroma or interspersed individually, or in clusters of two to four cells between tumor cells forming acinar or follicular structures. There was no evidence of gastrin or insulin localization within the tumor cells. The results support an APUD (ability for Amine Precursor Uptake and Decarboxylation) cell origin for strumal carcinoids. The presence of the calcitonin-producing C cells within the tumor raises interesting possibilities as to whether these lesions are derived from C cells or represent ovarian carcinoids with foci of C cell differentiation. (35 refs)

- 79-4051 Collision Tumors: Papillary Mucinous Cystadenocarcinoma of Ovary and Endometrial Carcinoma.** (Eng) Carideo, C. (Dept. Pathology, Mount Sinai Sch. Medicine, City Univ. New York, One Gustave L. Levy Place, New York, NY 10029); Marchevsky, A. *Mt Sinai J Med (NY)* 46(1): 21-24; 1979.

The case report of a 68-yr-old nulliparous woman in whom a mucinous papillary cystadenocarcinoma of the right ovary collided with an endometrial adenocarcinoma is presented. The literature is reviewed, and the histopathogenesis is discussed. (12 refs)

- 79-4052 Endobronchial Metastasis of Uterine Leiomyosarcoma (Letter to Editor).** (Eng) Giudice, J. C. (Coll. Medicine and Dentistry New Jersey, Camden, NJ); Komansky, H.; Gordon, R. *JAMA* 241(16): 1684; 1979.

The case report of a 56-yr-old woman with an endobronchial metastasis of uterine leiomyosarcoma is presented. The patient developed massive hemoptysis approx 1 yr following a total abdominal hysterectomy for uterine leiomyosarcoma. Fiberoptic bronchoscopy revealed bleeding polypoid lesions in the right lower and middle lobes. The presence of the metastasis was confirmed at autopsy. (1 ref)

- 79-4053 A Case of Papillary Fibroepithelial Proliferation with Focal Atypia Within an Epophoron**

Cyst. (Ger) Cremer, H. (Pathologisches Institut, Universität Bonn, 5300 Bonn, W. Germany). *Geburtshilfe Frauenheilkd* 39(2): 152-156; 1979.

A 47-yr-old woman underwent hysterectomy with right adnexectomy for a uterine fibromyoma. The histological examination revealed an epoophoron cyst characterized by intracystic papillary fibroepithelial proliferation with focal cellular and nuclear atypia. Despite the absence of capsular infiltration and vascular invasion, the cytological picture suggested an incipient malignant transformation (intracystic papillary carcinoma), probably originating from the intraligamentary portion of the Wolffian duct. (6 refs)

79-4054 Isolated Adenocarcinomatous Transformation of Uterine Adenomyosis. (Fre) Baril, A. (Service d'Anatomie Pathologique, CHU Bellevue, boulevard Pasteur, 42023 Saint-Etienne Cedex, France); Boucheron, S.; La Selve, A. *Sem Hop Paris* 55(5/6): 301-304; 1979.

A uterine fibromyoma was diagnosed in a 48-yr-old woman. Surgery performed about 4 yr later revealed adenomyosis with subserous and interstitial myomas and endometrial adenocarcinomas infiltrating the fibromyomatous tissue. The endometrium, ovaries, and fallopian tubes were intact. Corpus luteum was found in one ovary. The histological findings indicated that there was a progressive transformation of the adenomyosis into adenocarcinoma and that the adenomyosis had initially developed from atypical hyperplasia. An initial disequilibrium in the hormone receptivity between the different epithelial portions of the uterus may have played a role in the carcinogenesis. (31 refs)

79-4055 Local Reaction of Connective Tissue in Cervical Carcinoma. (Rus) Shenderova, T. S. (Dept. Pathomorphology, Moldavian Res. Inst. Oncology, Kishinev, USSR). *Vopr Onkol* 25(3): 9-14; 1979.

The results of histochemical examination of specimens of the uterus and its adnexa removed at surgery from 100 cervical carcinoma patients are presented. The group included 11 patients with carcinoma in situ, 72 with Stage I carcinoma, 6 with Stage II, and 11 with Stage III. Histologically, 9 patients had preinvasive carcinoma, 11 had microcarcinoma, 76 had invasive squamous cell carcinoma, and 4 had glandular carcinoma. Characteristic features of the local reaction of the connective tissue of the cervix uteri included focal and diffuse lymphohistiocytic infiltration, proliferation of fibroblasts, and stromal infiltration. The intensity of infiltration showed a progressive increase from the patients with carcinoma in situ to those with invasive carcinoma. The infiltration was interpreted as a manifestation of local immunity caused by alteration of the antigenic properties of the epithelium during malignant transformation. (12 refs)

79-4056 Carcinoma of the Apocrine Vulvar Glands with Invasive Paget's Disease of the Vulva. (Ger) Heberling, D. (Abteilung für gynäkologische Morphologie, Univ. Frauenklinik, 6900 Heidelberg, W. Germany); Dohnert, G.; Rummel, H. H. *Geburtshilfe Frauenheilkd* 39(2): 101-105; 1979.

Invasive intraepithelial Paget's disease of the vulva was diagnosed in a 71-yr-old woman. The histological examination performed after radical vulvectomy confirmed the Paget's disease and revealed a carcinoma of the vulvar apocrine glands. The carcinoma consisted of atypical Paget cells with light, vacuolized cytoplasm and atypical mitoses. The underlying cause of the difference between Paget's disease of the breast, in which carcinoma is always present, and extramammary Paget's disease, in which carcinoma is found in only 25% of cases, is still unknown. (19 refs)

79-4057 Immunohistological Demonstration of Prostatic Origin of Malignant Neoplasms (Letter to Editor). (Eng) Nadji, M. (Dept. Pathology, Univ. Miami Sch. Medicine, Miami, FL 33101); Tabei, Z.; Castro, A.; Morales, A. R. *Lancet* 1(8117): 671-672; 1979.

Of 26 formalin-fixed, paraffin-embedded surgical specimens of primary and metastatic prostatic carcinoma, all stained positively for prostate-specific acid phosphatase (PSAP), whereas none of 34 specimens of nonprostatic primary and metastatic tumors stained positively for this enzyme. Immunohistological demonstration of PSAP may prove invaluable in the management of patients with prostatic carcinoma, especially when the serum assay for PSAP is inconclusive. (1 ref)

79-4058 Malignant Paraganglioma of the Prostate and Retroperitoneum. (Eng) Mehta, M. (Kaiser Permanente Medical Center, 9400 E. Rosecrans Ave., Bellflower, CA 90706); Nadel, N.; Lonni, Y.; Ali, I. *J Urol* 121(3): 376-378; 1979.

A case of malignant paraganglioma of the prostate, metastatic to the retroperitoneal area, is reported. The patient, a 29-yr-old man, had edema of the entire right leg and asymmetric prostatic enlargement on the right side. Excretory urogram, cystoscopy, and a cystogram demonstrated a pelvic mass displacing the bladder. It is theorized that a ganglion or a neural crest derivative of the caudal parasympathetic-aortic chain underwent malignant degeneration. (3 refs)

79-4059 Consumption Coagulopathy in Adenocarcinomas. Report of Seven Cases. (Fre) Hauteville, D. (Clinique Medicale, Hopital d'Instruction

des Armees Sainte-Anne, 83800 Toulon Naval, France); Georges, A.; Chamfeuil, R.; Desbaumes, J.; Abgrall, J.; Flechaire, A.; Herne, N. *Sem Hop Paris* 55(9/10): 442-448; 1979.

Disseminated intravascular coagulation (DIC) was found in seven patients (aged 42-84 yr) with adenocarcinoma of the prostate (2 cases), pancreas (3 cases), or of undetermined origin (2 cases). All the tumors were metastatic: bone marrow metastases were found in three patients and hepatic metastases in four, two of whom also had peritoneal metastases. In four patients, the DIC was associated with hemorrhage. (63 refs)

79-4060 Concomitant Germ Cell Tumors in Monozygotic Twins. (Eng) Wilbur, H. J. (Div. Urology, Dept. Surgery, Albany Medical Center, Veterans Admin. Hosp., Albany, NY); Woodruff, M. W.; Welch, M. S. *J Urol* 121(4): 538-540; 1979.

Simultaneously occurring embryonal cell tumors in 38-yr-old monozygotic twin brothers are reported. Monozygosity was confirmed by histocompatibility testing, RBC typing, and chromosome analysis. This is the eighth documented case of testicular cancer in twins and the first report of bilateral testicular tumors in one twin. The role of heredity as a possible significant factor in germ cell tumor pathogenesis is explored. In the future, it may be possible to predict that a specific genotype in certain susceptible individuals may predispose various tissues, in this case germ cells, to malignant degeneration. In support of this hypothesis is the finding that cryptorchid testes, which show a higher incidence of neoplastic degeneration, predominantly display abnormal chromosome patterns. (15 refs)

79-4061 The Malignant Potential of the Dysgenetic Germ Cell in Klinefelter's Syndrome. (Eng) Sogge, M. R. (Letterman Army Medical Center, P.O. Box 32, Presidio of San Francisco, San Francisco, CA 94129); McDonald, S. D.; Cofold, P. B. *Am J Med* 66(3): 515-578; 1979.

Two cases of Klinefelter's syndrome (KS) associated with mediastinal germ cell tumors are presented, and previous reports of gonadal and extragonadal germ cell tumors in KS patients are reviewed. In one case (21-yr-old white man), the mediastinal tumor was a choriocarcinoma (the second such case reported); in the other case (26-yr-old white man), it was a yolk-sac tumor (the first such case reported). Six of the ten reported cases of germ cell tumors in KS patients have been primary extragonadal tumors rather than of testicular origin. Two of the 24 reported cases of primary mediastinal choriocarcinoma have been in patients with KS, a percentage much higher than would be ex-

pected for a chance occurrence. It is concluded that KS patients are uniquely predisposed to the development of germ cell tumors, particularly those of extragonadal origin. The pathogenesis of these tumors may be related to the abnormal nuclear sex pattern of the cells. (23 refs)

79-4062 Transplantable Tumor Cell Line Derived from a Spontaneous Fibrosarcoma in a Gerbil. (Eng) Gleiser, C. A. (Section Experimental Animals, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Pointer, J. M.; Jardine, J. H.; Raulston, G. L. *J Natl Cancer Inst* 62(6): 1579-1583; 1979.

The derivation of a cultured tumor cell line from a spontaneous fibrosarcoma of a gerbil (*Meriones unguiculatus*) is described. The cell type was predominantly neoplastic fibroblasts and, except for a few giant cells, was consistent in its morphology over successive tissue culture passages. All syngeneic gerbils inoculated sc with cultured cells developed rapidly growing tumors at the inoculation site. The tumors, which became clinically apparent at 7-10 days postinoculation, were large and discrete but not encapsulated. Metastases by direct extension or invasion into the skeletal muscle, pleura, and peritoneum were observed in 24/35 of the inoculated animals. Invasion of the chest wall was also apparent, and 8/35 animals had metastases in the lungs or liver. The overall tumor appearance was that of a poorly differentiated sarcoma consistent with fibrosarcoma. Chromosome counts ranged from 60 to 149, with 53% being in the range 75-81. (9 refs)

79-4063 Transmission of Transmissible Venereal Tumor of the Dog to the Coyote. (Eng) Cockrill, J. M. (Dept. Animal Sciences, Div. Agriculture, Univ. Arkansas, Fayetteville, AR 72701); Beasley, J. N. *Am J Vet Res* 40(3): 409-410; 1979.

Experiments to transmit a transmissible venereal tumor of the dog to the coyote and a number of other suckling animals are briefly described. Three coyotes (2 males, 1 female) were inoculated sc at three sites along the lateral thoracic wall with 2×10^6 tumor cells. The same number of tumor cells was also inoculated sc into 15 mice, 15 hamsters, 12 rats, 9 opossums, and 3 kittens. Sc pea-sized nodules were detectable in the coyotes by palpation 30 days after inoculation, and growth continued for the 4-mo observation period. The histopathologic appearance of the tumors in the coyotes was similar to that in the dog, except for an increased amount of fibrous stroma surrounding the tumor cells and the presence of small numbers of neutrophils. None of the suckling animals developed tumors. The successful transmission of the tumor to the coyote was expected, because the coyote interbreeds with the dog and it has the same karyotype. (16 refs)

79-4064 Gingival Metastasis from Primary Hepatocellular Carcinoma: Report of a Case.

(Eng) Wedgwood, D. (Faculty Dentistry, Univ. Manitoba, 780 Bannatyne Ave., Winnipeg, Manitoba R3E 0W3, Canada); Rusen, D.; Balk, S. *Oral Surg* 47(3): 263-266; 1979.

A case of primary hepatocellular carcinoma (HCC) metastatic to the gingiva is described. This is the eighth case of HCC metastatic to the oral cavity and the fourth involving metastasis to the gingiva reported in the English-language literature. The patient was a 56-yr-old white man who presented with a history of hematemesis and melena of sudden onset, increasing weakness, and right upper quadrant abdominal pain. Liver and spleen scan patterns were consistent with cirrhosis. Liver biopsy was interpreted as possible hepatoma. A transhepatic cholangiogram, ultrasound studies, and a celiac angiogram were suggestive of primary hepatoma or metastases. Needle biopsy of the liver gave results indicative of cirrhosis and possible HCC. Biopsy of a gingival mass resulted in a diagnosis of metastatic HCC. Bile pigment was demonstrated in the gingival lesion, and a greenish brown discoloration of the adjacent mucosa was noted. The gingival lesion thus afforded an opportunity to confirm the diagnosis in the absence of definite serologic and histologic evidence of hepatoma. (13 refs)

79-4065 A Case Report of Clear Cell Carcinoma of the Tongue. (Jpn) Miyakuni, Y. (Ear, Nose, and Throat Dept., Tottori Univ. Sch. Medicine, Yonago, Tottori Prefecture 683, Japan); Yutani, T.; Kobayashi, H. *Jibi Inkoka* 51(2): 147-150; 1979.

A clear cell carcinoma of the apex of the tongue was diagnosed in a 55-yr-old mildly diabetic woman whose only complaint was a swelling of the tongue. One older brother and one younger brother (2/9 siblings) had died of stomach cancer. A painless, nonhemorrhagic, and rather firm tumor showing gross signs of local inflammation and originally thought to be benign was histopathologically determined to be a clear cell carcinoma. No lymph node infiltration was seen in the submandibular or neck region. Along the borders of the multiangular and vertically aligned tumor cells were small nuclei containing profuse chromatin. Interstitial cells were scarce, and they were supported by a fibrous connective network. The tumor was resected without pre- or postoperative irradiation. There is no evidence of local recurrence or metastasis 2 yr, 4 mo after surgery. The tumor was thought to have originated in the minor salivary glands. (19 refs)

79-4066 Cytogenetic Observations on the Malignant Epithelial Cells and Infiltrating Lymphocytes of Nasopharyngeal Carcinoma. (Eng) Jarvis, J. E. (Dept.

Pathology, Univ. Bristol Medical Sch., University Walk, Bristol BS8 1TD, England); Finerty, S.; Epstein, M. A.; Trumper, P. A.; Ball, G.; Giovanella, B. C. *IARC Sci Publ* 20: 299-307; 1978.

Cytogenetic studies of both the malignant epithelial cells and the nonmalignant lymphoid cells of nasopharyngeal carcinoma (NPC) are described. These studies were prompted by the finding of a chromosome 14 marker abnormality in Epstein-Barr virus (EBV)-carrying cells from African Burkitt's lymphoma (BL) but not from other origins and the association of EBV with NPC as well as BL. Chromosome spreads from seven NPC-derived lymphoblastoid lines were examined after banding; five lines were diploid and two were tetraploid. No consistent chromosome 14 abnormalities were found. Apart from a secondary constriction near the centromere of both chromosomes 1 in all cells of one diploid line, no consistent significant abnormalities were seen. Five NPC tumors, freed of infiltrating lymphoid cells by passage through nude mice, were similarly examined after spindle arrest in vivo. Two tumors were near-diploid and three were hypotetraploid. Near-diploid cells had only minor chromosomal changes, but the hypotetraploid spreads from all tumors showed gross changes of uncertain origin, including frequent, major translocations. Chromosome 14 marker changes were not seen, and there was no other consistent pattern of abnormality in the tumor cells. (23 refs)

79-4067 Nasopharyngeal Carcinoma in an Alaskan Eskimo Family: Report of Three Cases. (Eng)

Lanier, A. P. (Bureau Epidemiology, Alaska Investigations Div., Center Disease Control, 225 Eagle St., Anchorage, Alaska 99501); Bender, T. R.; Tschopp, C. F.; Dohan, P. *J Natl Cancer Inst* 62(5): 1121-1124; 1979.

Nasopharyngeal carcinoma (NPC) has been reported to occur in Alaska natives (Eskimos, Indians, and Aleuts) at a rate >15 times the rate for US whites. The adjusted annual incidence rates per 100,000 in this population are 13.5 for men and 3.7 for women. Three cases of NPC were discovered in a Northern Alaskan Eskimo mother (case 2) and her two sons (3rd and 4th generations, respectively). This is the first report of multiple cases of NPC in a family of Alaskan Eskimos and the first report of more than two cases occurring in a family in the US. All three patients had poorly differentiated tumors and died <1 yr after diagnosis from metastatic NPC. Blood group, histocompatibility (HLA) antigens, and antibodies to Epstein-Barr virus (EBV) were studied in the family members. EBV titers were elevated in the 1 NPC patient tested and in 1/15 other family members tested. All family members studied except one were of blood group A, Rh-positive, and HLA types AW24, CW3, BW40/A2, CW2, and B27. The mother of case 2 may also have died from NPC, and two other members of the third generation died from other types of cancer. (16 refs)

79-4068 Frequency and Significance of Epithelial Atypia in Laryngeal Papillomatosis. (Eng)

Quick, C. A. (Div. Otolaryngology, West Virginia Univ. Medical Center, Morgantown, WV 26506); Foucar, E.; Dehner, L. P. *Laryngoscope* 89(4): 550-560; 1979.

Possible histologic characteristics correlated with the clinical behavior of laryngeal papillomatosis were studied in 32 patients with laryngeal papillomas. In nine patients, the onset of disease occurred in childhood or early adolescence, and they had a low frequency of operations for the disease and a minimal spread of the papillomas (Type I patients). The lesions remitted spontaneously before or during adolescence or in early adulthood, and they showed an orderly maturation of cells from the basal layers to the surface. The number of circulating eosinophils was increased in these patients. In 6 patients, the onset of disease also occurred in childhood or early adolescence, but all patients had had many operations due to rapid recurrences (Type II patients). In five of these patients, remission occurred in childhood or adolescence; in three, active disease persisted into adulthood; and in eight, there were no complete remissions. The histologic patterns in these patients progressed from initial typical papilloma to mild, moderate, and then severe atypia with no orderly maturation or progression from basal to surface layers. In seven patients, the onset of the disease occurred in adulthood and was generally solitary (Type III patients). The lesions showed a typical papilloma appearance with very little atypia. There was no evidence of invasive carcinoma in any of the 32 patients. (19 refs)

79-4069 Malignant Transformation of Tracheobronchial Juvenile Papillomatosis Without Prior Radiotherapy. (Eng)

Siegel, S. E. (Div. Hematology-Oncology, Children's Hosp. Los Angeles, 4650 Sunset Blvd., CA 90027); Isaacs, H.; Cohen, S. R.; Stanley, P. *Ann Otol Rhinol Laryngol* 88(2, part 1): 192-197; 1979.

A case of fatal squamous cell carcinoma of the trachea and bronchi developed in a 19-yr-old man who had been previously tracheotomized to relieve obstruction from recurrent laryngeal papillomas. The patient had undergone prolonged tracheotomy and numerous laryngoscopic and bronchoscopic procedures since the age of 4 yr for recurrent laryngeal papillomas. In addition, recurrent tracheal papillomas developed at age 8. Treatment with cytosine arabinoside produced no change in tumor growth; the patient did not receive radiotherapy until the appearance of tracheal and bronchial squamous cell carcinoma 15 yr following the initial diagnosis. Irradiation and systemic and topical chemotherapy were ineffective, and recurrent arterial compromise due to malignant tumor emboli was eventually fatal. The clinical course in this patient indicates that malignant change can occur in recurrent juvenile papillomatosis in the absence of prior radiotherapy. (27 refs)

79-4070 Tumorigenicity and Melanisation (Meeting Abstract). (Eng)

Aubert, C. (u.119 de l'INSERM, Marseille, France); Foa, C.; Rouge, F.; Galindo, R.; Pirisi, V. *J Invest Dermatol* 72(5): 289; 1979 (no refs)

79-4071 Genetic Control of Tumorigenicity in Interspecific Mammalian Cell Hybrids. (Eng)

Kucherlapati, R. (Dept. Biochemical Sciences, Princeton Univ., Princeton, NJ 08540); Shin, S. *Cell* 16(3): 639-648; 1979.

The genetic control of cellular malignancy was investigated by examining the tumorigenicity of interspecific mouse-human cell hybrids in the athymic nude mouse. Two highly malignant but genetically distinct mouse cell lines, A9 and PG19, were hybridized with three normal human diploid fibroblast strains, and 19 independent hybrid clones were isolated. Each of these clones was capable of forming progressive lethal tumors in the nude mouse and thus resembled the malignant parental mouse cells rather than the non-malignant parental human cells. There was no evidence for complete suppression of tumorigenicity in these cell hybrids. The absence of suppression was observed regardless of the extent and composition of the human chromosome complements retained in the hybrid clones. The results of detailed cytological and isoenzyme analyses would make it highly improbable that the lack of suppression was due to cellular selection in vivo for a more tumorigenic subpopulation in the injected hybrid cells. These data demonstrate that, at least for the parental cell combinations used in this study, no human chromosome, when present singly in the mouse-human cell hybrids, can suppress the tumorigenic phenotype of the mouse cells. The results are consistent with the view that the suppression of cellular malignancy previously demonstrated in intraspecific (mouse x mouse) somatic cell hybrids does not occur in interspecific (mouse-human) cell hybrids or, alternatively, that genetic determinants located on two or more human chromosomes are required simultaneously to suppress the malignancy of the mouse cells in cell hybrids derived from malignant mouse cells and nonmalignant human cells. (39 refs)

79-4072 Microfilament Bundles in Transformed Mouse CLID x Transformed CHO Cell Hybrids. Correlation with Tumorigenicity in Nude Mice. (Eng)

Celis, J. E. (Div. Biostructural Chemistry, Inst. Chemistry, Aarhus Univ., 8000 Aarhus C, Denmark); Small, J. V.; Kaltoft, K.; Celis, A. *Exp Cell Res* 120(1): 79-86; 1979.

The tumorigenicity and pattern of microfilament bundles of cell hybrids produced by polyethylene glycol fusion of tumorigenic mouse CLID cells and transformed Chinese hamster ovary (CHO) cells were studied. All of the hybrids

had segregated CHO chromosomes, and all hybrids but one (Hy 12) showed the crossed pattern of microfilament bundles observed in the CLID cells. Most Hy 12 cells had a microfilament pattern similar to that observed in CHO cells. The parental CLID line was highly tumorigenic in nude mice, resulting in a 100% tumor incidence after injection of 6×10^4 cells. CHO produced tumors in <15% of the mice tumors after injection of 10^6 cells. Only four of the hybrids consistently produced tumors at the site of injection when 10^6 cells were injected per mouse, and the latent period was always considerably longer than that observed with the CLID parent. A fifth hybrid produced tumors in 33% of inoculated animals, two others produced tumors in 16%, and the eighth hybrid (Hy 12) produced no tumors. The data showed no direct relationship between the presence of a crossed pattern of microfilament bundles and tumorigenicity. (26 refs)

- 79-4073 Analysis of a Multisequential Transformation in a Pig Cell Line (PFT) (Meeting Abstract).** (Eng) Bouillant, A. M. (Animal Disease Res. Inst., Agriculture Canada, Ottawa, Ontario K2H 8P9, Canada); Genest, P.; Greig, A. S. *In Vitro* 15(3): 227; 1979 (no refs)

- 79-4074 Racial and Age-related Differences in the Activity of α -N-Acetyl-D-glucosaminidase in Man.** (Eng) Singh, J. (Dept. Pathology, Veterans Admin. Hosp., Houston, TX 77211); Gyorkey, F. *Biochem Med* 20(3): 336-343; 1978.

Examination of the sera of 200 healthy Blacks and whites showed that Blacks had two- to threefold higher α -N-acetyl-D-glucosaminidase activity than whites. These differences were significant for all age groups (20-59 yr) in men, but only for age groups 20-29, 30-39, and 40-49 in women. In the entire population, the enzyme activity decreased significantly with age. (26 refs)

- 79-4075 Lysosomal Enzymes in Pure Pancreatic Juice from Normal Healthy Volunteers and Chronic Alcoholics.** (Eng) Rinderknecht, H. (Veterans Admin. Hosp., Sepulveda, CA 91343); Renner, I. G.; Koyama, H. *H. Dig Dis Sci* 24(3): 180-186; 1979.

Normal levels of some lysosomal hydrolases in the pancreatic juice of healthy individuals were established, and the possibility that conditions such as chronic alcoholism may bring about early biochemical changes in the pancreas that are reflected in the type or concentration of lysosomal enzymes in its secretions was explored. Investigation of pure human pancreatic juice obtained by direct cannulation of the main pancreatic duct of 11 healthy volunteers

and 10 chronic alcoholics without detectable pancreatic disease revealed the presence of numerous acid hydrolases in this secretion. The pH optima and substrate specificities of these enzymes suggested that they are of lysosomal origin. Stimulation of the pancreas by injection of cholecystokinin-pancreozymin (CCK-PZ: 1 Ivy dog unit/kg) markedly increased the activity of some of these hydrolases (N-acetyl- β -D-glucosaminidase, arylsulfatase, etc) similar to that observed for trypsin, amylase, and other pancreatic digestive enzymes. In a second group of hydrolases (β -glucuronidase, leucine naphthylamidase, etc), the effect of this hormone was greatly reduced or absent, particularly in normal individuals. In chronic alcoholics, enzyme activity in response to CCK-PZ injection was greater than that in normal subjects. Although this increase was statistically significant ($p < 0.05$) only for β -D-glucuronidase, it was observed for all lysosomal hydrolases tested and suggests either an increased synthesis or a more facile release of these enzymes from the pancreas of chronic alcoholics than from the pancreas of normal individuals. (18 refs)

- 79-4076 Studies on the Pathogenesis of Aplastic Anemia.** (Eng) Kagan, W. A. (Dept. Medicine, Univ. Hosp., 75 E. Newton St., Boston, MA 02215); Ascensao, J. L.; Fialk, M. A.; Coleman, M.; Valera, E. B.; Good, R. A. *Am J Med* 66(3): 444-449; 1979.

Three assays were used to study myelopoiesis in 14 patients (7 men, 7 women aged 8-74 yr) with aplastic anemia: (1) the soft agar colony assay for granulocyte-monocyte progenitors (CFU-c); (2) coculture of marrow from patients with normal marrows in the CFU-c assay; and (3) culture of marrow pretreated with antithymocyte globulin (ATG) in the CFU-c assay. Marrow from five patients gave low colony counts when cultured alone and suppressed colony formation by normal marrow cells in coculture. Suppressor cells may have caused the aplasia in these patients. Eight patients had low colony formation and no suppression in coculture. These patients may have absent or defective stem cells. Marrow from one patient produced normal colony formation, but did not contain suppressor cells; this patient may have a defective hematopoietic environment. Aplastic anemia thus may result from at least three different defects involving stem cells, the hematopoietic environment, or suppressor cells. (22 refs)

- 79-4077 Polyarteritis Nodosa and Hairy-Cell Leukaemia (Letter to Editor).** (Eng) Hughes, G. R. (Royal Postgraduate Medical Sch., London W12, England); Elkon, K. B.; Spiller, R.; Catovsky, D.; Jamieson, I. *Lancet* 1(8117): 678; 1979.

The case report of a patient in whom the coincidence of

hairy cell leukemia (HCL) and polyarteritis nodosa may have pathogenetic implications is presented. The patient developed intermittent claudication of the right leg following the diagnosis of HCL and after experiencing several infections. The susceptibility of HCL patients to infection is largely due to neutropenia, but it may also result from a profound monocytopenia and impaired monocyte function. Since both conditions are rare, a chance association is unlikely. (6 refs)

- 79-4078 Chromosomes and Causation of Human Cancer and Leukemia. XXXI. Dq- Deletions and Their Significance in Proliferative Disorders.** (Eng) Kohno, S. (Roswell Park Memorial Inst., Buffalo, NY 14263); Van Den Berghe, H.; Sandberg, A. A. *Cancer* 43(4): 1350-1357; 1979.

Three patients with myeloproliferative disorders associated with Dq- deletions in their marrow cells were studied. The cells of a 26-yr-old man with chronic myelocytic leukemia (CML) were initially positive for the Philadelphia (Ph¹) chromosome with no other karyotypic changes. Two years later, when the patient was in the blastic phase, all of the peripheral and bone marrow lymphocytes examined were positive for Ph¹ and almost all showed an interstitial deletion of the long arm of chromosome 15. Of 103 peripheral and bone marrow lymphocytes from a mentally retarded 56-yr-old woman with polycythemia vera, 39 showed a deletion of chromosome 13. The frequency of this deleted chromosome decreased in the blood and marrow with time. More than 70% of the lymphocytes of a 69-yr-old woman with CML were Ph¹-positive, and all of the cells in the blood and marrow contained a deletion of chromosome 15. A total of 14 patients with myelo- or lymphoproliferative disorders and with either 13q- or 15q- were observed in Leuven, Belgium. Data on these patients plus those reported in the literature are tabulated. The findings indicate that 13q and 15q should be added to the list of chromosomes showing particular vulnerability for breakage of the long arms in various proliferative disorders. (36 refs)

- 79-4079 Clinical-Morphological, Cytogenetic, and Genealogical Examination of Chronic Monocytic Leukemia Patients.** (Rus) Sherman, S. I. (Hematological Clinic, Inst. Hematology and Blood Transfusion, Leningrad, USSR); Abdulkadyrov, K. M.; Shandlorenko, S. K.; Kuraleva, V. V. *Vopr Onkol* 25(1): 38-43; 1979.

Clinical data on a group of 55 patients with chronic monocytic leukemia (CML) are reviewed. The group included 28 men and 27 women; 45/55 patients were >55 yr. Characteristic clinical features of CML were systemic hyperplasia of the hemopoietic tissue (53); enlargement of

the lymph nodes, spleen, and liver (53); anemia (31); leukopenia (35); and thrombocytopenia (30). Of 48 examined patients, 9 had an increased myelokaryocyte count and 8 had a decreased number of nucleolar elements; 22 patients had hypomegakaryocytosis and 6 had hypermegakaryocytosis. Almost all patients had an increased number of reticular cells and monocytes (5%-33.8%). Cytogenetic analysis was performed in 20 patients: the frequency of cells with a hypodiploid chromosome complement was 5.7% (compared with 5.2% in controls), and the frequency of cells with a hyperdiploid chromosome complement was 2.8% (compared with 0.7% in controls). The number of hyperdiploid cells was significantly greater in patients with a relapse of CML (3.9%) than in patients in remission (1.6%). Review of family histories showed that 7/55 patients had first-degree relatives who had died of malignant neoplasia. (23 refs)

- 79-4080 Malignant Lymphoma in Spontaneously Diabetic Rats (Letter to Editor).** (Eng) Kalant, N. (Lady Davis Inst. Medical Res., Jewish General Hosp., Montreal, Quebec, Canada); Seemayer, T. *N Engl J Med* 300(13): 737; 1979.

Study of a colony of nonobese Wistar rats (BB) with a high rate of spontaneous, apparently genetically determined diabetes mellitus revealed that of 95 insulin-treated diabetic rats that died during the observation period, 12 with diabetes of 7-11 mo duration had mesenteric tumors (vs 1/86 nondiabetic rats from the same colony). Cytologically, the tumors had the characteristics of malignant lymphoma, some with plasmacytoid differentiation and others with features of immunoblastic sarcoma. Although the cause of the insulinitis and lymphoma is unknown, a common viral or immunologic factor superimposed on a genetic susceptibility is possible. (6 refs)

- 79-4081 Massive Medullary Forms of Hodgkin's Disease and Acute Myelofibrosis.** (Fre) Duhamel, G. (Service du Maladies du Sang, Hopital Saint-Antoine, 184 rue du Fbg St.-Antoine, F75012 Paris, France); Stachowiak, J.; Monteiro, M. *Nouv Presse Med* 8(13): 1061-1064; 1979.

Hodgkin's disease with massive bone marrow invasion was found in 10 patients (6 men and 4 women, av age 38 yr). Pancytopenia, a depleted bone marrow, and few or no superficial lymph nodes were the typical features. The patients could be subdivided into two groups. In the first group (6 patients), this form of Hodgkin's disease was present from the onset. Manifest splenomegaly was found in six patients, hepatomegaly in four. Bone marrow biopsy showed myelofibrosis in all the patients, and cellular immunity was impaired. A combination of chemotherapy and corticotherapy brought about a 3-mo remission in one pa-

tient, but it aggravated the hematological syndrome in the other cases. The patients died within 14-16 mo. In the second group (4 mostly young patients), the bone marrow invasion was manifested in Stage IIIB. Chemotherapy followed by supra- and subdiaphragmatic radiotherapy caused remissions lasting 3-11 mo, after which the patients relapsed with fever, hepatomegaly, splenomegaly, and pancytopenia. Myelograms showed a depleted bone marrow in all cases, and bone marrow biopsy showed massive myelofibrosis. The recurrence was refractory to chemotherapy, and the patients died within 4-6 mo. A special and unexplained behavior of the host in response to these two forms of Hodgkin's disease indicate that these very atypical forms of Hodgkin's disease must be considered a possibility in the context of acute malignant myelofibrosis. (20 refs)

- 79-4082** Cytogenetic Evidence of the Intrauterine Origin of Acute Leukemia in Monozygotic Twins. (Eng) Chaganti, R. S. (Dept. Pediatrics, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021); Miller, D. R.; Meyers, P. A.; German, J. *N Engl J Med* 300(18): 1032-1034; 1979.

The occurrence of the same cytogenetic abnormalities in the leukemic blast cells of 15-mo-old male identical twins with acute lymphocytic leukemia is reported. The chromosomes of both children generally exhibited poor morphology and spreading. Giemsa-banded bone marrow cells showed two marker abnormalities: the long arm of the No. 1 chromosomes was absent distal to band 1q22, and an extra submetacentric chromosome in the size range of a No. 2 chromosome was present. It is likely that the leukemic cells populating the bone marrows of both twins originated in a single cell (ie, their disease was monoclonal). The same mechanism is probably important in relation to the origin of childhood cancer in general. The onset of leukemia in the twins so early after birth suggests leukemic transformation of a cell in one twin during fetal life, with subsequent proliferation and migration into the other twin's body via a shared circulation. (11 refs)

- 79-4083** Hodgkin's Disease of Small Intestine. (Eng) Bagree, M. M. (Dept. Surgery, J. L. N. Medical Coll., Ajmer, India); Kala, P. C.; Vijayvargiya, S. K. *J Indian Med Assoc* 71(6): 153-154; 1979.

A rare case of Hodgkin's disease of the jejunum occurred in a 22-yr-old man. The patient presented with an abdominal mass and pain associated with vomiting and distention. (7 refs)

- 79-4084** Kaposi's Sarcoma. Case Report and Review of Japanese Cases. (Eng) Tange, T. (Dept.

Pathology, Faculty Medicine, Univ. Tokyo, 3-1, Hongo 7-chome, Bunkyo-ku, Tokyo, Japan). *Acta Pathol Jpn* 29(2): 319-332; 1979.

The histogenesis of Kaposi's sarcoma in a 60-yr-old Japanese man was studied. The initial biopsy of the scalp tumor revealed a skin lesion suggestive of capillary hemangioma. The tumor was subsequently removed and examined microscopically. It showed wide morphological variations, including granulomatous, angiogranulomatous, angiomatous, and sarcomatous lesions. At postmortem several mo later, the recurrent tumor was found to contain a loose proliferation of dilated capillaries with wide intervascular collagenous and edematous stroma plus focal hemorrhage and a slight mononuclear cell infiltration. In addition, several hemorrhagic foci in the bone marrow of the calvarium without bone destruction were noted, and there were multiple patchy hemorrhagic lesions in the pleura and pericardium. Involvement of the pulmonary parenchyma by Kaposi's sarcoma was histologically characterized by diffusely distributed angiomatous and angiogranulomatous lesions confined to the pleura and subpleura. (30 refs)

- 79-4085** Immunoblastic Sarcoma in Donor Cells after Bone-Marrow Transplantation. (Eng) Gossett, T. C. (Dept. Pathology, UCLA Sch. Medicine, Center Health Sciences, Los Angeles, CA 90024); Gale, R. P.; Fleischman, H.; Austin, G. E.; Sparkes, R. S.; Taylor, C. R. *N Engl J Med* 300(16): 904-907; 1979.

An immunoblastic sarcoma (lymphoma) developed in donor cells following a bone marrow transplant in a 20-yr-old man with acute myelogenous leukemia. The patient had received a marrow graft from his sister, who was HLA identical but reactive on mixed lymphocyte culture (MLC). One month following the transplant, the development of graft-vs-host disease was evident, and over the next 6 wk a wasting syndrome developed, with severe diarrhea, ascites, and liver failure. On the day before death, the peripheral WBC count rose abruptly to 42.0×10^9 cells/liter, primarily with immunoblasts and plasmacytoid lymphocytes. Massive lymphadenopathy and hepatosplenomegaly were present. The unusual immunoproliferative disorder appeared to be malignant because of the marked degree of organ infiltration (gastrointestinal tract, liver, spleen, lungs, kidneys, lymph nodes) observed at autopsy, the high rate of mitotic activity, and immunologic evidence of monoclonality. Electrophoretic analysis of esterase D indicated that the tumor arose in donor cells. The tumor may have arisen as a result of an uncontrolled immune response, the stimulus possibly having been provided by the graft-vs-host disease. The latter may have been aggravated by the incompatibility between donor and recipient shown on MLC. (14 refs)

79-4086 Osteosarcoma in a Necrotic Papilla in a Patient with Analgesic Nephropathy (Meeting Abstract). (Ger) Gloor, F. (Institut für Pathologie, Kantonsspital, St. Gallen, Switzerland). *Schweiz Med Wochenschr* 109(10): 370; 1979 (no refs)

79-4087 Alveolar Soft Part Sarcoma: A Report of Two Cases with Some Histochemical and Ultrastructural Observations. (Eng) Ekfors, T. O. (Dept. Pathological Anatomy, Univ. Turku, 20520 Turku 52, Finland); Kalimo, H.; Rantakokko, V.; Latvala, M.; Parvinen, M. *Cancer* 43(5): 1672-1677; 1979.

In a survey of all soft tissue tumors in the extremities and limb girdles in Finland between 1960 and 1969, only one alveolar soft part sarcoma (ASPS) was found among 246 tumors (0.4%). Another ASPS, diagnosed in 1976, in the leg of a 22-yr-old man, was more thoroughly studied. There was evidence that the characteristic crystals of ASPS sarcoma are formed from the dense granules. Both tumors were PAS methenamine-positive at the ultrastructural level. No monoamines were detected in the cells by formaldehyde-induced fluorescence. This is further evidence to nullify the theory of the paraganglionic origin of alveolar soft part sarcoma, but the question of the histogenesis of the tumor remains unresolved. (19 refs)

79-4088 The Association of Neurofibromatosis and Hyperparathyroidism. (Eng) Chakrabarti, S. (Dept. Surgery, Lutheran Medical Center, 150 55th St., Brooklyn, NY 11220); Murugesan, A.; Arida, E. J. *Am J Surg* 137(3): 417-420; 1979.

Two women with coexisting neurofibromatosis and hyperparathyroidism (due to parathyroid adenoma) are described, bringing the number of such cases in the world literature to seven. The association of these two disorders adds to the growing list of examples of the association of tumors of neuroectodermal and endodermal origin. Other examples include the association of medullary thyroid carcinoma (MTC) and pheochromocytoma; pheochromocytoma, thyroid carcinoma, and neurofibromas; and MTC, pheochromocytoma, and parathyroid hyperplasia. The association of neurofibromatosis and hyperparathyroidism may be a variant of multiple endocrine neoplasia type 2. (17 refs)

79-4089 Hepatoma in a Child with Neurofibromatosis. (Eng) Ettinger, L. J. (Dept. Pediatrics, Univ. Rochester Medical Center, Strong Memorial Hosp., Rochester, NY); Freeman, A. I. *Am J Dis Child* 133(5): 528-531; 1979.

The case report of 6.5-yr-old girl who had neurofibromatosis (NFT) and hepatocellular carcinoma (hepatoma) is presented. Medical history revealed that numerous café au lait spots had developed at age 1 yr. They increased in size and number until the time of death at age 7.5 yr. At age 1.5 yr, a soft-tissue mass in the left periorbital and zygomatic regions was diagnosed as a nerve sheath myxoma (neurofibroma). The tumor recurred twice over the following 2 yr. The child's mother had taken birth control pills throughout the first trimester of pregnancy, and she continued to have normal menstrual periods throughout her pregnancy. There was no family history of NFT or liver disease. Hepatic cancer, Wilms' tumor, and adrenocortical neoplasia are seen with increased frequency in association with certain congenital anomalies, particularly with hemihypertrophy and various hamartomas. NFT, a hamartomatous disorder, is also seen with these congenital anomalies, and it is reported with an unusually high incidence in patients with Wilms' tumor. It is suggested that these disorders may be related. (24 refs)

79-4090 Primitive Neuroectodermal Tumor (Neuroepithelioma) of Spinal Nerve Root. Report of an Adult Case and Establishment of a Cell Line. (Eng) Ishikawa, S. (First Dept. Pathology, Niigata Univ. Sch. Medicine, Niigata, Japan); Ohshima, Y.; Suzuki, T.; Oboshi, S. *Acta Pathol Jpn* 29(2): 289-301; 1979.

A primitive neuroectodermal tumor (neuroepithelioma) arose in the cervical nerve root of a 28-yr-old man. Histologically, the tumor was characterized by a proliferation of primitive neuroectodermal cells and the formation of numerous Homer-Wright-type rosettes. A cell line (Nagai line) was established from the tumor. Electron microscopic examination of Nagai cells revealed numerous microrosette formation with microvillilike cytoplasmic processes projecting into the central lumina. Neurosecretory granules appeared in the cytoplasmic processes when Nagai cells were treated with dibutyryl cyclic AMP. Primitive satellite cells that completely surrounded other tumor cells with their tongue-like slender cytoplasmic processes were also found. The possible histogenesis of this unique tumor was compared with that of neuroblastoma of the sympathetic nervous system, medulloblastoma of the CNS, and tumors induced by adenovirus type 12 in animals. It was concluded that the tumor was a neuroepithelioma that was derived from a primitive stem cell of neural crest origin and that it possessed the biopotential to differentiate along both neuroblastic and neurilemmal lines. (21 refs)

79-4091 Simultaneous Clinical Manifestation of Subependymoma of the Fourth Ventricle in Identical Twins. Case Report. (Eng) Clarenbach, P. (Dept. Neurology, Univ. Freiburg, Freiburg, W. Germany);

Kleihues, P.; Metzel, E.; Dichgans, J. *J Neurosurg* 50(5): 655-659; 1979.

The simultaneous development of subependymomas of the fourth ventricle in 22-yr-old identical twin brothers is reported. The brothers simultaneously developed symptoms of intracranial pressure, including vomiting, disturbances of balance, pulsating headaches, visual impairment, and papilledema. Radiologic investigation revealed cerebellar midline tumors with occlusive hydrocephalus of the third and lateral ventricles. At surgery, subependymomas with identical histologic features were found in the fourth ventricles of both twins. The tumors showed characteristic isomorphous glial cells with rounded or slightly elongated nuclei embedded in a dense fibrillary matrix. In both cases there was a clear demarcation of the tumors from adjacent brain tissue. Approx 2 yr later, computerized tomography scans showed residual tumors adjacent to the fourth ventricle; these had not increased in size since the postoperative scan performed 2 mo after surgery. The identical time course of tumor growth in the twins suggested that not only histology and topology but also growth dynamics were prenatally determined. The family history showed no evidence of inheritance, and the observations favored the view that the tumors were due to a developmental anomaly. (29 refs)

79-4092 Lymphoma of the Brain Associated with Polyposis of the Colon. Report of a Case and Review of Turcot's Syndrome. (Eng) Hara, M. (Dept. Pathology, Yokohama City Univ. Sch. Medicine, 2-33, Urafune-cho, Minami-ku, Yokohama, Japan); Misugi, K.; Suda, T.; Kuwana, N. *Acta Pathol Jpn* 29(2): 233-241; 1979.

Pathologic findings in a 15-yr-old girl with polyposis of the colon who subsequently developed a primary lymphoma of the brain are reported. This case was considered to be a rare example of Turcot's syndrome, although the histological typing of the brain tumor was not classical and a familial background of polyposis was not demonstrated. The terminal ileum and rectum contained 129 adenomatous polyps of various sizes, 15 of which showed malignant transformation. Metastasis to the bifurcation of the common iliac artery and liver was observed. The hepatic metastatic lesion was a mucoid adenocarcinoma. There was no evidence of malignant lymphoma in the lymphoid tissue, spleen, or bone marrow. The right thalamic region, basal ganglia, hypothalamus, and periventricular area of the third and lateral ventricles and left temporal lobe showed evidence of neoplastic infiltration, and marked lymphocytic cuffing of the vessels was seen along the borders of the neoplastic invasion. The picture was consistent with a primary reticulum cell sarcoma of the brain. It is felt that the type of brain tumor involved in Turcot's syndrome is variable and not necessarily limited to glioma and that the present case is an example of this syndrome. The patient

showed no immunologic abnormalities other than a slight elevation in circulating IgM. (29 refs)

79-4093 Pathogenesis of Attic Cholesteatoma (Letter to Editor). (Eng) Sade, J. (Meir General Hosp., Kfar-Saba, Israel). *J R Soc Med* 72(3): 230-231; 1979.

In response to a previous article it is stated that although keratinization occurs in epidermoid cholesteatomas, it is also seen in several metaplastic processes, including ozena and, accompanying inflammatory processes, in the ear. There is, however, no information regarding the amount or rate of keratin synthesis in either process (epidermoid or metaplastic), and the use of the term hyperkeratinization is probably inaccurate. There is no firm evidence to support the theory that attic cholesteatoma (AC) is an invasive phenomenon originating from the external canal. AC's have often been found to be associated with various metaplastic processes in the middle ear. (6 refs)

79-4094 Cytogenetics of Extragonadal Tumors. (Eng) Kaplan, C. G. (Dept. Pathology, Univ. California, San Diego, La Jolla, CA 92093); Askin, F. B.; Benirschke, K. *Teratology* 19(2): 261-266; 1979.

Tissue from six human extragonadal teratomas (2 gluteal lesions from infant females, and thyroid, mediastinal, sacral, and gastric lesions from males) was obtained for cytogenetic study. The mediastinal lesion was predominantly tetraploid in culture and in the uncultured material. The other lesions all showed the same karyotype as the host, 46,XX in lesions from females and 46,XY in lesions from males. Polymorphisms between members of chromosome pairs were also similar in both host and tumor. The data support a mitotic origin for extragonadal teratomas. All available data support the origin of extraovarian teratomatous lesions from cells identical to host cells, similar to identical twins. These lesions are considered to be true tumors, rather than peculiar malformations. (21 refs)

79-4095 Locally Aggressive Fibrous Histiocytoma of Bone. A Case Report. (Eng) Nunnery, E. W. (Dept. Pathology and Radiology, North Carolina Memorial Hosp., Chapel Hill, NC 27514); Kahn, L. B.; Guilford, W. B. *S Afr Med J* 55(19): 763-767; 1979.

A fibrohistiocytic tumor involving the distal diaphysis of the left femur of a 44-yr-old woman is reported. The patient experienced acute onset of pain in the left knee, which had been injured 16 yr earlier. A biopsy specimen was diagnosed as malignant fibrous histiocytoma with areas

having a benign appearance. The leg was amputated, and the amputation specimen and tumor were examined. The tumor occupied the entire width of the medullary cavity of the femur, causing slight expansion of the bone. In one area, there was complete disruption of the cortex by tumor, which had extended into the surrounding soft tissue. Microscopically, the tumor was composed of an admixture of foamy histiocytes and spindle-shaped cells arranged in a storiform pattern. The diagnosis was changed to locally aggressive fibrous histiocytoma. There was no cytologic anaplasia and there was a complete absence of mitotic activity in the sections examined. Metaphyseal fibrous defect, benign fibroxanthoma, and malignant fibrous histiocytoma must be considered in the differential diagnosis of locally aggressive fibrous histiocytoma of bone. (26 refs)

- 79-4096 Malignant Transformation of Polyostotic Fibrous Dysplasia.** (Eng) Johnson, C. B. (Dept. Pathology, Univ. Wisconsin Medical Sch., Center Health Sciences, Madison, WI 53706); Gilbert, E. F.; Gottlieb, L. I. *South Med J* 72(3): 353-356; 1979.

The evolution of nonirradiated polyostotic fibrous dysplasia (PFD) into a poorly differentiated sarcoma is reported. A 25-yr-old white woman in whom a diagnosis of PFD was made at 2 yr of age developed a tumor of the left hip. A hip disarticulation disclosed a bosselated, 8- x 12-cm, partially encapsulated tumor occupying the neck of the femur. The cut surface of the tumor was focally excavated and light tan-gray to red, and there were several areas of pale nodularity extending down into the medullary portion of the femoral shaft, with similar areas in the medullary portion of the tibia. The tumor progressed and metastasized extensively following surgery and chemo- and radiotherapy. At postmortem, the tumor showed the histologic characteristics of a malignant mesenchymal tumor with features of rhabdomyosarcoma. Masculinization was also noted, and analysis of an autopsy serum specimen revealed the presence of a high level of testosterone (143 nanograms/ml). The high androgen level, which would explain the masculinization, may have been related to ovarian metastasis. (10 refs)

- 79-4097 Cutis Verticis Gyrata, Hypertrophic Gastritis, Motor Diarrhea, and Horsetail Syndrome; a New Association Indicative of Apudoma (Carcinoid or Chemodectoma).** (Fre) Moulas, R. (Service de Medecine Interne, Hopital de la Salpetriere, 47, bd de l'Hopital, 75634 Paris Cedex 13, France); Noble, J. P.; Auriol, M.; Ernst, D.; Bouvier, A. M.; Devars du Mayne, J. F.; Congy, F.; Loeper, J. *Sem Hop Paris* 55(9/10): 435-441; 1979.

A malignant osteolytic sacral tumor was found in a 70-yr-old man with cutis verticis gyrata (without hypertrophic

osteopathy), hypertrophic gastritis (Menetrier's disease), motor diarrhea of the endocrine type, flush syndrome, liver angiomas, and caida equina. The urinary 4-hydroxyindolacetic acid level and the serotonin level were increased. Scalp biopsy revealed sebaceous hyperplasia. The patient died despite melphalan treatment. Autopsy revealed a small necrotic tumor in the posterior wall of the rectum, which was believed to be the primary tumor with sacral metastases. Due to the necrosis, the primary tumor could not be identified histologically; it was thought to be a chemodectoma or a locally invasive carcinoid tumor. The paraneoplastic syndrome associated with cutis verticis gyrata is assumed to be specific for APUD (amine precursor uptake and decarboxylation) cell tumors. (41 refs)

- 79-4098 Extraskelatal Plasma Cell Tumors.** (Fre) Audhuy, B. (Clinique des Maladies du Sang, Centre Hospitalo-Universitaire, 1, place de l'Hopital, 67005 Strasbourg Cedex, France); Lang, J. M.; Bergerat, J. P.; Weill-Bousson, M.; Oberling, F. *Sem Hop Paris* 55(5/6): 229-233; 1979.

Three cases of extraskelatal plasmacytoma in 2 men aged 72 yr and 1 woman aged 67 yr are reported. Multiple plasma cell sarcomas of the testicle, scalp, and forearm were diagnosed in one male patient. Retroperitoneal and ip lymph node metastases were also found. He developed skeletal (femoral) plasmacytoma during the terminal phase. Monoclonal dysglobulinemia was not seen. Another patient had plasma cell sarcomas of the stomach, sigmoid colon, and parietal portion of the skull. Epidermoid epithelioma of the lungs was also discovered. Monoclonal dysglobulinemia and plasma cell invasion of the bone marrow were absent. The female patient developed small multiple tumors diagnosed as plasma cell sarcomas a few mo after she received sc injections of fresh cells (probably sheep cells) in connection with a "rejuvenating cure." The tumors developed at the injection sites. Extraskelatal plasmacytoma often seems to arise from a pathologic plasma cell reaction to antigenic stimulation. (30 refs)

- 79-4099 Sister Chromatid Exchanges and Their Relevance to Defective DNA Repair in Xeroderma Pigmentosum Cells.** (Eng) Pant, G. S. (Dept. Radiotherapy, All India Inst. Medical Sciences, New Delhi 110016, India); Kamada, N. *Indian J Exp Biol* 16(11): 1194-1196; 1978.

A study was performed to determine whether the formation of sister chromatid exchanges (SCE's) is due to DNA repair processes by comparing the SCE yield in normal (3 donors) and xeroderma pigmentosum (XP: 1 donor) cells. WBC from the four donors were stimulated in culture with phytohemagglutinin and treated with 5-bromodeoxyuridine. One culture from each of the four donors was also

treated with mitomycin C (0.01 $\mu\text{g/ml}$) for the last 24 hr. SCE's in the fixed cells were detected by the fluorescence plus Giemsa technique. An av of 12.1 and 16 SCE's/cell was detected in the chromosomes of normal donors and the XP patient, respectively. Mitomycin C increased the yield of SCE's equally in the normal and XP cells. If excision or postreplication repair were involved in SCE formation, the data would have been different for the two cell groups, with or without mitomycin C. Therefore, DNA repair seems to have no effect on the incidence of SCE's. (18 refs)

79-4100 Correlation of Plasminogen Activator Production with Tumor Metastasis (Meeting Abstract). (Eng) Wang, B. S. (Harvard Medical Sch., Boston, MA); McLoughlin, G. A.; Richie, J. P. *Proc Am Assoc Cancer Res* 20: 161; 1979 (no refs)

79-4101 Prostaglandin D_2 Formation by Malignant Melanoma Cells Correlates Inversely with Cellular Metastatic Potential. (Eng) Fitzpatrick, F. A. (Dept. Physical and Analytical Chemistry, Upjohn Co., Kalamazoo, MI 49001); Stringfellow, D. A. *Proc Natl Acad Sci USA* 76(4): 1765-1769; 1979.

B16 malignant melanoma cell lines transformed arachidonic acid and its transient metabolite prostaglandin endoperoxide H_2 into prostaglandin D_2 . A highly metastatic line, B16 F₁₀, formed less prostaglandin D_2 compared with its moderately metastatic parent line, B16 F₁. Since platelet aggregation may be one factor involved in B16 metastasis and since prostaglandin D_2 inhibits platelet aggregation, this prostaglandin could affect the outcome of platelet-tumor interactions, which may contribute ultimately to metastasis. Arachidonic acid metabolism may be another one of the intrinsic biochemical properties of tumor cells that affects their metastasis. The results suggest that quantitative release of unusual prostaglandins must be considered in this context. (35 refs)

79-4102 Age-related Changes in Melanocytic Naevi. (Eng) Maize, J. C. (50 High St., Suite 1406, Buffalo, NY 14203); Foster, G. *Clin Exp Dermatol* 4(1): 49-58; 1979.

The nature and statistical significance of histologic changes in melanocytic nevi with advancing age were studied based on 279 nevi removed from 210 patients (140 women and 70 men). There was no sex difference in the types of nevi. Patients with Group I compound nevi were significantly older (mean, 32.81 yr) than those with Group II compound nevi (mean, 16.07 yr). Patients with flat lesions were significantly younger (mean, 29.76 yr) than those with dome-shaped (mean, 38.19 yr) and polypoid (mean, 38.08 yr) lesions; these data may indicate that younger patients are more likely to have flat pigmented lesions removed for cosmetic reasons. Patients with lesions showing stromal fibrosis (mean, 49.92 yr), fatty infiltration (mean, 54.14 yr), mucin deposition (mean, 53.69 yr), and/or neural tubes (mean, 51 yr) were significantly older than the mean of all patients with intradermal nevi (42.49 yr). Nineteen nevi had two or more age-related changes within the dermis. This was proportionately greatest in the case of nevi showing fatty infiltration. The results are consistent with the hypothesis that junctional proliferation and total cellularity of nevi decrease with age, with nevus cells within the dermis replaced by connective tissue elements including collagen, elastin, ground substance, and fat. The results also support the hypothesis that the formation of cylindrical neuroid structures represents the end stage of differentiation of nevus cells and not a source of origin of intradermal nevi. (22 refs)

See also:

- * (Rev.): 79-3601, 79-3623, 79-3624, 79-3626, 79-3635, 79-3636, 79-3643, 79-3644, 79-3645, 79-3651.
- * (Chem.): 79-3658, 79-3663, 79-3664, 79-3669, 79-3676, 79-3677, 79-3683, 79-3690, 79-3691, 79-3701, 79-3706, 79-3718, 79-3746, 79-3752, 79-3762.
- * (Phys.): 79-3765, 79-3769, 79-3771, 79-3772.
- * (Viral): 79-3796, 79-3808, 79-3820, 79-3822, 79-3851, 79-3864, 79-3893, 79-3918, 79-3937, 79-3942, 79-3944, 79-3958.
- * (Immun.): 79-3981, 79-3983, 79-3994, 79-3997, 79-4002, 79-4007, 79-4010, 79-4019.
- * (Epid.-Biom.): 79-4105, 79-4106, 79-4120, 79-4123, 79-4125, 79-4128, 79-4136, 79-4139, 79-4143, 79-4147, 79-4149, 79-4153, 79-4157, 79-4158, 79-4167, 79-4170, 79-4174, 79-4175, 79-4179.

- 79-4103 **Rising Cervical Cancer Mortality in Young New Zealand Women.** (Eng) Green, G. H. (Postgraduate Sch. Obstetrics and Gynaecology, Univ. Auckland, Auckland, New Zealand). *NZ Med J* 89(629): 89-91; 1979.

Trends in the cervical cancer mortality rate among New Zealand women since 1941 were studied. The mortality in women aged ≥ 35 yr has shown a steady decline since 1941, but there has been no sign of any acceleration of this decline since the introduction of screening in 1954. In contrast, there has been a significant rise in mortality among women aged 20-34 yr since 1960; this is the age group most heavily screened by cytology. Possible causes of the increased mortality in young women include a cohort effect, increased promiscuity, and the effect of steroidal compounds on the cervical epithelium. These findings cast doubt on the value of screening. Not only has cytology screening failed to accelerate an already present decline in cervical cancer mortality among older women, it has also failed to prevent a significant rise in mortality among younger women. (14 refs)

- 79-4104 **Retrospective Analysis of Cases of Cancer of the Uterine Cervix in Tenerife, Spain, During 1969-1975.** (Spa) Cotter Cotter, J. M. (Departamento de Tocoginecologia, Residencia Sanitaria de la Seguridad Social, "Nuestra Senora de la Candelaria", Santa Cruz de Tenerife, Spain); Outeirino Miguez, F.; Martinez Vinjoy, J. S. *Tokoginecol Pract* 37(416): 189-196; 1978.

Carcinoma of the uterine cervix was diagnosed in 54/13,784 women with gynecological and pregnancy-related diseases who were seen at one department in Tenerife, Spain, from 1969 to 1975. Forty-three of the cancer patients were 30-59 yr old. One patient was a nullipara, one a primipara, and the others were multipara. Epidermoid carcinoma was diagnosed in 43 cases, adenocarcinoma in 6; no histological data are available for the other tumors. (4 refs)

- 79-4105 **Epidemiological Study of Breast Pathology in Vizcaya.** (Spa) Usandizaga, J. M. (Facultad de Medicina, Hosp. Civil de Basurto, Bilbao, Spain); Lopez Valverde, M. *Tokoginecol Pract* 37(418): 263-268; 1978.

Of 1,100 women from Vizcaya, Spain who underwent gynecologic examinations during a 1-yr period, 40 had breast-related complaints. Twelve of the latter had felt lumps in their breast before the examination. The presence

of nodes in the breasts was confirmed clinically in 7/40 patients. The histopathological diagnosis was intracanalicular fibroadenoma in 5 cases, fibroadenoma of the breast in 1, and solid ductal carcinoma with scirrhous growth in 1. (no refs)

- 79-4106 **Risk Factors for Breast Cancer.** (Spa) Fernandez-Cid, A. (Instituto Dexeus, Paseo Bonanova 67, Barcelona-17, Spain); Dexeus, S. *Tokoginecol Pract* 37(418): 239-248; 1978.

The low-, and medium-, and high-risk factors for breast cancer are described. First childbirth beyond age 30, menarche under age 10, high social class, sterility, short-term or no breastfeeding, single status, frequent infections, hormone treatment, family history of cancer, liver diseases, and mastodynia are considered factors of slightly increased breast cancer risk. Age over 40, hyperestrogenism, obesity, diabetes, menopausal hypertension, family history of breast cancer, long-term exposure to radiation, mastopathy, nipple secretion, and immune deficiency are medium-risk factors. Alterations of scar tissue after operation for benign conditions, palpable axillary lymph nodes without suspect findings in the breast, and mastectomy are high-risk factors. Women with no risk factors should be screened for breast cancer every other year, women with low- and medium-risk factors annually, and women with high-risk factors semiannually. (14 refs)

- 79-4107 **Breast Cancer Incidence in Native and Immigrant Women in an Industrial Polish Town.** (Pol) Zemla, B. (Instytut Onkologii, ul. Wyrbrzeze Armii Czerwonej 14, 44-101 Gliwice, Poland); Kolosza, Z. *Nowotwory* 29(1): 51-58; 1979.

Breast cancer incidence among the 102,267 female residents of the Zabrze region of Poland was studied as a function of place of birth and environmental conditions during 1965-1975. A total of 251 cases of breast cancer were diagnosed during this period; the patients were aged 21-86 yr. Immigrant women accounted for 99/251 cases. The overall incidence of breast cancer was 22.4/100,000; the rate was 28/100,000 for the native women and 17.1/100,000 for the immigrants. The incidence of breast cancer in highly industrialized areas was significantly greater among the native women than among the immigrants, which shows that environmental pollution has an effect on breast cancer incidence. (6 refs)

79-4108 Malignant Breast Tumors Among Atomic Bomb Survivors, Hiroshima and Nagasaki, 1950-74. (Eng) Tokunaga, M. (Dept. Pathology, Radiation Effects Res. Foundation, Hiroshima-730, Japan); Norman, J. E.; Asano, M.; Tokuoka, S.; Ezaki, H.; Nishimori, I.; Tsuji, Y. *J Natl Cancer Inst* 62(6): 1347-1359; 1979.

During 1950-1974, 360 cases of breast cancer were identified among the 63,000 women included in the Radiation Effects Research Foundation's Extended Life-Span Study of the survivors of the Hiroshima and Nagasaki bombings. Of the 360 cancer patients, 288 were residing in one of these two cities at the time of bombing (ATB), and 108 patients received an estimated breast tissue dose of at least 10 rads. Excess risk estimates for women aged 10-19, 20-29, 30-39, and 50+ yr ATB were 7.3, 4.2, 2.6, and 4.7 cases/million women/yr/rad, respectively. Women aged 40-49 or <10 ATB showed no dose effects. Women whose breasts were irradiated did not develop mammary tumors earlier than nonirradiated controls. The relative risk estimates suggested a minimum latency period of 10 yr or less, and there was an inverse relationship between age at risk and age at irradiation. Among all women who received at least 10 rads, those irradiated before age 20 yr will experience the highest rates of breast cancer throughout their lifetimes. Risk estimates for Hiroshima and Nagasaki did not differ significantly, which indicates that the carcinogenic effects of neutrons (emitted only in Hiroshima) and gamma radiation may be equal. The distribution of histologic types of breast cancer did not vary significantly with the radiation dose. The data suggest that irradiation prior to menarche is associated with a higher risk than irradiation after menarche. (22 refs)

79-4109 Concurrent Cancer of the Esophagus in Japan. (Eng) Nakayama, K. (No affiliation given.); Abo, S. *Int Adv Surg Oncol* 2: 243-249; 1979.

Questionnaires completed by 63 Japanese institutions concerning the occurrence of esophageal cancer (EPC) with cancer of other organs were analyzed. A total of 11,732 cases of EPC were reported, 387 of which were associated with a second cancer (251 synchronously, 136 nonsynchronously). Men predominated overwhelmingly in both groups. In the synchronous group, the peak occurred at age 61-70 (129 cases), but in the nonsynchronous group, the peak occurred at age 51-60 (71 cases). Stomach cancer was the most common second cancer in the synchronous group, with 186 cases, 36 of which were in the early stage. Most (72%) of the synchronous cancer patients did not survive 1 yr. Five patients (all men in their 60's) had synchronous multiple EPC's. In the nonsynchronous group, stomach cancer was again the most common second cancer, with 62 cases, but only 2 were in the early stage. The next most common second cancer was that of the larynx (24 cases). In the nonsynchronous patients in which

EPC was the first cancer to be diagnosed, the 12-mo mortality rate was 22%, a better prognosis than that of the synchronous group. There were nine patients (1 woman, 8 men aged 55-74) with nonsimultaneous multiple tumors. One had an extremely rare combination consisting of cancer of the esophagus, lung, and stomach and leukemia. (no refs)

79-4110 Epidemiological Follow-up Study of Japanese Thorotrast Cases. (Eng) Mori, T. (Kanagawa Prefectural Coll. Nursing and Medical Technology, 50-1 Nakao-cho, Asahi-ku, Yokohama 241, Japan); Maruyama, T.; Kato, Y.; Takahashi S. *Environ Res* 18(1): 44-54; 1979.

A follow-up study was made of 243 Thorotrast exposed, war-wounded exservicemen in 1975, 30-38 yr after the Thorotrast injections. Most (224) of the men had been inoculated intravascularly. A total of 33 malignant tumors (18 in the liver) were found among the latter. In addition, 2 cases of blood diseases, and 9 cases of liver cirrhosis were found in this group. The incidence of hepatic and other malignant tumors, blood diseases, and liver cirrhosis was significantly higher than that in a control group. The total number of deaths in the Thorotrast-exposed men was also significantly higher than that in the controls. Among the remaining 19 men who had been given Thorotrast by another route, there were no deaths related to Thorotrast administration. Among the men still living, however, one sarcoma developed at the Thorotrast injection site. (6 refs)

79-4111 Statistical Analysis of Japanese Thorotrast-administered Autopsy Cases. (Eng) Mori, T. (Kanagawa Prefectural Coll. Nursing and Medical Technology, 50-1 Nakao-cho, Asahi-ku, Yokohama 241, Japan); Kato, Y.; Shimamine, T.; Watanabe, S. *Environ Res* 18(1): 231-244; 1979.

The causes of death of 144 Japanese subjects who were inoculated intravascularly with Thorotrast (TT) between 1927 and 1949 and autopsied between 1945 and 1975 were compared with those of non-TT-exposed autopsy cases in the same age bracket. The incidence of malignant hepatic tumors was >10 times higher in the TT-exposed cases. The increase was attributable to an increased incidence of hemangioendothelioma and cholangiocarcinoma of the liver. The only significant increase of liver cirrhosis in the TT group occurred in the female cases. Some of the TT-exposed cases had myeloid leukemia and erythroleukemia. There was also a significant increase in the number of cases of aplastic anemia in the TT group, but, clinically and pathologically, these were atypical. Lymphatic leukemia was not observed. No significant difference was found in the incidence of malignant lymphomas or osteosarcomas in the TT group and the controls. Lung cancer, on the other hand, showed a significantly higher incidence among the controls than among the TT group. (23 refs)

- 79-4112 Prospective Epidemiological Study of Thorotrast-exposed Patients in Portugal. (Eng) da Motta, L. C. (Natl. Sch. Public Health, Secretariat State Health, Lisbon, Portugal); da Silva Horta, J.; Tavares, M. H. *Environ Res* 18(1): 152-172; 1979.

The results of an epidemiological study of 2,436 Portuguese patients who had received Thorotrast (TT: thorium dioxide colloid) contrast medium between 1930 and 1950 and 2,086 controls are reported. Four-fifths of the former had received TT systemically. Approx half (1,244) of the TT-exposed group has been traced and followed up since then. Similarly, 924/2,096 of the control group, injected for the same purpose with nonradioactive contrast drugs, have also been traced and followed up. As of December 31, 1976, 955 of the traced TT and 656 of the traced control individuals have died. Sixteen TT-exposed individuals died as a direct result of the consequences of local granulomas that developed from drug spillage around blood vessels (cervical granulomas in 15 cases), 137 from malignant tumors [6 that developed on the edge of the local granulomas, 87 primary liver tumors (32 confirmed histologically, 18 of these being hemangioendotheliomas), 4 biliary duct carcinomas (Ca's), 8 stomach Ca's, 5 Ca's of the bronchus and lung, 2 Ca's of the larynx, 5 primary bone tumors, and 20 tumors at other sites], 23 from fatal blood dyscrasias (12 of which were leukemias, mostly acute and myeloid in type), and 27 from liver "cirrhosis" (actually, a particular form of liver fibrosis). Of the 656 control deaths, only 15 were due to malignant tumors (4 stomach Ca's, 1 Ca of the bronchus, 1 Ca of the larynx, and 1 probable primary liver tumor). There were no deaths from leukemia or other fatal blood diseases among controls, and only 6 deaths were attributed to liver cirrhosis. A statistical analysis of the data showed that the number of deaths due to malignancies (particularly primary liver, bone, bronchial, and laryngeal tumors and leukemias) and from liver cirrhosis among the TT subjects was significantly ($p > 0.05$) higher than that among controls and also higher than the number expected for the general Portuguese population. (31 refs)

- 79-4113 Thorium Dioxide and the Liver: Up-dated Clinical and Biochemical Findings. (Eng) Tavares, M. H. (Dept. Pathology, Faculty Medicine, Univ. Lisbon, Lisbon, Portugal); Saragoca, A.; Oliveira, E. A.; Rocha Oliveira, M. P.; da Silva Horta, J. *Environ Res* 18(1): 173-177; 1979.

Clinical and biochemical findings in 187 nonselected patients (89 men and 98 women, aged 22-87 yr) injected with Thorotrast 10-44 yr previously are summarized. The most frequent presenting symptoms were dyspepsia, nausea, vomiting, and pain in the right or left upper abdomen. Thirty-three patients died 3-21 mo after the onset of symptoms. A clinical diagnosis of liver tumor was confirmed histologically in 15 patients (5 hemangioendotheliomas, 8 cholangiocarcinomas, and 2 hepatomas). Hepatic fibrosis

was present in an additional 25 patients. The biochemical tests revealed low serum albumin, increased serum α_2 - and γ -globulins, and, particularly in the tumor patients, increased levels of γ -glutamyltranspeptidase and alkaline phosphatase. α -Fetoprotein was not detected in the 28 subjects tested. The incidence of abnormal biochemical findings was correlated with the time elapsed since Thorotrast injection. Almost all of the patients injected >25 yr earlier had abnormal laboratory tests, and the liver tumors appeared only in the patients injected ≥ 29 yr earlier. (6 refs)

- 79-4114 Liver Diseases in Patients Injected with ^{224}Ra . (Eng) Spiess, H. (Kinderpoliklinik, Universität München, Pettenkofer Strasse 8a, Munich, W. Germany); Mays, C. W. *Environ Res* 18(1): 55-60; 1979.

Observations of the occurrence of liver diseases in patients inoculated with ^{224}Ra , a decay product of Thorotrast, are reported. Nine-hundred patients [682 adults and 218 juveniles (1-20 yr old)] followed an av of about 20 yr since repeated injections, totaling an av of $18 \mu\text{Ci } ^{224}\text{Ra/kg}$, were given for the treatment of tuberculosis, ankylosing spondylitis, and other diseases. Although injected ^{224}Ra accumulates mainly in bone, an appreciable fraction of the 3.62-day ^{224}Ra and its daughters decay in the liver. However, the liver dose in rads in the ^{224}Ra patients is very much less than that in Thorotrast-exposed patients. Chronic liver diseases, mostly cirrhosis, were found in 20 ^{224}Ra cases, 18 of them injected as adult men. Two liver cancers occurred, one hepatic cell carcinoma and one bile duct carcinoma, but this incidence is similar to that expected. The lack of excess liver cancers in the ^{224}Ra patients is in sharp contrast to the high incidence, about 5%, reported for the Thorotrast patients. The difference is probably mainly due to the much higher liver doses in the Thorotrast patients. However, some of the difference might be due to the promoting effect of continuous α -irradiation from the Thorotrast, in contrast to the limited period of α -irradiation from the ^{224}Ra injections, averaging 6 mo in the adults and 11 mo in the juveniles. (12 refs)

- 79-4115 A Field Study in 156 Thorotrast Cases: Results of Biophysical Measurements and Clinical Examinations. (Eng) Kemmer, W. (Bundesministerium des Innern, D-5300 Bonn 1, W. Germany). *Environ Res* 18(1): 147-151; 1979.

A field study was made of 156 patients who had undergone Thorotrast (thorium dioxide suspension) angiography during 1934-1951. ThO_2 deposits were found in the reticulohematopoietic system of 116 of these patients. The concentration of ^{220}Rn in the subjects' exhaled air (the ^{224}Ra -equivalent value) corresponded to the dose of ThO_2 injected. Up to 50% of the patients had clinical symptoms of liver disease, and laboratory findings (eg, SGOT, SGPT,

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bilirubin, Hb) were in accord with the clinical examinations. (3 refs)

- 79-4116 Pathogenesis of Colonic Carcinoma. An Epidemiological Study.** (Ger) Hopker, W. W. (Pathologisches Institut der Universität, Westring 17, D-4400 Münster, W. Germany); Bohrer, M. H.; Storch, B. *Z Gastroenterol* 17(3): 162-170; 1979.

The incidence of carcinoma of the colon and rectum in the township of Karlsruhe during 1971-1975 was determined. The incidence of colon carcinoma was low in those districts of the township in which the incidence of gastric carcinoma was high. The mean age at onset of rectal carcinoma in women increased significantly from 61.5 yr in 1971 to 70.9 yr in 1975, while the average age at onset of colonic carcinoma in men decreased significantly from 67.5 yr in 1971 to 65 yr in 1975. Internationally, Karlsruhe ranks 16th in the incidence of colon cancer in men (13.2/100,000), but it has the second highest incidence of rectal carcinoma in men (18.5/100,000). (24 refs)

- 79-4117 Dietary Factors and Colorectal Cancer in Scandinavia.** (Eng) Jensen, O. M. (Unit Epidemiology and Biostatistics, International Agency Res. on Cancer, 150 cours Albert Thomas, 69008 Lyon, France); MacLennan, R. *Isr J Med Sci* 15(4): 329-334; 1979.

The role of various dietary elements, including beer, in the etiology of colorectal cancer in Scandinavia was studied. Diets of men in Copenhagen, Denmark, and rural Kuopio, Finland, were compared; the latter municipality has a four-fold lower incidence of colorectal cancer than the former. The Copenhagen subjects reported a significantly greater consumption of white-wheat breads, meat, pork, and beer, and a lower consumption of potatoes and milk. The daily intakes of protein, fiber, and total energy were significantly higher in Kuopio, that of fat was nonsignificantly higher. The results did not support the hypothesis that cancer is related to fat intake, but they did suggest that fiber deficiency plays a role in the development of colon cancer. The differences in the incidence of colon cancer in Copenhagen and Kuopio could not be explained by differences in the mouth to anus transit time. Beer consumption was 9x higher in Copenhagen than in Kuopio. However, a cohort study of Danish brewery workers showed no increased risk of colon or rectum cancer, although there were significantly increased risks for upper respiratory, digestive tract, liver, and lung cancers. Thus, there is no evidence of a causal association between beer consumption and colorectal cancer. (15 refs)

- 79-4118 Epidemiology of Colon Cancer in Scandinavia.** (Eng) Teppo, L. (Dept.

Pathology, Univ. Helsinki, SF 00290 Helsinki 29, Finland); Saxen, E. *Isr J Med Sci* 15(4): 322-328; 1979.

The occurrence of colon cancer in Finland, Denmark, Norway, and Sweden was studied. The incidence rate was highest in Denmark and lowest in Finland (<50% the rate in Denmark). The rates have increased with time in all four countries, the increase being greatest in Finland and Norway. The difference in total Danish and Finnish rates was attributable to differences in age groups >40 yr; the age-incidence pattern was identical in men and women. The ascending colon and sigmoid were the most common tumor sites in Finland, Norway, and Sweden, the former site predominating in young adults and the latter being more common in older subjects. Correlations between incidence rates were negative for cancers of the colon and stomach, but positive for cancers of the colon and breast. The general pattern of an association between high rates of colon cancer and high rates of coronary heart disease did not exist in Finland. Colon cancer in the four countries was positively correlated with socioeconomic status and meat intake and negatively correlated with intake of cereals and milk; these correlations were not statistically significant. In Finland, there was a positive correlation between the size (population) of a municipality and the incidence of colon cancer and a negative correlation between family size and colon cancer. The differences in the risk for colon cancer among the Scandinavian countries can be largely accounted for by differences in socioeconomic factors. (11 refs)

- 79-4119 Twenty-Eight Years of Continuous Follow-up of Patients Injected with Thorotrast for Cerebral Angiography.** (Eng) Faber, M. (Finsen Lab., Finsen Inst., Strandboulevarden 49, 2100 Copenhagen O, Denmark). *Environ Res* 18(1): 37-43; 1979.

A follow-up study of 1,026 Danish neurosurgical patients injected with Thorotrast (TT) between 1935 and 1946 revealed 150 deaths due to cancers other than brain tumors. Neoplasms attributed to TT included liver tumors (50 vs 0.75 expected), lung tumors (14 vs 7.49), myeloma (4 vs 0.77), and leukemia (14 vs 1.60). Among the tumors diagnosed in recent years were 4 mesotheliomas (2 pleural, 2 peritoneal), 2 extrahepatic hemangioendotheliomas, and 1 neck sarcoma that appeared after a 20-yr latency period. An analysis of yearly incidence rates showed that there was a sharp increase in TT-induced tumors during the last 10-15 yr. (6 refs)

- 79-4120 Prognosis after Treatment of Villous Adenomas of the Colon and Rectum.** (Eng) Christiansen, J. (Dept. Surgery D, Glostrup Hosp., DK-2600 Glostrup, Copenhagen, Denmark); Kirkegaard, P.; Ibsen, J. *Ann Surg* 189(4): 404-408; 1979.

Prognosis after treatment of villous adenomas (VA) of the colon and rectum was determined by reexamination of 174 Danish patients treated for VA of the colon and rectum between 1960 and 1975. Recurrent benign VA were found in 36/120 patients treated for a benign VA, and four patients treated for benign VA developed and died of cancer of the colon. The expected number of deaths from colon cancer during this period (based on mortality from colon cancer in the Danish population) was 0.49 ($p < 0.01$). The 5-yr survival rate among the 174 patients was 70% compared with 85% for the general population, and the corresponding figures at 10 yr were 57% and 66%, respectively. The 5-yr survival rate among patients treated for malignant VA was 47% vs 84% for the general population ($p < 0.01$), and the 10-yr survival rates were 38% and 58%, respectively ($p < 0.01$). The results support the importance of primary total excision of colonic polyps as a diagnostic measure; 36% of VA diagnosed on biopsy were found to be carcinomas on examination of the totally excised polyp. (20 refs)

- 79-4121 **Fecal Bacteria in South African Rural Blacks and Other Population Groups.** (Eng) Koorhof, H. J. (Dept. Microbiology, Sch. Pathology, Univ. Witwatersrand, POB 1038, Johannesburg 2000, S. Africa); Richardson, N. J.; Wall, D. M.; Moore, W. E. *Isr J Med Sci* 15(4): 335-340; 1979.

The fecal flora of 20 rural black South Africans and 22 Japanese (representing groups at low risk of carcinoma of the colon) were compared with those of 41 North Americans from a high-risk population. The counts of bacteroides, and especially bifidobacteria, were lower among the South Africans than among the North Americans or Japanese. The incidence of *Bacteroides vulgatus* and *B. distasonis* correlated positively with the risk for colon cancer, but an inverse relationship was found with *Eubacterium aerofaciens* II, *B. fragilis*, and *Escherichia coli*. The *Peptostreptococcus productus* species complex was also found in larger numbers in the high-risk group. The percentage of fecal isolates stimulated by bile was slightly higher in populations with a high-fat intake and a high risk of colon cancer. (23 refs)

- 79-4122 **Epidemiology of Chronic Intestinal Disease in Middle Africa.** (Eng) Hutt, M. S. (Geographical Pathology Unit, Dept. Morbid Anatomy, St. Thomas' Hosp. Medical Sch., London, England). *Isr J Med Sci* 15(4): 314-317; 1979.

The pattern of chronic intestinal disease in middle Africa is changing as more people adopt an urban and western way of life. Traditional patterns were high frequencies of parasitic and infectious diseases and sigmoid volvulus and low incidences of polyps and carcinoma of the colon, ap-

pendicitis, diverticulosis, ulcerative colitis, and Crohn's disease. Differences in diet and bowel physiology between rural African and western populations are pointed out. (39 refs)

- 79-4123 **Cholecystectomy and Carcinoma of the Large Intestine.** (Cze) Choluj, B. (Voljensky lazensky ustav, Karlovy Vary, Czechoslovakia). *Cesk Gastroenterol Vyz* 33(1): 13-25; 1979.

The relationship between cholecystectomy (Ch-x) and carcinoma of the large intestine (CLI) was studied in an autopsy series of 410 subjects (172 men and 238 women) who had undergone Ch-x. CLI was found in 34 of the autopsy cases (14 men and 20 women, av age 59.3 yr). The interval between Ch-x and detection of the carcinoma at autopsy averaged 9.47 yr (6.8 yr in men and 12.15 yr in women). In a control series of patients who had not undergone Ch-x, the incidence of CLI was 0.3%-1.78%. Compared with other tumors, CLI was most frequent among the Ch-x patients. In another autopsy series of 660 subjects who died of CLI, 10.7% had undergone abdominal surgery, with Ch-x being the most common procedure (4.5%). The findings indicate that there is a relationship between Ch-x and the incidence of CLI, which is linked with the elevated deoxycholic acid and lithocholic acid levels in the intestine of Ch-x patients. Therefore, subjects who have been subjected to Ch-x have an increased risk of CLI. (44 refs)

- 79-4124 **Solution of the Problem of Benzo(a)pyrene and Irritants from Mineral Oil Burning in Heavy Section Rolling Mills.** (Cze) Novacek, E. (Zavodni ustav narodniho zdravi, Vitkovice, Czechoslovakia); Maruskanicova, V. *Prac Lek* 31(3): 96-98; 1979.

A precancerous condition was found in the larynx of some nonsmoking workers at a heavy section rolling mill. Benzo(a)pyrene concentrations in the work area were 0.016-0.04 mg/m³. Benzo(a)pyrene was generated by the burning of a mineral oil lubricant. The technology was modified to eliminate the need for lubrication. (no refs)

- 79-4125 **Incidence of Carcinoma of the Gastric Stump after Resection for Peptic Ulcer.** (Cze) Pavlik, Z. (Chirurgicke oddeleni, OUNZ, 886 68 Uherske Hradiste, Czechoslovakia); Klimes, J. *Cesk Gastroenterol Vyz* 33(1): 8-12; 1979.

Of 811 patients who underwent surgery for peptic ulcer of the stomach or duodenum during 1927-1960, 20 (16 men and 14 women aged 42-85 yr, av 62.9 yr) developed carcinoma of the gastric stump 12-40 yr (av 21.2 yr) after gastric resection for gastric ulcer (12 cases) or duodenal

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ulcer (8). All 20 patients underwent a Billroth II resection for the ulcer. The incidence of gastric carcinoma in this series was 1.34%, vs 0.9% in the general population. Adenocarcinoma was diagnosed histologically in 10 cases, solid carcinoma in 4, and anaplastic carcinoma in 6. (14 refs)

- 79-4126 Primary Malignant Lymphoma of the Small Intestine in Israel. Changing Incidence with Time.** (Eng) Selzer, G. (Dept. Pathology, Chaim Sheba Medical Center, Tel Aviv, Israel); Sacks, M.; Sherman, G.; Naggan, L. *Isr J Med Sci* 15(4): 390-396; 1979.

The incidence of primary small intestinal lymphomas in Israel between 1960 and 1975 was studied. During this period, these tumors were diagnosed in 145 Jews and 36 Arabs. The mean annual incidence was 5.3/million among men and 2.9/million among women ($p < 0.001$), the rates being significantly higher among children and older adults than in teenagers or young adults. The crude annual incidence rates were significantly higher among Arabs (7.2/million) than among Jews (4.1/million). This difference was particularly great in boys aged 0-9 yr and in men aged 20-39 yr. Among Jews, the crude and age-adjusted rates were higher in those born in Northern Africa or Asia than in those born in Europe; the corresponding rates in Israel-born subjects were closest to those in the European group. The greatest differences between Jews born in North Africa or Asia and those born in Europe were seen among men and women aged 20-39 yr. The mean annual incidence of primary small intestinal lymphoma fell from 4.8/million during 1960-1967 to 3.6/million during 1968-1975. This was due to a marked decline in the rates among children and young adults, the rates in persons aged ≥ 40 yr having increased with time. The pattern of change with time suggests that environmental factors are important in the etiology of this disease in young adults. (15 refs)

- 79-4127 Patterns of Gastrointestinal Neoplasms in Israel.** (Eng) Modan, B. (Dept. Clinical Epidemiology, Chaim Sheba Medical Center, Tel-Hashomer, Israel). *Isr J Med Sci* 15(4): 301-304; 1979.

The incidence of gastrointestinal (GI) neoplasms among the major ethnic groups in Israel was compared. The ratio of the incidence rates among the European-born Israelis compared with those of Asian or African origin increased with progression down the GI tract. Gastric cancer was most prevalent among the higher socioeconomic group (European born). For colon cancer, the incidence among Jewish Israel-born women was close to that of women of Asian-African origin, whereas among men it was intermediate between the two groups. This pattern is similar to that reported for myocardial infarction. During 1960-1971, the incidence of esophageal and gastric cancers

declined, but that of rectal cancer increased. To assess the etiologic role of diet, the consumption of 243 food items was studied in 166 gastric cancer, 198 colon cancer, and 77 rectal cancer patients and matched controls. Excess starch consumption was the only differential for gastric cancer, lower fiber intake was the only differential for colon cancer, and there were no differences associated with rectal cancer. In a recent study, only a reduction in fiber intake was associated with the development of coronary heart disease. (23 refs)

- 79-4128 Primary Intestinal Lymphoma in South Africa.** (Eng) Novis, B. H. (Gastrointestinal Unit, Meir Hosp., Kfar Saba, Israel). *Isr J Med Sci* 15(4): 386-389; 1979.

All 63 adult cases of primary intestinal lymphoma seen between 1953 and 1977 at a Cape Town, South Africa, hospital were reviewed. Seventy percent of the patients with solitary lymphoma (SL) and 80% of those with immunoproliferative small intestinal disease (ISID) were mulatto. SL patients generally presented with acute or subacute intestinal obstruction, and those with ISID generally presented with malabsorption. One or more intestinal pathogens were present in 30% of the ISID group. Four of 15 ISID patients had α -heavy-chain disease (HCD). The SL's were usually of the monomorphic lymphocytic or histiocytic variety; tumors in ISID patients were usually of a mixed type. The av survival time after diagnosis was 12 mo for SL patients and 30 mo for ISID patients. Immunologic and genetic parameters were studied in two HCD and six ISID patients and their first- and second-degree relatives. IgM levels were significantly elevated among the children in the families of the two HCD patients; IgA levels were significantly reduced in one family, and IgG levels were significantly reduced in the second family. No particular pattern in the minor blood group profiles was found among the six ISID patients and their families, and the ABH secretor distribution and Pi phenotype were similar to those in the normal population. The histocompatibility antigen (HLA) gene frequency, particularly that of HLA-A9, was increased in eight ISID patients compared with the frequency in the normal mulatto population. Intestinal alkaline phosphatase was elevated in 4/6 ISID patients and in a high proportion of their relatives. Three of the six patients were of blood group B. The data suggest that genetic factors may be relevant in the pathogenesis of ISID. (7 refs)

- 79-4129 Multiple Myeloma in South African Blacks** (Letter to Editor). (Eng) Blattner, W. A. (Environmental Epidemiology Branch, NCI, Bethesda, MD 20014); Jacobson, R. J.; Shulman, G. *Lancet* 1(8122): 928-929; 1979.

A study at a hospital in which almost all malignancies occurring in the black population of Johannesburg, South Africa, are treated demonstrated that the age-adjusted rates (world standard) per 100,000 for multiple myeloma among South African blacks (7.47 in men, 5.11 in women) are similar to those reported in US blacks (7.52 and 5.17, respectively). (12 refs)

- 79-4130** A Comparison of the Mortality Rates of Various Population Groups in the Republic of South Africa. (Eng) Wyndham, C. H. (Inst. Biostatistics, 7 Esselen St., Hillbrow, Johannesburg 2001, South Africa); Irwig, L. M. *S Afr Med J* 55(20): 796-802; 1979.

Malignant neoplasm was a significant cause of death in South African whites (4.2/1,000) and blacks (5.3/1,000) only in the age group 55-64 yr. It was ranked second, after circulatory diseases. The most common malignancies in this group were tumors of the digestive organs and lung cancer. (14 refs)

- 79-4131** The Incidence of Breast Cancer in Northern Sweden During 1959-1971. (Swe) Cumberbatch, J. (Umea datacentral, Umea, Sweden); Johnsson, M.; Larsson, L. G.; Sandstrom, A. *Lakartidningen* 76(15): 1413-1416; 1979.

The age-standardized incidence rates of female breast cancer were studied in the three northernmost counties of Sweden during 1959-1971. The rate was 58.8/100,000 for the study region vs 76.3/100,000 for all of Sweden. Great variations that could not be explained by random distribution were also found within the study region. However, a general pattern could be recognized, with lower incidences being found in the less urbanized communes in the western and northern parts and higher incidences in the more urbanized eastern and southern parts. The greatest relative difference between the low- and high-incidence communes was seen in menopausal women. The age-specific incidence curve for the low-incidence communes was bimodal. From 1959 to 1971, the crude incidence increased by 3% per year and the age-standardized incidence increased by 1.5% per year. (21 refs)

- 79-4132** Lung Cancer and Residency--A Case-Referent Study on the Possible Impact of Exposure to Radon and Its Daughters in Dwellings. (Eng) Axelsson, O. (Dept. Occupational Medicine, Univ. Hospital, S-581 85 Linköping, Sweden); Edling, C.; Kling, H. *Scand J Work Environ Health* 5(1): 10-15; 1979.

To test the hypothesis that low concentrations of radon and its daughters might contribute to the development of lung

cancer, a case-control study was made of type of residence in 28 parishes in rural southern Sweden. Recently, there has been some concern in Sweden about increasing radon concentrations in dwellings due to decreased ventilation as a result of energy-saving measures. The study was confined to the rural population. Data were obtained from the records of 37 patients who died from malignant lung tumors and 178 controls who died at approx the same time from causes other than cancer. Sixty-five percent of the patients had lived in stone houses with basements (assumed to be associated with high-level exposure to radon and its daughters) or 'mixed type' houses (medium exposure), and 35% had lived in wooden houses without a basement (low-level exposure). Only 43% of the controls had lived in stone or mixed-type houses. There was a slight exposure-effect relationship over the residence exposure categories and a clear-cut difference between extreme exposure categories. Eight of the 12 male patients for whom such information was available were smokers, compared with 6/13 controls. Smoking may potentiate the effects of the radiation. The data suggest that there may be a relationship between type of residence and lung cancer in rural areas. (18 refs)

- 79-4133** Carcinogenesis by Thorotrast and Other Sources of Irradiation, Especially Other α -Emitters. (Eng) Mole, R. H. (MRC Radiobiology Unit, Harwell, Didcot, Oxfordshire OX11 0RD, England). *Environ Res* 18(1): 192-215; 1979.

During carcinogenesis by α -particles, tissue irradiation is always inhomogeneous, sometimes extremely so. Tissue damage will be focal because cells out of reach of α -tracks (often most cells) will not be irradiated at all. Since these unirradiated cells allow tissue regeneration, random decay of radionuclides increases the nonhomogeneity of the distribution of α -emitters in tissue as time progresses. Focal damage implies that cancer induction is linear with dose but, because the length of an α -track in tissue exceeds the dimensions of a single cell, linearity does not necessarily imply that malignant transformation is exclusively intracellular. Cellular inactivation (CI) of transformed cells will reduce the frequency of observed tumors and is easier to allow for in α -particle carcinogenesis than in carcinogenesis by low-LET (linear energy transfer) radiation, because after high-LET radiation there is no shoulder on the curve for retention of clonogenicity. With high-LET radiation, the dose response for carcinogenesis is sublinear. When tissue exposures are protracted, CI and tissue repopulation by cell division will proceed side-by-side throughout the exposure. Cellular repopulation will tend to neutralize the influence of CI on tumor frequency so that, as the daily dose of protracted exposure decreases, observed tumor frequency may increase and become linear with dose. Human experience of carcinogenesis by α -emitters in bone and lung provides dose responses compatible with linearity for induction, even in the case of ^{226}Ra . In bone, there may be quantitatively important age differences in

sensitivity to sarcoma induction. Hepatic carcinogenesis by Thorotrast (TT) is an unsatisfactory model for other α -emitters, because in all subjects bearing TT, the liver is structurally abnormal as a consequence of tissue damage. The linear risk coefficient for TT-induced malignant disease originating in bone marrow (mostly leukemia) and for death from bone marrow failure implies that the relative biological effectiveness for comparisons with low-LET radiation is small (1-3). This may not be unreasonable, since α -irradiation of the marrow will have been largely focal, leaving many marrow cells unaffected. However, an alternative possibility is that CI has led to a falsely low value for the risk coefficients for α -irradiation by TT. (47 refs)

- 79-4134 **Health Problems of Anaesthetists and Their Families (5 Letters to Editor).** (Eng) Doll, R. (Dept. Regius Professor Medicine, Univ. Oxford, Oxford OX2 6HE, England); Vessey, M. P.; Conway, C. M.; Vowles, M.; Pethybridge, R. J.; Brimblecombe, F.; Nunn, J.; Halsey, M.; Sturrock, J. *Br Med J* 1(6170): 1078-1079; 1979.

Five letters criticize an article in which cancer was concluded to be an occupational hazard of the anesthetist and his family. The conclusion is derived from an inappropriate comparison of cumulative rates over the subjects' life-span with national incidence rates for a single year. In fact, a study of 547 male anesthetists for 20 yr revealed only 29 deaths from cancer, vs 32 expected. The statistical significance of an increased incidence of congenital malformations in children of anesthetists is also questioned. Recent studies have failed to produce conclusive evidence that commonly used anesthetics are carcinogenic, mutagenic, or teratogenic. (11 refs)

- 79-4135 **A Study of Radioactivity and Health Status of Former Thorium Workers. Preliminary Report.** (Eng) Rundo, J. (Center Human Radiobiology, Argonne Natl. Lab., Argonne, IL 60439); Polednak, A. P.; Brues, A. M.; Lucas, H. F.; Patten, B. C.; Rowland, R. E.; Stehney, A. F. *Environ Res* 18(1): 94-100; 1979.

The late effects of inhaled thorium are being studied in a series of 4,316 workers (80% men) employed at a Th refinery from the mid-1930's to 1973. The methodology of the mortality and morbidity studies is described briefly, as are the methods and results to date of radioactivity measurements in vivo and in the exhaled breath. Of the 46 men examined so far, all but 3 had readily measurable amounts of ^{220}Rn in their breath but there was little correlation between breath activity and ^{212}Bi level in the chest. The ratio of emanating ^{224}Ra to retained ^{212}Bi ranged from 0.013 to 0.47 (median value, 0.05). A higher value would be expected if the Th had been in the lung proper, which suggests that much of it had migrated to lymph nodes. (3 refs)

- 79-4136 **Evidence of Cadmium Toxicity in a Population Living in a Zinc-mining Area. Pilot Survey of Shipham Residents.** (Eng) Carruthers, M. (Dept. Clinical Pathology, Maudsley Hosp., Denmark Hill, London SE5, England); Smith, B. *Lancet* 1(8121): 845-847; 1979.

Twenty-two of 31 residents of an English village where soil cadmium levels are high had raised blood Cd levels (≥ 9 nanomoles/liter for nonsmokers and ≥ 18 nanomoles/liter for smokers). Nine women and 10 men had treated or untreated hypertension, and 5 men and 2 women had evidence of renal tubular damage (as indicated by elevated urinary β_2 -microglobulin levels). Both conditions could be due to toxic effects of the metal. (19 refs)

- 79-4137 **Leukemia among Workers Exposed to Benzene.** (Eng) Infante, P. F. (Office Carcinogen Identification and Classification, Occupational Safety and Health Administration, Dept. Labor, 3rd & Constitution Avenue, N.W., Washington, DC 20210). *Tex Rep Biol Med* 37: 153-161; 1978.

Mortality patterns were studied among 748 workers occupationally exposed to benzene between January 1940 and December 1949 in a natural rubber cast film facility. Vital status was determined for 75% of this population. The expected number of deaths was generated from data on two control groups, US white men (Group 1) and the employees of a fibrous glass construction products facility (Group 2) during the same period. There were 9 deaths due to lymphatic and hematopoietic cancer in the study group compared with 3.4 expected. There were 7 observed leukemia deaths vs 1.39 expected based on Group 1 or 1.47 expected based on Group 2. These data show a statistically significant excess of leukemia deaths among the workers exposed to benzene. Of the leukemia cases, 4 were acute myelogenous, 2 monocytic, and 1 chronic myelogenous. One other case was identified in an employee of the study facility, but he was not included in the statistics since he began work in 1950. Benzene exposure levels were generally within 10-100 ppm. Additional data show that a commonly sold paint stripper, when used in a garage, generates up to 225 ppm benzene. This is an unacceptable hazard to the general public, especially since an alternative product that contains no benzene is available. It is concluded that benzene is leukemogenic. (4 refs)

- 79-4138 **Hodgkin's Disease in Patients with Previous Infectious Mononucleosis.** (Eng) Kvale, G. (Cancer Registry Norway, Montebello, Oslo 3, Norway); Hoiby, E. A.; Pedersen, E. *Int J Cancer* 23(5): 593-597; 1979.

The occurrence of Hodgkin's disease (HD) and other lym-

phomas in Norwegian patients with a positive reaction to the Paul-Bunnell test was investigated. Patients with a positive reaction to this test during 1961-1972 were identified at nine laboratories and matched against cases of malignant lymphoma registered in the Cancer Registry of Norway during 1961-75. Among 5,840 patients having a positive Paul-Bunnell test, 6 developed malignant lymphoma, 3 of these >1 yr after the positive test. The expected number of malignant lymphomas was 2.04. Five of the six patients had HD. The results agree with those of other epidemiological studies. There seems to be a small excess of HD among patients who have had infectious mononucleosis, but both diagnostic problems and possible confounding factors must be taken into account before a possible causal association can be considered. (34 refs)

- 79-4139 Hematological Changes in Patients Hospitalized for Nonsurgical Diseases.** (Ger) Luthy, P. (Medizinische Universitäts-Klinik, Inselspital, CH-3010 Bern, Switzerland); Straub, P. W. *Schweiz Med Wochenschr* 109(8): 270-282; 1979.

A series of 3,621 patients hospitalized for nonsurgical diseases, exclusive of leukocytosis, were investigated for hematological changes. One or more abnormal hematological changes were found in 810 patients: anemia in 613, thrombocytopenia in 211, lymphocytopenia in 107, neutropenia in 63, thrombocytosis in 44, monocytosis in 43, polycythemia in 42, eosinophilia in 39, lymphocytosis in 27, plasmacytosis in 19, and basophilia in 7. Malignant tumors were the underlying cause of anemia in 212 patients, of thrombocytopenia in 79, of neutropenia in 34, and of lymphocytopenia in 38. Hemoblastoses accounted for 36% of the 225 tumors, malignant lymphoma for 15%, and other tumors for 49%. Approx 28% of the hematological changes were due to medications (ie, anticoagulants, immunosuppressive drugs, and drugs with an ulcerogenic side effect) and to exogenous toxins (mainly alcohol and phenacetin). (59 refs)

- 79-4140 Incidence of Cancer of the Buccal Cavity in the Cantons of Geneva and Neuchatel, Especially in Men.** (Fre) Obradovic, M. (Registre Genevois des Tumeurs, Geneva, Switzerland); Roch, R.; Pellaux, S. *Rev Med Suisse Romande* 99(2): 63-65; 1979.

The incidence of cancer of the buccal cavity was studied in the cantons of Neuchatel and Geneva. The crude incidence per 100,000 subjects in the canton of Neuchatel was 16 for men and 4.9 for women during 1972-1975. The corresponding figures for the canton of Geneva during 1970-1975 were 20.9 and 5.0. The risk of cancer of the buccal cavity or pharynx was found to be 1/300 men under the age of 65 yr, and 1/200 men under the age of 75 yr. In the male population of the canton of Geneva, the incidence of cancer of the

tongue, buccal cavity, oropharynx, and hypopharynx was 3.6, 4.3, 5.7, and 4.9, respectively. In Western Europe, the incidence of cancer of the tongue, buccal cavity, oropharynx, hypopharynx, and esophagus is highest in the canton of Geneva. The use of tobacco and/or alcohol, is considered a risk factor. (4 refs)

- 79-4141 Premature Mortality Attributable to Smoking and Hazardous Drinking in Canada.** (Eng) Ouellet, B. L. (Long Range Planning Directorate, Dept. Natl. Health and Welfare, Jeanne Mance Building, Tunney's Pasture, Ottawa, Ontario K1A 0K9, Canada); Romeder, J. M.; Lance, J. M. *Am J Epidemiol* 109(4): 451-463; 1979.

All 1974 causes of death in Canada related to smoking and hazardous drinking were reviewed, and the extent of premature mortality in Canada attributable to each of these two risk factors was determined. For each cause, existing epidemiologic data were analyzed and used to determine the fraction of premature mortality that could be attributed to each factor (attributable fraction, AF). This fraction was then multiplied by the corresponding Canadian premature mortality measured in terms of deaths between ages 1 and 70 and potential years of life lost (PYLL) between ages 1 and 70, which gives a higher wt to younger deaths. In overall importance in terms of deaths or PYLL attributable to current smoking, lung cancer ranked second (2,655 deaths, AF 63.4%). In terms of fraction of total deaths and PYLL attributable to current smoking, cancer of the oral cavity and pharynx, larynx, and esophagus ranked first (AF 66%), but this joint cause represented relatively few actual deaths (563) and PYLL compared to the two leading causes, ischemic heart disease (4,531 deaths) and lung cancer. Cirrhosis of the liver ranked second (after alcoholism and alcoholic psychosis, AF 100%) in terms of the fraction of deaths (AF 68%) and PYLL attributable to hazardous drinking, but it was surpassed only by motor vehicle accidents in terms of actual numbers of attributable deaths and PYLL (1,502 vs 2,212 deaths). In terms of number of deaths and PYLL attributable to hazardous drinking, cancer of the oral cavity and pharynx, larynx, and esophagus ranked fifth (AF 21.7%). Regardless of whether premature mortality is expressed in terms of deaths or PYLL, about 18% of Canadian premature mortality is attributable to current smoking and/or drinking (with the range of possible values being 14%-22%). (34 refs)

- 79-4142 Skin Cancer Morbidity in Turkmen Republic, USSR, During 1967 and 1976.** (Rus) Meredova, G. S. (Turkmenian Res. Inst. Oncology, Ashkhabad, USSR); Nuriagdyev, S. K. *Zdravookhr Turkm* 22(9): 9-10; 1978.

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Skin cancer morbidity rates in Turkmen Republic, USSR, are reviewed. From 1967 to 1976, skin cancer was diagnosed in 2,118 patients (946 men, 1,172 women). The standardized rate (per 100,000 individuals) ranged from 12.0 in the Ashkhabad region to 5.0 in the Tashauz region. The observed difference might be due to geographical location or to variations in the amount of rainfall and sunlight. The overall morbidity rate was 1.5 times greater among the urban population than among the rural population, and the rates for men and women were 10.3 and 11.0, respectively. (no refs)

- 79-4143 Pathomorphologic Observations on the Relation Between Late Schistosomiasis Colitis and Colorectal Cancer. (Eng) Chi-yuan, C. (Dept. Pathology, Soochow Medical Coll. Hosp., Soochow, People's Republic China); Pei-yu, C.; Jen-chun, H.; Ming-chai, C. *Chin Med J [Engl]* 92(2): 113-118; 1979.

The pathology of colorectal carcinoma was studied in 289 cases in which the tumor was associated with schistosomiasis (AS) and 165 cases in which it was not associated (NS). The AS patients averaged 40.3 yr of age compared with 46.8 yr for the NS patients. There were no significant differences in gross pathology between the two groups. In the AS group, 91.6% of the tumors were well-differentiated mucoid adenocarcinomas and 8.4% were poorly differentiated adenocarcinomas and signet-ring carcinomas. In the NS group, 69.1% of the tumors were differentiated mucoid adenocarcinomas and 30.9% were poorly differentiated adenocarcinomas and signet-ring cell carcinomas. Diminutive polyps, pseudopolyps, ectopically proliferating glands, disintegrated muscularis mucosa, denudation, and multicentric carcinoma were encountered frequently in the AS group, but papillary and adenomatous polyps were most common in the NS group. Pseudopolypoid, ectopically regenerating glands, and multicentricity are thought to play an important role in the development of malignant change. (5 refs)

- 79-4144 Gastrointestinal Lymphomas in Hong Kong Chinese. (Eng) Ho, F. (Dept. Pathology, Univ. Hong Kong, Hong Kong); Gibson, J. B. *Isr J Med Sci* 15(4): 382-385; 1979.

Forty-six Chinese patients (28 men, 17 women, 1 not recorded) with primary gastrointestinal lymphoma were studied. The mean age of the patients was 44.8 yr (range, 4-87 yr). The common presenting symptoms were abdominal pain, bleeding, and a palpable mass, and most of the tumors were located in the stomach and small intestine. The most common gross lesion was a diffuse or nodular thickening of the viscus with ulceration. Thirty-one of the tumors were classified as histiocytic lymphomas. Only one patient presented with a malabsorption syndrome that was

not associated with a fistula. A diffuse histiocytic lymphoma in another patient was associated with *Schistosoma ova* in the tumor and adjacent submucosa. The findings indicate that primary lymphomas of the gastrointestinal tract in Hong Kong Chinese resemble the "western" type more than the "Mediterranean" type. (16 refs)

- 79-4145 Environmental Factors in the Etiology of Nasopharyngeal Carcinoma: Report on a Case-Control Study in Hong Kong. (Eng) Geser, A. (International Agency Res. Cancer, Lyon, France); Charnay, N.; Day, N. E.; De-The, G.; Ho, H. C. *IARC Sci Publ* 20: 213-229; 1978.

Environmental factors that may contribute to the development of nasopharyngeal carcinoma (NPC) were investigated in a case-control study of 150 NPC patients, hospital controls, normal controls, and healthy controls living in the same households as the NPC patients. Significantly more NPC patients belonged to the four lowest occupational classes than controls [$p < 0.01$, relative risk (RR) 3.17]. More NPC patients than controls adhered to Buddhism and ancestor worship ($p < 0.01$, RR 2.1) and had a history of ear or nose illness after the age of 15 yr ($p < 0.001$). Consumption of bread and canned food and the use of fennel, mustard paste, chili sauce, and Chinese wine as spices or flavorings were significantly less frequent in the patients than in controls. NPC patients also consumed green tea less frequently than controls ($p < 0.05$, RR 0.57). There was a higher proportion of cigarette smokers among male controls than among NPC patients. Seventy-five percent of the mothers of NPC patients said that they had fed their babies salted fish after weaning, compared with 53.4% of the control mothers ($p < 0.01$, RR 2.6). Multivariate analysis showed that traditional life-style and the consumption of salted fish during weaning are independent risk factors for NPC. (12 refs)

- 79-4146 Malignant Nasopharyngeal Neoplasms with Special Reference to Nasopharyngeal Carcinoma. Experience from the Nijmegen Otorhinolaryngological Clinic. (Dut) Fischer, A. J. (KNO Kliniek, Sint-Radboudziekenhuis, Philips van Leydenlaan 15, Nijmegen, Netherlands); Huygen, P. L.; van den Broek, P. *Acta Otorhinolaryngol Belg* 32(6): 717-721; 1978.

Malignant nasopharyngeal tumors were diagnosed in 63 patients over a 20-yr period. There were three times as many men as women, and 24 patients were <40 yr old. Many of the young patients had been exposed occupationally to oil vapors in workshops and garages. (no refs)

- 79-4147** Nasopharyngeal Carcinoma in Children and Adolescents in Tunisia: Clinical Aspects and the Paraneoplastic Syndrome. (Eng) Ellouz, R. (Dept. Head and Neck Surgery, Salah Azaiz Inst., Tunis, Tunisia); Cammoun, M.; Ben Attia, R.; Bahi, J. *IARC Sci Publ* 20: 115-129; 1978.

The epidemiological, clinical, and pathogenic characteristics of nasopharyngeal carcinoma (NPC) were studied in 82 Tunisian children and adolescents (aged 10-19 yr). The male:female ratio was 3.5:1. The largest percentage of patients (28.5%) were from northeastern Tunisia; the proportion of patients from the other four regions ranged from 10% to 18%. The first sign was lymph node enlargement in 83% of the cases. Well-differentiated squamous cell carcinoma did not occur in any patient, whereas poorly differentiated squamous cell carcinoma occurred in 37 patients and "nasopharyngeal" carcinoma in 39 patients; 6 patients could not be classified. The 5-yr survival rate was 32.5%, the 2-yr survival rate 37%. Twelve patients had the paraneoplastic syndrome. This syndrome, which involves hypertrophic pulmonary osteoarthropathy, periostitis, clubbing, and gynecomastia, was seen in association with pulmonary metastases in 10/12 patients. Differences in the incidence of various epidemiologic, clinical, and histological parameters in these patients in comparison with adults suggest that NPC in young people is different from that seen in adults. (22 refs)

- 79-4148** Nasopharyngeal Carcinoma and Histocompatibility Antigens. (Eng) Simons, M. J. (Immunology Res. and Training Center, Univ. Singapore, Singapore); Chan, S. H.; Wee, G. B.; Shanmugaratnam, K.; Goh, E. H.; Ho, J. H.; Chau, J. C.; Darmalingam, S.; Prasad, U.; Betuel, H.; Day, N. E.; de-The, G. *IARC Sci Publ* 20: 271-282; 1978.

The relationship of susceptibility and survival in nasopharyngeal carcinoma (NPC) to histocompatibility locus antigen (HLA) type was investigated by HLA typing 141 blood samples from newly diagnosed (ND) patients (within 2 mo), 39 samples from long-term survivors (>5 yr after diagnosis), and 238 control samples. HLA A2 occurred in 61.0% of the ND patients and 52.9% of the controls (relative risk 1.39). All occurred in only 40.4% of these patients vs 60.5% of controls (relative risk 0.44). This highly significant deficit of A11 among ND patients was related to an excess of BW17 antigen in the patients (28.4% vs 14.3% in controls, relative risk 2.38), since there was a high negative disequilibrium value between these two antigens among NPC patients. B-Sin 2 occurred in 34.0% of the ND patients and in 22.7% of controls (relative risk 1.76). Thus, in ND patients, apart from the increased risk associated with the joint occurrence of A2 and B-Sin 2, there was also an increased risk associated with BW17 and a decreased risk associated with A11. Among long-term survivors, the frequency of BW17 decreased appreciably,

whereas A2 in the absence of B-Sin 2 or BW17 increased. Among Malays, a non-Chinese group, there was an excess of a locus A blank among NPC patients; this blank was probably associated with the AW19 complex. (7 refs)

- 79-4149** Etiological Factors in Nasopharyngeal Carcinoma: A Hospital-based, Retrospective, Case-Control, Questionnaire Study. (Eng) Shanmugaratnam, K. (Dept. Pathology, Univ. Singapore, Outram Road, Singapore 3, Singapore); Tye, C. Y.; Goh, E. H.; Chia, K. B. *IARC Sci Publ* 20: 199-212; 1978.

A total of 379 Singapore Chinese patients with nasopharyngeal carcinoma (NPC) were given a questionnaire covering occupation; level of education; language medium of education; personal and family history of nasal illnesses; types of medicines used (Chinese or other); use of Chinese medicines for the nose and throat; ingestion of soya sauce, Chinese tea, cooling drinks and alcohol; cigarette smoking habit (number and duration); type of cooking fuels used; use of incense (duration and frequency); and use of antimosquito coils. The same questionnaire was given to two groups of controls: 595 patients with diseases of the ear, nose, and throat other than NPC and 1,004 patients with diseases other than cancer or otorhinolaryngeal disease. NPC patients differed significantly from both groups of controls in that they showed stronger associations with personal history of nasal illnesses, family history of nasal illnesses, use of Chinese medicines for the nose and throat, and exposure to smoke from antimosquito coils. (30 refs)

- 79-4150** Epidemiology of Malignant Tumours of the Nasopharynx in France: Retrospective and Prospective Studies. (Eng) Brugere, J. (Head and Neck Unit, Medical Section, Fondation Curie, Paris, France); Point, D.; Sancho-Garnier, H.; Schwaab, G. *IARC Sci Publ* 20: 241-249; 1978.

The results of a retrospective and a prospective epidemiological study of French patients with malignant tumors of the nasopharynx are presented. Among 641 adult patients with malignant tumors of the nasopharynx treated at two French centers between 1951 and 1976, 80% had nasopharyngeal carcinoma (NPC), and 16% had malignant lymphoma. Malignant lymphomas, embryonal sarcomas, and epidermoid carcinomas were the only malignancies observed in 54 children treated at one center between 1952 and 1973. Among the total 509 NPC patients, the male:female ratio was 3:1. Most cases were observed between the ages of 40 and 59 yr. Only 38% percent of the patients treated at the Institut Gustave Roussy and 59% of those seen at the Fondation Curie were French. A prospective study led to the collection of data on 133 new malignant nasopharyngeal tumor patients treated between

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1975 and 1976. Of these patients, only 54 were French. Most well-differentiated carcinomas (79%) occurred in Western Europeans. The male:female ratios for lymphoepitheliomas, undifferentiated carcinomas, and malignant lymphomas were 2:1, 3:1, and 1:1, respectively, but the ratio for well-differentiated carcinoma was 20:1. Patients with epidermoid carcinomas smoked twice as much as patients with lymphoepitheliomas. (1 ref)

- 79-4151 Epidemiology of Asbestos Mesotheliomas in the Saint-Etienne Region of France. A Report of 35 Cases.** (Fre) Emonot, A. (Hopital Bellevue, F 42100 Saint-Etienne, France); Marquet, M.; Baril, A.; Berardj; Brailion *Ann Med Interne (Paris)* 130(2): 71-74; 1979.

Thirty-five cases of malignant pleural mesothelioma were diagnosed in the Saint-Etienne region of France. Occupational exposure to asbestos was found in only 6 cases (metallurgical plant workers). Electron microscope investigations revealed asbestos fibers in five tumors from patients who had no history of occupational exposure to asbestos: chrysotile was found in three, mostly amphibole in another, chrysotile + amphibole at an approx equal ratio in the fifth patient. Twenty-eight of the 35 patients lived in a highly industrialized and urbanized area with a population of 350,000, corresponding to an incidence of 20-40/10,000. Seven patients lived in a rural area on the periphery of this area (corresponding to an incidence of 2-4/10,000). The findings indicate the significance of nonoccupational exposure to asbestos originating from industrial emissions, the building industry, and automobiles. (4 refs)

- 79-4152 Differences in EBV Antibody Titres of Patients with Nasopharyngeal Carcinoma Originating from High, Intermediate and Low Incidence Areas.** (Eng) de The, G. (Unit Biological Carcinogenesis, International Agency Res. Cancer, Lyon, France); Lavoue, M. F.; Muenz, L. *IARC Sci Publ* 20: 471-481; 1978.

The association between Epstein-Barr virus (EBV) and nasopharyngeal carcinoma (NPC) was compared in different geographical areas. Differences in serological reactivities of NPC patients from high-, intermediate-, and low-incidence areas to EBV-determined antigens were assessed using 288 NPC sera from Hong Kong, Singapore, Nairobi, Tunis, Paris, Marseille, Stockholm, and Los Angeles, together with sera from patients with ear, nose, and throat tumors other than NPC and from normal individuals from the same areas. The sera were tested "blind" with the same batches of antigen. Important differences (up to 3-fold) in the geometric mean titers of antibodies directed against virus capsid antigen, early antigen, and Epstein-Barr nuclear antigen were observed between different ethnic groups. Although the stages of the disease varied somewhat between groups, the main cause of

the variations appeared to be due to the socioeconomic environment of the patients. Apart from these variations and regardless of geographical or ethnic origins, NPC was consistently found to be associated with an active infection (or reactivation) by Epstein-Barr virus, which was not the case for patients with other tumors or for normal individuals from the same areas or of the same ethnic groups. (21 refs)

- 79-4153 Genetic Components in Susceptibility to Nasopharyngeal Carcinoma.** (Eng) Kirk, R. L. (John Curtin Sch. Medical Res., Canberra, Australia); Blake, N. M.; Serjeantson, S.; Simons, M. J.; Chan, S. H. *IARC Sci Publ* 20: 283-297; 1978.

To investigate the possibility that genetic susceptibility may be involved in the increased frequency of nasopharyngeal carcinoma (NPC) in the Chinese, a survey of 25 RBC enzyme and 5 serum protein systems was carried out in Chinese patients and controls living in Singapore. In 4/11 systems that showed a variation there were gene frequency differences of 4% or more between the NPC patients and controls. For the 6-phosphogluconate dehydrogenase (PGD) and transferrin systems, these differences were significant. Multivariate analysis, using genetic distance statistics, showed a significant difference between NPC patients and controls that was also evident when patients and controls were separated according to dialect groups. This study indicates that etiological factors resulting in clinically and histologically confirmed NPC operate on a genetically distinct subpopulation of Chinese in Singapore. (17 refs)

- 79-4154 Environmental Backgrounds of Young Chinese Nasopharyngeal Carcinoma Patients.** (Eng) Anderson, E. N. (Dept. Anthropology, Univ. California, Riverside, CA 92502); Anderson, M. L.; Ho, H. C. *IARC Sci Publ* 20: 231-239; 1978.

Environmental factors that may have been responsible for nasopharyngeal carcinoma (NPC) in 24 Chinese patients who were <25 yr old at the time of diagnosis were investigated. Twenty-two patients were questioned in the presence of their families, and two were interviewed briefly in the hospital. Questioning concerned the environmental background during infancy and early childhood. Analysis of the results eliminated exposure to household inhalants, contaminants in the air, medicines, spices, fresh foods, and soya sauce as likely factors in the genesis of NPC. Most subjects came from relatively poor backgrounds, with only five being from middle-class backgrounds. All families felt that vegetables and fruits were bad for infants and that meat and fish were good; the patients had been fed accordingly. Fish was the chief source of animal protein. All patients had eaten salted fish and had received it as one of their first solid foods. Most patients had a history of poor health, inactivity, strong food prejudices, a preference for

relatively bland foods, and poor infant nutrition. It appears that consumption of salted fish and a vitamin C deficiency in early childhood could be important environmental factors in the development of NPC and that a certain personality type may be associated with an increased risk. (6 refs)

- 79-4155 Germinal Cell Tumors of the Testis after Orchiopexy.** (Eng) Martin, D. C. (Div. Urology, Dept. Surgery, Univ. California at Irvine, Irvine, CA). *J Urol* 121(4): 422-424; 1979.

To determine whether early orchiopexy (before the time when the undescended testis lags behind its scrotal mate in the process of maturation) might reduce subsequent malignant degeneration of an undescended testis, 220 literature cases of germinal cell tumor of the testis that had occurred after orchiopexy were reviewed. Of these cases, 97 were accurately documented with regard to scrotal placement of the undescended testis and the subsequent tumor. Only 6 cases were reported after orchiopexy in children <10 yr old. Since the number of patients at risk is unknown, the possible protection of early orchiopexy against subsequent malignant degeneration cannot be established. The small number of cases reported after orchiopexy in children <10 yr old is encouraging. The accumulated reports of germinal cell tumors of the testes after orchiopexy in patients between 10 and 20 yr old militate against such a procedure in patients with a unilateral undescended testis. (44 refs)

- 79-4156 Risk Factors for Cancer of the Testis in Young Men.** (Eng) Henderson, B. E. (Dept. Community and Family Medicine, Univ. Southern California Sch. Medicine, 2025 Zonal Ave., Los Angeles, CA 90033); Benton, B.; Jing, J.; Yu, M. C.; Pike, M. C. *Int J Cancer* 23(5): 598-602; 1979.

A case-control study of testis cancer was conducted in 131 men under age 40 to investigate antecedent risk factors, including events during prenatal life. Of the 125 patients for whom the information was available, 69 had right-sided, 55 had left-sided and 1 had bilateral cancer. Fifty-eight patients had embryonal cell carcinomas, 53 seminomas, 13 teratomas, 5 choriocarcinomas, and 2 interstitial cell carcinomas. Risk ratios were ≥ 2.0 for a history of kidney infection and hernia repair, as reported on the patients' questionnaires. According to the mothers' questionnaires, 10 patients were born with an undescended testis, vs only 2 controls. Two other risk factors were uncovered: (1) six patient mothers received hormones during pregnancy, compared with only one control mother, to give a risk factor of 5.0; (2) eight patient mothers but only two control mothers reported excessive nausea as a complication of pregnancy, for which all but two received medication, to give a risk factor of 4.00. The risk of cryptorchidism, excessive

nausea, and hormone use are combined into a single hypothesis: a major risk factor for testis cancer (and cryptorchidism) is a relative excess of certain hormones (estrogen and, perhaps, progesterone) at the time of testicular differentiation (7th week). (20 refs)

- 79-4157 The Pathogenesis of Ovarian Neoplasia.** (Eng) Woodruff, J. D. (Dept. Obstetrics and Gynecology, Johns Hopkins Hosp., Baltimore, MD 21205). *Johns Hopkins Med J* 144(4): 117-120; 1979.

Several aspects of intraabdominal neoplasia involving the ovary, a disease that causes 11,000 deaths/yr in the US, are reviewed. It is the third commonest primary malignancy of the female genital canal, but it kills more women than cervical and endometrial cancer combined. Current statistics demonstrate the prevalence of ovarian cancer in industrialized societies, which suggests that carcinogens have been introduced into industrialized urban communities. During 1973-1977, 240 cases of ovarian malignancy were recorded in one laboratory, but no malignant epithelial tumors of the testes were documented. One possible explanation for this difference in epithelial neoplasia in the male and female gonad may be that the recurrent breaks in the celomic epithelium that occur during ovulation subject the ovary to greater risk for the development of neoplasia, in contrast to the undisturbed mesothelial surface of the testes. In the tumors involving or arising in the ovary, those classically called ovarian cancer follow the pattern of classic mesothelioma or of the differentiated lesions unique to the ovary. Experimental data have documented the transfer of material from the cervix into the abdominal cavity within 25 min through the patent fallopian tube. It is possible that some agent enters the peritoneal cavity through the fallopian tube, irritates the pelvic peritoneum, produces proliferation, and, with an added unknown factor, results in the development of malignancy. (12 refs)

- 79-4158 Gastrointestinal Mucosa and Primary Gastrointestinal Lymphoma.** (Eng) Borochovit, D. (Dept. Pathology, Medical Coll. Virginia, Richmond, VA 23298); Dutz, W.; Kohout, E.; Vessal, K. *Isr J Med Sci* 15(4): 397-404; 1979.

The primary gastrointestinal lymphomas occurring during 1960-1974 in patients at two institutions, one in Shiraz, Iran, and the other in Richmond, VA, were compared. The number of patients was 27 and 29, respectively. Duodenojejunal lymphoma (18 cases) occurred only among the Iranian patients. It was associated with Grade 3 or 4 mucosal atrophy and with a diffuse plasma cell infiltration; all cases showed lymph follicles touching on the muscularis mucosa. The lymphomas developed in multiple separate foci from atypical follicles and pseudofollicles, and most were of the lymphocytic plasmacytic or immunoblastic type. Serum

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α -heavy-chain levels were elevated in 10 patients, IgA was elevated in 6 patients, and IgG was elevated in 2 patients, with considerable overlap. The mean age of these patients was 24.5 yr. Ileocecal, ileal, and colonic lymphoma developed in reexisting lymph plaques of the gut. These tumors were predominantly small lymphocytic and immunoblastic types. They occurred in the first or fifth decades of life in the American patients (8) and in the third decade in the Iranian patients (5). Gastric lymphomas occurred more commonly in the American patients (21) than the Iranian patients (4), the mean age being 55.18 yr for both. They were mostly of the lymphocytic plasma cell or centrocytic centroblastic types, and they were associated with a diffuse plasma cell infiltration and lymph follicle formation. Nearly all were associated with a long-standing history of alcoholism or upper abdominal disease. Hypotheses for the pathogenesis of the different types of gastrointestinal lymphomas are presented. (51 refs)

- 79-4159 **Cancer of the Pancreas in Olmsted County, Minnesota, 1935-1974.** (Eng) Maruchi, N. (Dept. Health Admin., Tokyo Univ., Tokyo, Japan); Brian, D.; Ludwig, J.; Elveback, L. R.; Kurland, L. T. *Mayo Clin Proc* 54(4): 245-249; 1979.

All confirmed cases of pancreatic carcinoma that occurred in residents of Olmsted County, Minnesota, from 1935 through 1974 were identified and reviewed. The age-adjusted incidence rates were 7.4 for men and 3.5 for women per 100,000 population. Rates increased with age for both sexes and increased slightly over the 40-yr study period with lower incidence rates being observed among rural inhabitants in the first two decades. At the time of diagnosis, almost 75% of the patients were ≥ 60 yr of age, and adenocarcinoma was histologically identified in 92% of the patients. The 1-yr survival was 11%, and all patients died within 3 yr of the initial diagnosis. An association between pancreatic carcinoma and diabetes was noted. There did not appear to be a clear association with cholelithiasis or chronic pancreatitis. There was a high percentage (20%) of multiple primary carcinomas and an overrepresentation of metal workers among the patients. (34 refs)

- 79-4160 **Epidemiology of Thorotrast-induced Hepatic Angiosarcoma in the United States.** (Eng) Falk, H. (Chronic Diseases Div., Bureau Epidemiology, Center Disease Control, US Dept. Health, Education and Welfare, Public Health Service, Atlanta, GA 30333); Telles, N. C.; Ishak, K. G.; Thomas, L. B.; Popper, H. *Environ Res* 18(1): 65-73; 1979.

Twenty-six cases of Thorotrast (TT)-induced hepatic angiosarcoma (HAS) were identified in an epidemiologic investigation of HAS's occurring in the US during 1964-1974. TT was administered to the 26 patients (19 men, 7

women) during 1931-1953, with 68% of the patients being injected in the 1940's. The mean age at the time of exposure was 28.5 yr, and the mean age at death was 54.5 yr. Indications for TT studies were carotid arteriography (14 cases), hepatolienography (9), femoral arteriography (2), and phlebography (1). Information on the TT dose administered was available for 14 patients, 9 of whom received a mean dose of 39.9 ml for carotid angiography, 4 a mean dose of 61.8 ml for hepatolienography, and 1 a dose of 10-20 ml for a femoral arteriogram. Four patients had a history of preexisting liver disease, two had hemolytic anemia, and one had arteriovenous malformations. None of the patients had a history of exposure to vinyl chloride, but two had been exposed to arsenic and two to iron dust. An additional two patients were chronic alcoholics. These additional risk factors may have potentiated the effects of TT. (35 refs)

- 79-4161 **Estimated Risk of Liver Cancer Due to α Emitters and β - α -emitting Parent-Daughter Chains: An Application of Thorotrast Data.** (Eng) Nelson, N. S. (US Environmental Protection Agency, Office Radiation Programs, 401 M St., S.W., Washington, DC 20460); Ellett, W. H.; Cook, J. R.; Hodge, F. A. *Environ Res* 18(1): 101-114; 1979.

Thorotrast (ThO₂ colloid) dose-effect relationships were used to estimate the risk of liver cancer due to chronic exposure to transuranium elements (eg, ²³⁸Pu, ²³⁹Pu, ²⁴⁰Pu, ²⁴¹Pu, ²⁴¹Am, ²⁴²Cm, and ²⁴⁴Cm). Absolute and relative risk models for liver cancer are discussed in relation to changes in parameters such as latent period, age sensitivity, and plateau period. Uncertainty as to childhood sensitivity introduces more variation in health effects estimates for radiation exposure than that introduced by selection of the risk model or other parameters. Thus, the ThO₂ data on liver carcinoma should be refined further. Identification of the latent period from exposure to first development of cancer and quantification of the sensitivity of children relative to adults are of primary importance. If the ThO₂ data could be analyzed to provide age-specific data on cancer risk, it might be possible to determine whether the absolute or the relative risk model is more appropriate. (8 refs)

- 79-4162 **Association Between Hepatobiliary Cancer and Typhoid Carrier State.** (Eng) Welton, J. C. (Bureau Preventable Diseases, New York City Dept. Health, New York, NY 10013); Marr, J. S.; Friedman, S. M. *Lancet* 1(8120): 791-794; 1979.

A case-control study of deceased typhoid carriers registered by the New York City Health Department between 1922 and 1975 was conducted to test for an association between the typhoid carrier state and death due to hepatobiliary

cancer (HBC). Each of the 471 carriers was matched with 2 controls for sex, age at death, year of death, borough in which the carrier died, and place of birth. Of the 471 carriers, 103 died from cancer, and 28 of the cancer deaths were due to HBC. In the control group, there were 146 cancer deaths, 9 of which were due to HBC. This difference in the incidence of HBC was significant. The findings are consistent with the hypothesis that cancer of the liver may be due to a reflux of altered bile into bile canaliculi or to chronic infection of intrahepatic biliary passages with *Salmonella typhi*. An alternative explanation consistent with these findings is that carriers were treated with agents that were later identified as being carcinogenic. However, since the proportion of carriers who died of HBC and were registered before 1940 is similar to that registered after 1940, several years after such treatment was discontinued, it is unlikely that early carcinogenic treatment is a significant factor in these cancers. (28 refs)

- 79-4163 Epidemiologic Considerations in the Design of Toxicologic Studies: An Approach to Risk Assessment in Humans.** (Eng) Woods, J. S. (Battelle Human Affairs Res. Centers, 4000 N.E. 41st St., Seattle, WA 98105). *Fed Proc* 38(5): 1891-1896; 1979.

A six-step procedure for including epidemiologic considerations in the design and analysis of laboratory studies designed to estimate risk of toxicity in humans during low-level exposure to environmental chemicals is described. Epidemiologic data are first used to identify the incidence of toxicity associated with high-level exposure of humans to a particular agent. This information is used to design chronic toxicity studies in animals that establish a dose-response relationship for that substance. Clinical, epidemiologic, and laboratory research data are then used to adjust for differences in major biological determinants of responsiveness between test animals and humans, and biostatistical procedures are used to describe the nature of the dose-response relationship in the low-dose region where community exposure occurs. Human incidence of toxicity under prevailing environmental conditions is then estimated from the adjusted animal model. Finally, epidemiologic studies are conducted to confirm (or deny) the validity of the predictive model. Vinyl chloride (VC)-induced hepatic angiosarcoma is used to show how epidemiologic data might be useful in the experimental assessment of toxicity risk in human populations. Despite numerous uncertainties and assumptions inherent in the procedure, the VC example suggests the importance of a combined epidemiologic-toxicologic approach to risk assessment in humans and to setting exposure limitations for toxic agents in the environment. (30 refs)

- 79-4164 Epidemiologic Studies of Gastric Carcinoma: Comparison Between Iranians and Two Racial**

Groups in the USA. (Eng) Dutz, W. (Dept. Pathology, Medical Coll. Virginia, Richmond, VA); Kohout, E.; Vessal, K. *Isr J Med Sci* 15(4): 410-413; 1979.

The patterns of gastric carcinoma among 226 black and white patients in Richmond, Virginia, and 75 patients from Shiraz, Iran were studied, and the results were compared with data from Colombia. The ratio of intestinal to diffuse carcinoma was higher for the indigent population in Shiraz (2.07) than for a comparable population in Colombia (1.52). The ratio for the wealthy population of Shiraz (0.8) was comparable to that of a similar group in Colombia and slightly higher than that for private white patients in Richmond. Among the Richmond patients, the ratio was consistently higher for blacks than whites, reflecting the generally poorer socioeconomic, nutritional, and general-health background of the former. A comparison of age groups showed a steady, almost linear, progression from the Shiraz indigent group (the youngest) to the Colombia high- and low-risk groups, to the Richmond indigent population, and to the Richmond private patients (the oldest). The age differences between the Richmond indigent white and black populations was not significant. (34 refs)

- 79-4165 Use of Artificial Sweeteners by Cancer Patients.** (Eng) Morrison, A. S. (Boston Collaborative Drug Surveillance Program, Boston Univ. Medical Center, 400 Totten Pond Road, Waltham, MA 02154). *J Natl Cancer Inst* 62(6): 1397-1399; 1979.

Current daily use of artificial sweeteners (AS) and diet drinks was determined for 1,862 adult cancer patients from seven countries and for 10,874 control patients hospitalized for other conditions considered unrelated to the use of these substances. For all cancer sites combined, the age-standardized proportion of AS users was 2.8% for men and 6.0% for women; the percentages among controls were 6.1% for men and 9.5% for women. There were no AS users with cancer of the bladder, ureter, or renal pelvis and no male users with cancer of the stomach. However, 15.7% of the women with stomach cancer were AS users. Adjustment for country reduced but did not eliminate the case-control differences, and the inverse association between AS and cancer was not substantially changed by adjustment for smoking habits. The age/sex/country-standardized incidence of cancer among AS users was estimated at 1.0, based on interview of 455 cancer patients during their initial hospitalization for the disease. The age-standardized percentages of daily users of diet drinks were 0.6% and 0.8% among male cancer patients and controls, respectively, and 1.4% and 1.0% among female cancer patients and controls. The data do not support an overall positive association between AS and cancer. (22 refs)

- 79-4166 Heart Disease Risk Factors and Hormone Use in Postmenopausal Women.** (Eng) Barrett-

Connor, E. (Dept. Community Medicine, M-007, Univ. California, San Diego, CA 92093); Brown, V.; Turner, J.; Austin, M.; Criqui, M. H. *JAMA* (20): 2167-2169; 1979.

A population of 1,496 women, 55-74 yr old, was studied for the distribution of heart disease risk factors in the presence or absence of postmenopausal estrogen (PME) usage. Thirty-nine percent were PME users, with peak hormone consumption (55%) occurring in the age group 55-59 yr. The users had significantly lower levels of plasma cholesterol and higher levels of plasma triglycerides than nonusers. Blood pressure and fasting plasma glucose concentration tended to be lower among users. However, the confirmed benefit of PME use, which must be weighted against the confirmed risk of cancer, does not yet include the prevention of postmenopausal atherogenesis. Further studies are needed before final recommendations about PME use are made. However, the annual risk of estrogen-associated endometrial cancer deaths is <1:1,000, whereas the risk of cardiovascular death after age 55 is >10:1,000, so that only a small benefit would be required to balance the carcinogenic risk of PME use. (20 refs)

79-4167 Health Effects of Vinyl Chloride. (Eng) Tamburro, C. H. (Digestive Diseases and Nutrition Section, Sch. Medicine, Univ. Louisville, 511 South Floyd Street, Room 535, MDR Building, Louisville, KY 40201). *Tex Rep Biol Med* 37: 126-144; 1978.

The health effects of vinyl chloride (VC) are reviewed, and a medical surveillance system for detecting and following its effects is described. An early finding in exposed humans and animals is activation of morphological changes in the sinusoidal lining cells of the liver without evidence of hepatocellular toxicity. With progressive exposure to VC there is an increase in atypia of the sinusoidal lining cells, progressive worsening of the focal sinusoidal dilatation, and malignant transformation of the sinusoidal lining cells. Most data suggest that it is the endothelial lining cell that becomes malignant and forms the angiosarcoma. Peliosis hepatis is often seen in nontumorous portions of the liver; whether this is a precarcinogenic or preangiosarcomatous lesion in VC workers is under study. Preliminary epidemiological and histological data suggest that the frequency of lung cancer is increased among VC workers. Two cases are reported in which the duration and degree of exposure to VC were almost identical, yet one man developed primary hepatocellular carcinoma and the other angiosarcoma. The man in whom hepatocellular carcinoma developed had a history of extensive alcohol consumption, which may have played a role in the inability of the hepatocytes to detoxify VC metabolites. A prospective medical surveillance system has been designed based on classification of industrial environments into three categories according to the level of possible carcinogens. (10 refs)

79-4168 A Study of Lobular Carcinoma of the Breast Based on the Third National Cancer Survey in the United States of America. (Eng) Henson, D. (Lab. Pathology and Biometry Branch, NCI, Bethesda, MD 20014); Tarone, R. *Tumori* 65(2): 133-142; 1979.

Cases of in situ and infiltrating lobular carcinoma of the breast reported in the Third National Cancer Survey were reviewed according to age, sex, race, and geographic distribution. The age-specific incidence rates indicate that there are two peak periods of risk for invasive lobular carcinoma, the first occurring at age 40-50 yr and the second after age 65. The incidence rates for lobular carcinoma were compared with those for intraductal and infiltrating ductal carcinoma reported in the Third National Cancer Survey and with those for total breast cancer in the Miyagi prefecture of Japan. The results indicate that factors that influence geographic variations in breast cancer play important roles in the etiology of infiltrating ductal carcinoma, but probably have little effect upon the incidence of lobular carcinoma and intraductal carcinoma. The limitations of large histological epidemiological surveys are presented. (15 refs)

79-4169 The Influence of Sex of Progeny on the History of Breast Cancer (Meeting Abstract). (Eng) Juret, P. (Centre Francois Baclesse, Route de Lion-sur-mer, 14021 Caen Cedex, France); Couette, J. E.; Delozier, T.; Leplat, G. In: *Medical Oncology. Abstracts of the 4th Annual Meeting of the Medical Oncology Society and the Bi-annual Meeting of the Immunology and Immunotherapy Group, Nice, France, December 2-4, 1978*. (New York: Springer-Verlag): 29 pp.; p. 16; 1978 (1 ref)

79-4170 Occurrence of Mammary Neoplasms in Bitches in Relation to Breed, Age, Tumor Type, and Geographical Region from Which Reported. (Eng) Priester, W. A. (Dept. Epidemiology and Preventive Medicine, Univ. California Sch. Veterinary Medicine, Davis, CA 95616). *J Small Anim Pract* 20(1): 1-11; 1979.

Mammary neoplasms in bitches reported by 14 veterinary schools in the US and Canada were analyzed by type, breed and age of the bitches, and geographical region. Of the total 2,075 microscopically confirmed primary mammary gland tumors, 1,187 were malignant, 557 benign, and 331 in the malignancy-not-determined (MND) category. Adenocarcinoma was the most frequent type of malignant tumor (752), followed by mixed tumors (293), and mastosarcoma (a malignant mast-cell tumor; 23). The most frequent benign tumors were mixed tumors (312) and adenoma (122). There were 12 breeds at significantly high risk for one or more of the major mammary tumor types, and 7 of these were hunting breeds. Mixed breeds were at low risk for all tumor types and collies for benign

neoplasms. The curves for age at first diagnosis of mammary adenocarcinomas, malignant mixed tumors, and benign tumors were similar in shape, showing an increase with age. For these three major tumor types, neutered bitches had one-third the risk observed in intact bitches. Benign tumors were much more frequently diagnosed in the South and in the West than in the East North Central and West North Central regions. Otherwise, the age-adjusted rates for the main categories of mammary neoplasms for the four regions showed no clear indication of geographical trends or large differences. (19 refs)

- 79-4171 **Hair Dye Use and Breast Cancer** (2 Letters to Editor). (Eng) Burnett C. M. (Clairol, Inc., 2 Blachley Road, Stamford, CT 06902); Menkart, J.; Shore, R. E.; Pasternack, B. S. *J Natl Cancer Inst* 62(6): 1327-1328; 1979.

A report implicating hair dye in the development of breast cancer is criticized. The large range of dye concentration between dark and light shades was ignored, and the interpretation of another report concerning the relationship between breast cancer and hair dye use was incorrect. It is also pointed out that a risk analysis performed with the use of NCI bioassay data on 2,4-diaminoanisole demonstrated that the yearly probability of an av hair coloring user's getting cancer due to this ingredient was 1 in 160,000,000. These claims are disputed in an accompanying letter from the authors of the original article. (4 refs)

- 79-4172 **Causes of Death among Laundry and Dry Cleaning Workers.** (Eng) Blair, A. (Environmental Epidemiology Branch, NCI, NIH, Bethesda, MD 20014); Decoufle, P.; Grauman, D. *Am J Public Health* 69(5): 508-511; 1979.

Causes of death among 330 deceased laundry and drycleaning workers were compared with those in the general population. There were 87 deaths from cancer, vs only 67.9 expected. Cancers of the lung, cervix, uterus, and skin contributed to the excess of cancer deaths, and slight excesses of leukemia and liver cancers were noted. There was a deficit of breast cancers among the study population. The increase in cancer deaths was apparent for male and female whites and nonwhites, and in each group there was a lower than expected frequency of deaths from circulatory disease. In general, the length of membership in the Laundry, Dry Cleaning, and Dye House Workers' International Union was similar for those who died of cancer and those who died of other causes. The increased proportion of cancer deaths among these laundry and drycleaning workers suggests an elevated risk resulting from exposure to drycleaning fluids (carbon tetrachloride, trichloroethylene, and tetrachloroethylene). (24 refs)

- 79-4173 **Melanomas, A Caucasian Problem?** (Eng) Allison, S. D. (John A. Burns Sch. Medicine, Univ. Hawaii, Hawaii, HI); Burch, T. A. *Hawaii Med J* 38(2): 39-41; 1979.

Deaths due to melanoma in Hawaii were studied based on data from death certificates and from the Hawaii Tumor Registry. Melanomas were most common in Caucasians, but they affected all major races. Not every black lesion is a melanoma. Three positive signs of melanoma should be looked for in a pigmented lesion: variegated color, with shades of blue being most ominous; an irregular border; and irregular elevations on the surface. Itching and tenderness in a mole may also suggest melanoma. The ideal biopsy is an excision of the entire lesion, which permits identification of the type of lesion and the depth of invasion. There is no evidence that incisional biopsy leads to metastasis. Melanomas may occur anywhere from head to foot and in all orifices. The data do not support the frequent suggestion that sun exposure plays a role in the development of melanomas. The greatest number of deaths due to melanoma occur in midlife, and the rates increase sharply with increasing age. Although death rates are increasing in Hawaii, this increase is occurring only among Caucasians, particularly Caucasian men. (10 refs)

- 79-4174 **Cancer in Families with Xeroderma Pigmentosum.** (Eng) Swift, M. (Biological Sciences Res. Center, 220 H, Univ. North Carolina, Chapel Hill, NC 27514); Chase, C. *J Natl Cancer Inst* 62(6): 1415-1421; 1979.

The incidence of skin and other cancers in the living and deceased relatives of 31 patients with xeroderma pigmentosum (XP) was studied. The proportion of adult (over 30 yr) blood relatives with nonmelanoma skin cancer (30/1,046) was greater than that of the spouse controls (11/855; $p < 0.01$). The highest proportion of affected blood relatives was among those with a 0.67 or 1.0 probability of being heterozygous for an XP gene. The difference between blood relatives and spouse controls was greatest among those living in the southernmost states ($p < 0.005$). Skin cancer was more common among relatives and spouses with outdoor occupations than among those with other jobs, and it was more common among relatives with outdoor occupations than among spouses similarly employed. Although the 30 relatives with nonmelanoma skin cancers belonged to 13 families, they were concentrated in 4 families. In these families, the difference in incidence between blood relatives (20/219) and spouse controls 81/164) was striking ($p < 0.002$). Malignant melanomas occurred in three spouses and no blood relatives. Deaths from lung cancer among relatives were greater than expected (17 vs 8.8; $p < 0.005$), as were deaths from gastric cancer among female relatives (8 vs 2.3; $p < 0.05$); however, the latter finding may have been an artifact. There was a nonsignificant excess of deaths from

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prostate cancer among the XP relatives. The data suggest that heterozygosity for XP genes may predispose to skin cancer, particularly in individuals exposed to substantial amounts of sunlight. (16 refs)

- 79-4175 **A Clinicopathologic Study of Oral Leukoplakia with Emphasis on the Keratinization Pattern.** (Eng) Platka, M. A. (Sch. of Dentistry, Dept. Oral Pathology, Georgetown Univ., Washington, DC 20007). *J Can Dent Assoc* 45(3): 107-115; 1979.

The clinical and histologic features of intraoral leukoplakia (LP) are reviewed. LP may occur anywhere in the oral cavity, involvement may be uni- or multifocal, and the lesions may be flat, elevated, fissured, ulcerated, or erythematous. The male:female ratio is rapidly approaching 1:1. The etiology is obscure, but chronic local irritation, alcohol, tobacco, accumulated actinic radiation, aging, and, possibly, *Candida albicans* infection are implicated. The pathologic process involves the formation of a white patch through a thickening of the keratin layer (hyperorthokeratosis) and/or the prickle cell layer (acanthosis); parakeratosis, which involves the presence of a keratin layer with nuclear remnants, occurs fairly frequently. LP lesions may or may not show variable degrees of dysplasia that may be reversible or irreversible. The irreversible lesions exhibit a potential for malignant transformation. Most of the lesions that become malignant do so within the first year. LP, especially speckled LP, is preinvasive; in addition, erythroplakia is often an invasive carcinoma. A study of 8,162 oral biopsy specimens revealed 559 benign LP lesions, 91 of which showed some degree of epithelial dysplasia (114 could not be analyzed meaningfully). Benign LP occurred most often in persons aged 51-60 yr, dysplastic LP in those aged 61-70 yr. More than half of the dysplastic lesions occurred on the ventral surface of the tongue and the floor of the mouth; 71% occurred on the tongue in women and on the lower lip in men. In a significant number of dysplastic lesions, the epithelial surface was covered by parakeratin; these lesions may show an increased risk of malignant transformation. (44 refs)

- 79-4176 **Bone Tumors in Thorotrast Patients.** (Eng) Mays, C. W. (Radiobiology Lab., Univ. Utah, Medical Center, Salt Lake City, UT 84132); Spiess, H. *Environ Res* 18(1): 88-93; 1979.

As of 1974, nine bone sarcomas (6 histologically confirmed, 3 possible) were reported in Thorotrast patients throughout the world. Their appearance times ranged from 16 to 33 yr (av 26) after Thorotrast injection. In some countries, the population at risk is not known but in Denmark, Germany, and Portugal, about 3,000 patients have been followed >10 yr (45,000 person-years at risk beyond the first 10 yr). Among these 3,000 patients, three to six bone sarcomas have occurred, compared with 0.5 case expected.

Using dosimetric results obtained previously and assuming that translocating ^{224}Ra was mainly responsible, the provisional risk coefficient was calculated to be 55-120 bone sarcomas/ 10^6 person-rad of av dose to the skeleton without marrow. This risk coefficient is highly tentative, because it is based on only three to six bone sarcomas, all reported since 1972. If additional bone sarcomas occur among living Thorotrast patients, the risk coefficient could increase appreciably. For comparison, the risk coefficient for protracted injections of ^{224}Ra is about 200 bone sarcomas/ 10^6 person-rad, based on 54 cases of bone sarcoma. (20 refs)

- 79-4177 **Risk of Radiation-Related Subsequent Malignant Tumors in Survivors of Ewing's Sarcoma.** (Eng) Strong, L. C. (Section Medical Genetics, Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Herson, J.; Osborne, B. M.; Sutow, W. W. *J Natl Cancer Inst* 62(6): 1401-1406; 1979.

The risk factors for second malignant neoplasm(s) (SMN) following Ewing's sarcoma (ES) were studied in 24 patients who had remained disease-free for 3 yr following the diagnosis of ES. Among these patients, one black patient (the proband) developed a dermatofibroma and an osteosarcoma following heavy irradiation. Three other patients developed osteosarcomas in irradiated bone >3 yr after irradiation, and one developed cutaneous malignant fibrous histiocytoma in the heavily irradiated area 18 mo after treatment. The latter patient was omitted from statistical consideration because the second tumor occurred so soon after diagnosis. The ratio of observed to expected SMN showed significant relative risks of 83 for all cancer and 2,400 for bone and joint cancer for these ES patients. The radiation risk was 7.2 cases in exposed tissues per million person-year(s) per rad. The median tumor dose for patients with SMN was 6,100 rads as compared with 6,000 rads for those without SMN. The cumulative cancer risk as 10 yr was estimated as 35% for the 22 irradiated patients, excluding the proband. The cumulative cancer risk over 10 yr was 70% for patients treated with intensive multimodal therapy (chemotherapy + irradiation) compared with 25% over 20 yr for patients treated primarily with radiation ($p < 0.06$).

- 79-4178 **Evidence Against Transmission of Hodgkin's Disease in High Schools.** (Eng) Grufferman, S. (Dept. Pediatrics, Duke Univ. Medical Center, P.O. Box 2953, Durham, NC 27710); Cole, P.; Levitan, T. R. *N Engl J Med* 300(18): 1006-1011; 1979.

The hypothesis that persons exposed to adolescents with preclinical or with manifest Hodgkin's disease (HD) have an increased risk of the disease themselves was evaluated. A survey in Greater Boston identified 1,577 new cases of histologically diagnosed HD between 1959 and 1973. Of these cases, 448 were of ages such that the patients might

have attended high school during 1960-1973. High-school attendance was ascertained for 96% of these persons. First, a study was made of the incidence of HD at high schools in two time periods. The 13 high schools with cases present in 1960-1964 were matched with 26 schools that had no cases in this period, and these two groups were compared for the occurrence of HD during 1965-1969. The proportion of "exposed" high schools that had cases diagnosed in the second period was 0.62, and that for the matched high schools was 0.65. This two-time-period analysis was performed for 25 additional combinations of time periods; in none of them was a statistically significant positive result found. Secondly, the risk of HD among persons who had attended high school at the same time as a diagnosed case was studied. These "exposed" persons were followed through 1973; 12 cases were observed among them, vs an expected number of 13.9. Thirdly, the extent to which members of all possible pairs of cases had attended the same or nearby high schools at the same time was ascertained. There was no excess of classmate pairs than might have occurred by chance alone. These data suggest that there is no transmission of an etiologic agent for HD at high schools. (28 refs)

- 79-4179 **Bloom's Syndrome. VII. Progress Report for 1978.** (Eng) German, J. (310 E. 67th St., New York, NY 10021); Bloom, D.; Passarge, E. *Clin Genet* 15(4): 361-367; 1979.

Recent accessions to the Bloom's Syndrome (BS) Registry, new cases of neoplasia, and recent clinical and experimental observations are reported. Since the Registry was last published in 1977 with 71 entries, 11 more cases have been confirmed. Growth retardation is the most impressive clinical feature of the syndrome. Five new malignant neoplasms were recognized in four patients. These malignancies were acute lymphocytic leukemia (2 cases), lymphoma of the epipharynx, squamous cell carcinoma of the epiglottis, and disseminated lymphoma (the last 2 in 1 patient). Of the 77 individuals with BS who survived to age 3, 16 developed 18 cancers, predominantly leukemia. Four of the eight leukemias were nonlymphocytic and four were lymphocytic. The diagnostic value in BS of a great increase in the number of sister chromatid exchanges (SCE's) is emphasized. The primary biochemical defect in BS remains unknown. Defective DNA repair mechanisms have been sought but not found. BS lymphocytes respond to ethyl methanesulfonate with increased SCE production. The rate of DNA chain maturation is lower than normal in BS cells. An increased spontaneous occurrence of heavy/heavy DNA was demonstrated in replicating BS fibroblasts, an observation that may indicate a greater than normal amount of exchange between parental and daughter DNA strands in BS cells. (26 refs)

- 79-4180 **An Analysis of 346 Cases of Clear Cell Adenocarcinoma of the Vagina and Cervix**

with Emphasis on Recurrence and Survival. (Eng) Herbst, A. L. (Registry Res. - Hormonal Transplacental Carcinogenesis, 5841 S. Maryland Ave., Chicago, IL 60637); Norusis, M. J.; Rosenow, P. J.; Welch, W. R.; Scully, R. E. *Gynecol Oncol* 7(2): 111-122; 1979.

An analysis of recurrence and survival rates for 346 cases of clear cell adenocarcinoma of the vagina and cervix accessioned into the Registry for Research on Transplacental Carcinogenesis (as of April 1, 1978) is presented. In the early 1970's, these cancers reached a peak incidence that has continued on the same level to the present. Two-thirds of the patients with a maternal history had been exposed to diethylstilbestrol (DES) or related compounds in utero. Follow-up for periods up to 15.3 yr (mean, 4.0) yielded an actuarial 5-yr survival of 78%. The survival figures for Stage I carcinomas of the vagina and cervix were 87% and 91%, respectively. Local excision of Stage I appeared to carry an increased risk of recurrence. The overall recurrence rate for all cases was 23% at 5 yr. Recurrences were most frequent in the pelvis and lungs. (12 refs)

- 79-4181 **Cancer Incidence in the Western United States: Ethnic Differences.** (Eng) Hu, J. H. (9909 Fleming Ave., Bethesda, MD); White, J. E. *J Natl Med Assoc* 71(4): 345-348; 1979.

Cancer incidences reported by the California and New Mexico Tumor Registries were compared and analyzed according to major ethnic groups. Among whites, cancer of the lung and bronchus, colon and rectum, and prostate showed the highest incidence rates for men and breast cancer and uterine cancer showed the highest rates for women. Blacks had the highest incidences of prostate, lung, and bronchial cancer among the racial groups. The Chinese had high rates of liver and nasopharyngeal cancer and a low rate of prostate cancer. The Japanese had the highest stomach cancer rate. Cancer problems of different ethnic groups were compared with those in their native countries. Compared with native Mexicans, the US Spanish population had increased prostate and breast cancer incidences and a lower rate of stomach cancer. In US blacks, prostate and lung cancers were increased compared with blacks in the Dominican Republic, and stomach cancer incidence was decreased among men. Breast and colorectal cancers were elevated and uterine cancer was reduced among US black women. Breast, lung, bronchial, and colorectal cancers were increased in US Chinese compared with Chinese in Taiwan, but the incidence of stomach cancer was reduced. Similar trends were observed in Japanese in the US and Japan. These results reveal the impact of environmental or cultural changes on lung, breast, and stomach cancer and a possible genetic influence on the high incidence of nasopharyngeal cancer among the Chinese in the US and Taiwan. (14 refs)

- 79-4182 **Estrogen Use and Endometrial Cancer (7 Letters to Editor).** (Eng) Greenblatt, R. B.

(Medical Coll. Georgia, Augusta, GA 30901); Boyers, S. P.; Buster, J. E.; Marshall, J. R.; Blesius, C. K.; Sturgis, S. H.; Studd, J. W.; Thom, M.; Horwitz, R. I.; Feinstein, A. R.; Stolley, P. D.; Davies, J. L. *N Engl J Med* 300(16): 921-923; 1979.

Seven letters were written in response to a study implicating postmenopausal estrogen therapy in the development of endometrial carcinoma (EC). The fact that cyclic progesterone had not been added to the estrogen regimen in the study population was emphasized, and evidence suggesting that progesterone protects against the development of EC is cited. In addition, 29/65 EC patients were receiving continuous estrogen therapy and 9 were receiving diethylstilbestrol. The authors of the original article argue that cyclic estrogen use involves about the same relative

risk as continuous use. Other issues debated included the study design and the fact that types of estrogens used were not defined. (11 refs)

See also:

*(Rev.): 79-3603, 79-3604, 79-3608, 79-3609, 79-3610, 79-3611, 79-3612, 79-3613, 79-3618, 79-3625, 79-3628, 79-3633, 79-3638, 79-3641, 79-3642, 79-3646, 79-3647, 79-3648, 79-3649, 79-3650, 79-3651, 79-3652, 79-3653.

*(Chem.): 79-3662, 79-3673, 79-3693, 79-3694, 79-3696, 79-3698, 79-3699, 79-3700, 79-3715, 79-3745, 79-3756.

*(Immun.): 79-3980, 79-3997, 79-4012.

*(Path.): 79-4028, 79-4087, 79-4102.

MISCELLANEOUS

79-4183 Transformed Mammalian Cells Secrete Specific Proteins and Phosphoproteins. (Eng)

Senger, D. R. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Wirth, D. F.; Hynes, R. O. *Cell* 16(4): 885-893; 1979.

Proteins secreted into the culture medium by hamster, mouse, and rat cell lines and their RNA and DNA tumor virus-transformed derivatives were compared. Cells were labeled with ^{35}S -methionine or ^{32}P -orthophosphate, and the labeled conditioned medium was analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and fluorography. Eight of nine transformed cell lines secreted a 58,000-dalton (58K) protein that was either absent or at a much lower level in the media of normal cells. The 58K proteins secreted by transformed hamster, rat, and mouse cells were antigenically related. Experiments with temperature-sensitive mutants indicated that expression of the 58K protein was closely correlated with transformation. Seven of the eight ^{32}P -labeled transformed cell lines secreted a transformation-dependent 58K or 62K phosphoprotein, depending on the species. The quantities of these components were much lower in the media from untransformed cell lines. Immunoprecipitation indicated that the phosphoproteins and the 58K ^{35}S -methionine-labeled proteins were not antigenically related. Incubation of media from transformed cell cultures with γ - ^{32}P -ATP resulted in the labeling of 58K or 62K phosphoproteins, indicating the presence of both a protein kinase and a substrate in the media. The results suggest that the phosphoproteins detected in the media of the transformed cell cultures play an important role in determination of the transformed phenotype. (31 refs)

79-4184 The Analysis of Malignancy by Cell Fusion (Meeting Abstract). (Eng) Sidebottom, E. (Sir William Dunn Sch. Pathology, Oxford, England). *In Vitro* 15(3): 167; 1979 (no refs)

79-4185 Puzzling Role of Cell Surfaces. (Eng) Meyer, D. I. (Dept. Biochemistry, Biocenter, Univ. Basel, CH 4056 Basel, Switzerland); Burger, M. M. *Cancer Outlaw Cell* 61-72; 1978.

The properties of normal and cancer cell surfaces and the role of the cell surface in transformation are reviewed. In contrast to normal cells, transformed cells in culture grow past confluence, grow in agar, and exhibit anchorage-independent growth. A glycoprotein termed large, exter-

nal, transformation-sensitive (LETS) protein, which is found in large amounts on normal cells at confluence, decreases or disappears in transformed cells. Treating normal cells with proteases induces many changes similar to those seen in transformed cells. Proteases may also cause LETS proteins to disappear in transformed cells. The cell skeleton is also altered in transformed cells. Actin, which is present in large, thick cables in normal cells, seems to disappear in transformed cells. Treating transformed cells with a protease inhibitor causes the cables to reform. Cancer-causing agents alter some feature of the cell surface and/or cell skeleton. The cell then loses its capacity to halt growth when the cell population becomes too crowded. Cancer cells, which are less adhesive than normal cells, can dislodge from a growing tumor and be transported to other sites of the body. This would account for the invasive and metastatic properties of tumor cells. Cell-surface changes, however, might not be directly responsible for the production or maintenance of cancer but might be a secondary effect. (no refs)

79-4186 A New Parameter for Discriminating Malignantly Transformed Cell Lines from Nontransformed Counterparts: Culture in Liquid Medium Containing Neutral Protease. (Eng) Hiragun, A. (Dept. Cell Biology, Tokyo Metropolitan Inst. Medical Science, Honkomagome 3-18-22, Bunkyo-ku, Tokyo 113, Japan); Sato, M.; Mitsui, H. *Gann* 70(2): 187-193; 1979.

The growth properties of malignantly transformed mouse or rat cell lines and their nontransformed counterparts were studied in liquid medium containing a bacterial neutral protease (Dispase I) and correlated with colony formation in soft agar and tumorigenicity. Trypsin-dispersed cells were suspended in the growth medium at 2×10^4 cells/ml, and 2.5-ml aliquots were placed in test dishes. Protease was added to give a final concentration of 50-100 units/ml. The number of viable cells was determined until day 5 after seeding. The three nontransformed cell lines (BALB/3T3, NIH-L₁, and NRK) and three nontransformed cell strains did not grow in the protease-containing medium. All but one of the transformed cell lines that formed colonies in soft agar grew in the protease-containing medium. Transformed cell line cl.7T² formed colonies in soft agar with a high frequency, but it failed to proliferate in the presence of the protease, perhaps because of its high sensitivity to the growth inhibitory effect of Dispase I. Two other tumorigenic cell lines, which lacked the ability to form colonies in soft agar, were unable to grow in the protease medium. It is concluded that malignantly transformed cell lines can be distinguished

from their nontransformed counterparts by simply counting the number of cells on day 5 after seeding in a growth medium containing 100 units/ml Dispase I. (7 refs)

- 79-4187 Behaviour of Normal and Neoplastic Cultured Mouse Cells on the Chorioallantoic Membrane of the Chicken.** (Eng) Mauersberger, B. (Akademie der Wissenschaften der DDR, Institut für Wirkstoffforschung, Alfred-Kowalke-Strasse 4, DDR-1136 Berlin, E. Germany); Jakob, W.; Zipper, J. *Exp Pathol (Jena)* 17(1): 18-24; 1979.

The effect of cell suspensions from transformed (L) and normal (C3H mouse lung) cells on the chorioallantoic membrane (CAM) of the embryonated herd egg was studied. Addition of suspensions of free-floating normal and transformed cells to the CAM induced an angiogenic reaction in the CAM mesoderm. This effect seemed to be dependent on the infiltrative growth of the cultured cells. The first reaction of the host tissue consisted of hyperplasia of the mesoderm and, to a certain degree, the epidermal layer. During this stage, the degree of vascularization of the mesoderm tissue was low. As soon as the cells could infiltrate the epidermal layer of the CAM, forming small bulks of cells inside the mesoderm, the number of vessels increased significantly. The mode of invasion of the normal cells was somewhat different from that of the transformed cells, and the macroscopically visible angiogenic responses on the surface of the CAM was weaker with the former. Treatment of the transformed cells a broad-spectrum protease inhibitor (Contrykal) prior to inoculation onto the CAM significantly inhibited their infiltrative capacity. (29 refs)

- 79-4188 ^{31}P Nuclear Magnetic Resonance Studies on Relaxation Parameters and Line Broadening of Intracellular Metabolites of HeLa Cells.** (Eng) Evans, F. E. (Div. Chemistry, Natl. Center Toxicological Res., Jefferson, AR 72079). *Arch Biochem Biophys* 193(1): 63-75; 1979.

Phosphorus-31 nuclear magnetic resonance studies of the relaxation parameters and line broadening of intracellular metabolites of HeLa cells are reported. The spin-lattice relaxation times (T_1) ranged from 0.3 sec for ATP to 3 sec for inorganic phosphate (P_i) and monophosphate compounds. Nuclear Overhauser enhancements (NOE) were induced by proton irradiation with the possible exception of ATP. In all cases, T_1 and NOE were smaller for intracellular metabolites than for cell-free compounds. The relaxation parameters for ATP were affected the most. The larger change in T_1 and NOE of intracellular ATP could be accounted for by selective binding of paramagnetic ions. This phenomenon also explains some of the line broadening in the cell spectrum, especially that of ATP. The spin-

spin relaxation times (T_2) of P_i and monophosphate compounds, as measured by a pulse technique, did not account for the observed linewidths. This is due to the presence of chemical shift envelopes arising from pH heterogeneity. All resonances were broader at 146 megahertz (MHz) because of the line broadening by paramagnetic ions and the presence of chemical shift envelopes. There was little difference in the resolution of spectra at 40 and 146 MHz. Water proton linewidths and T_2 values were measured for HeLa cells and for some minced tissue preparations. The water linewidth in tissue samples was broader than that in the cell suspensions mainly because of chemical shift envelopes caused by magnetic field nonuniformity in the tissue samples. There also appeared to be a small chemical shift envelope from magnetic nonuniformity in HeLa cells. (29 refs)

- 79-4189 Transplanted Cultured Cells from Pregnancy-dependent Mammary Tumors Have a Heterogenous Developmental Potential.** (Eng) Aidells, B. D. (Dept. Molecular Biology, Univ. California, Berkeley, CA 94720); Lee, A. E. *Int J Cancer* 23(5): 718-721; 1979.

The developmental potentials of cultured cells derived from pregnancy-dependent mouse mammary tumors (plaques) were studied. Cell cultures were established from a pooled sample of six spontaneously arising plaques removed from BR6 mice on gestation days 15-19 and from secondary transplants from two primary plaques. Intact plaques and dissociated plaque cells, cultured and uncultured, were injected into cleared fat pads and allowed to grow for 4-6 mo before the mice were mated. Some of the mice were not mated. Fat pads containing the transplants were examined at the end of pregnancy. In virgin hosts, transplants from intact plaques were ductal, but outgrowths from dissociated cells consisted of three variants: hyperplastic alveolar nodules (HAN's), ductal hyperplasias, and autonomous mammary carcinomas. Some of the outgrowths that produced mammary tumors also contained normal mammary ducts on the periphery. In pregnant mice, some outgrowths also produced areas of normal mammary development, especially the transplants from cultured cells. Of 30 successful outgrowths from cultured cells, 16 contained areas of normal development and 2 contained no neoplastic areas. Of 14 outgrowths derived from uncultured dissociated cells, only 2 contained normal areas. It is concluded that cells derived from pregnancy-dependent mammary tumors are capable of giving rise to normal mammary glands, HAN's, plaques, and autonomous mammary tumors. It is not known whether this is due to the heterogeneity of the cells or to the multi-developmental potential of a given cell. (8 refs)

- 79-4190 Proteases are Mitogenic to Mesenchyme In Vivo. A Study Using the Chick Embryo**

Chorioallantoic Membrane. (Eng) Grumm, F. G. (Dept. Zoology, Univ. California, Davis, CA 95616); Armstrong, P. B. *Exp Cell Res* 119(2): 317-326; 1979.

The growth of mesenchymal tissue *in vivo* was studied using chorioallantoic membranes (CAM's) from 9- and 10-day-old chick embryos. The av labeling indices for the chorionic epithelium (CE), allantoic epithelium (AE), and mesenchymal layers were 23.4, 27.9, and 39.2/100 cells, respectively. Trypsin (0.05-0.20 mg/200 μ l medium) and medium conditioned by activated macrophages (dilutions containing 5%-40% reconstituted medium from 2×10^6 activated macrophages) increased the labeling index of the mesenchymal cells by 42%-181% and 46%-160%, respectively. Activated macrophages (1 or 2×10^6 /CAM), which rarely invaded the CE, increased the labeling index of the mesenchymal cells by 56%-104%. The labeling index was increased by 6 hr and remained elevated for at least 24 hr. No significant changes in the labeling indices of the CE and AE were noted after exposure to trypsin, macrophages, or conditioned medium. The mesenchymal cells were more densely packed and less randomly arranged in the mitogen-treated CAM's than in untreated CAM's; these morphologic changes were restricted to the mesenchyme. This system could be used to evaluate the mitogenicity *in vivo* of other substances known to be mitogenic to fibroblasts *in vitro*. (52 refs)

79-4191 Construction of a Site-specific, Deletion-Frameshift Mutation in an Essential Gene of Bacteriophage Φ X174. (Eng) Humayun, M. Z. (Dept. Biochemistry, New York Univ. Sch. Medicine, 550 First Ave., New York, NY 10016); Chambers, R.W. *Nature* 278(5704): 524-529; 1979.

The *in vitro* construction of a site-specific deletion-frameshift mutant, ZH1, from which residues 2,513-2,592 (80 bases) of the wild-type sequence (gene G of the virulent bacteriophage Φ X174) were deleted, is reported. ZH1 was shown to be a non-temperature-sensitive gene G mutant that did not grow on any of the nonsense suppressor strains tested. It appeared to be capable of all phage functions except those performed by the gene G product(s). It had a low burst size on p Φ XG-bearing strains and a recombination frequency with p Φ XG that was lower than that of the point mutant, Gam9. Except for the deletion, the wild-type sequence was apparently conserved in ZH1. The mutant also contained a -2 frameshift in which 14 in-frame, nonsense codons were generated at and downstream from the deletion site. These results demonstrate that it should be possible to rescue most Φ X174 gene G mutants regardless of the origin or nature of the mutation. (20 refs)

79-4192 Induction of the DNA Repair Enzyme Uracil-DNA Glycosylase in Stimulated Human

Lymphocytes. (Eng) Sirover, M. A. (Fels. Res. Inst., Temple Univ. Sch. Medicine, Philadelphia, PA 19140). *Cancer Res* 39(6,part 1): 2090-2095; 1979.

The capacity of human cells to modulate the synthesis of DNA repair enzymes was investigated by measuring the induction of uracil-DNA glycosylase during lymphocyte stimulation. Treatment of peripheral lymphocytes with phytohemagglutinin (PHA) increased glycosylase activity tenfold. Glycosylase stimulation was coordinate with the activation of DNA synthesis and DNA polymerase activity. Two chromatographically distinct species of the glycosylase were resolved, only one of which is induced during PHA stimulation. The effect of actinomycin D and cycloheximide on glycosylase induction was determined. Treatment with either inhibitor at 96 hr after PHA addition (max induction) decreased glycosylase activity after an appreciable lag period. This suggested that induction of the uracil-DNA glycosylase requires transcription and translation even though the enzyme may be quite stable once induced. (34 refs)

79-4193 Transplantation of Human Malignant Mesothelioma (HMM) into Nude Mice (Meeting Abstract). (Eng) Chahinian, A. P. (Mount Sinai Sch. Medicine, New York, NY 10029); Beranek, J. T.; Suzuki, Y.; Bekesi, J. G.; Selikoff, I. J.; Holland, J. F. *Proc Am Assoc Cancer Res* 20: 171; 1979 (no refs)

79-4194 Establishment and Characterization of a Human Null-Cell Lymphoblastic Lymphoma Cell Line (K-LL-3). (Eng) Smith, S. D. (Dept. Pediatrics, Univ. Kansas Medical Center, Kansas City, KS 66103); Rosen, D. *Int J Cancer* 23(4): 494-503; 1979.

The establishment and characteristics of a null-cell lymphoma cell line (K-LL-3) derived from a bone marrow aspirate from a 13-yr-old boy with diffuse, poorly differentiated lymphoblastic lymphoma are described. The lymphoblastic colonies in the primary culture grew side by side with normal myeloid and monocytic colonies; the normal colonies were not passaged successfully. K-LL-3 has been cultured for >1 yr, and >70 passages have been performed. The cells grow rapidly in the agar assay system, with a doubling time of 24-36 hr after a latent period of approx 48 hr. The cells are dependent on a feeder layer and on complete medium supplemented with calf serum; human plasma enhances the colony growth. The K-LL-3 cells show a basic pseudodiploid male karyotype, and loss of A- and C-group chromosomes plus the gain of extra B- and F-group chromosomes occur fairly consistently. The cells are negative for Epstein-Barr nuclear antigen, lack specific cell-surface markers, and do not secrete IgG. The HL-A type of the first cell line established was: A-2, A-11, B-37, and B-5 or B-15. It appears that the K-LL-3 line is malig-

nant and that it originated from the patient's tumor cells. (29 refs)

- 79-4195 Comparison of Rat Liver and Walker Carcinoma tRNAs.** (Eng) Roe, B. A. (MRC Lab. Molecular Biology, Hills Road, Cambridge CB2 2QH, England); Stankiewicz, A. F.; Rizi, H. L.; Weisz, C.; DiLauro, M. N.; Pike, D.; Chen, C. Y.; Chen, E. Y. *Nucleic Acids Res* 6(2): 673-688; 1979.

Normal rat liver and Walker 256 mammary carcinoma transfer RNA's (tRNA's) were fractionated by anion-exchange chromatography, their nucleoside composition was analyzed, and the nucleotide sequence of the major species of both normal and carcinoma tRNA-Asn was compared. Relative quantitative differences were observed in the elution profiles of tRNA-Asp, tRNA-Asn, tRNA-His, and tRNA-Tyr. These differences were due to alterations in tRNA distribution rather than to differences in aminoacylation. The four tRNA's were analyzed by anion-exchange chromatography following treatment with cyanogen bromide, which affects the Q nucleoside, and aminoacylation. Cyanogen bromide treatment caused a retarded elution of the four normal liver tRNA's but did not alter the elution profiles of the Walker 256 carcinoma tRNA, indicating that the tumor tRNA may lack the Q nucleoside. Comparison of the nucleotide sequence of rat liver and Walker 256 carcinoma tRNA-Asn indicated that all fragments produced by complete RNase T₁ and RNase A digestion are identical except for those from the anticodon loop region. Rat liver tRNA contains the Q nucleoside in the wobble position of the anticodon loop and Walker 256 carcinoma contains a guanosine. Both tRNA's contain an unknown nucleoside, Z, in the position usually occupied by the ribothymidine, as well as an X nucleoside in loop I rather than in loop III. These results establish that Walker 256 mammary carcinoma tRNA contains significantly less Q and Q* nucleoside than normal rat liver tRNA and that the major species of tumor tRNA-Asn contains a guanosine rather than a Q nucleoside in the wobble position of its anticodon. (77 refs)

- 79-4196 Plasma Triiodothyronine Concentrations in Breast Cancer.** (Eng) Rose, D. P. (Div. Clinical Oncology, Univ. Hospitals, 600 Highland Ave., Madison, WI 53706); Davis, T. E. *Cancer* 43(4): 1434-1438; 1979.

Plasma triiodothyronine (T₃), thyroxine (T₄), and thyroid-stimulating hormone (TSH) were measured in 44 patients with early breast cancer, 48 patients with advanced breast cancer, 20 women with colon cancer, and 56 healthy age-matched women. There was no correlation between plasma T₃ concentration and age. Plasma T₃ concentrations were

reduced significantly in both groups of breast cancer patients, but they were reduced only in those colon cancer patients with metastatic disease. Plasma T₄ levels were similar in control and cancer patients. The mean plasma TSH level was significantly higher in patients with advanced breast cancer and nonsignificantly higher in patients with early breast cancer than in controls or patients with colon cancer. There was a strong negative correlation between plasma TSH and T₃ in patients with early breast cancer but not in those with advanced breast cancer. It is concluded that a proportion of breast cancer patients are mildly hypothyroid and that in advanced cancer, other nonspecific factors also act to reduce the plasma T₃ concentration. (26 refs)

- 79-4197 Phospholipid Composition and DNA Synthesis in Nuclei of Liver and Tumor Cells During the Growth of Ehrlich Ascites Carcinoma.** (Rus) Pal'mina, N. P. (Inst. Chemical Physics, Moscow, USSR); Mal'tseva, E. L. *Dokl Akad Nauk SSSR* 245(2): 483-486; 1979.

NK mice were inoculated with Ehrlich ascites carcinoma cells (6×10^6 cells/mouse) and then sacrificed after various time intervals, and the phospholipid composition and the rate of DNA synthesis in liver and tumor cell nuclei were determined. Tumor growth was associated with an increase in levels of minor phospholipids such as sphingomyelin, phosphatidylserine, and phosphatidylinositol and an almost twofold decrease in the phosphatidylcholine level. In liver cells, there was a progressive decrease in the phosphatidylcholine level and an increase in the phosphatidylserine and sphingomyelin levels. Changes in the levels of the minor phospholipids were not correlated with the rate of DNA synthesis in the tumor cells. (14 refs)

- 79-4198 Lipid Content and Asymmetry of Lysosomal Phospholipids in Normal and Malignant Growth.** (Rus) Dzishkariani, O. S. (State Univ., Tbilisi, USSR); Lursmanashvili, T. A.; Tsartsidze, M. A.; Lomsadze, B. A. *Soobshch Akad Nauk Gruz SSR* 92(2): 441-444; 1978.

The level and distribution of lysosomal phospholipids isolated from the liver of normal random-bred rats, from Guerin hepatocellular carcinoma tissue, and from liver tissue surrounding the carcinoma were compared. Tumor growth was associated with marked qualitative and quantitative changes in phospholipid content: the phosphatidylserine level increased from 2% in normal liver to 4.4% in liver tissue surrounding the tumor to 12.5% in the carcinoma tissue. The phosphatidylcholine level increased from 35.1% to 45.9% to 50.7%, respectively, but the phosphatidylinositol levels were 2.9%, 8.7%, and 3.2%, respectively. There were significant differences in

the location of the phospholipids on the lysosomal membranes: the membranes of the normal lysosomes contained approx 78% of the total phosphatidylinoside, 42% of the total phosphatidylethanolamine, and approx 3% of the total phosphatidylcholine, but the membranes of the tumor lysosomes contained approx 80% of the total phosphatidylserine, 21.5% of the total phosphatidylcholine, and 5.4% of the total phosphatidylinoside. (7 refs)

- 79-4199 Comparison of Salt-extractable Nuclear Proteins of Regenerating Liver, Fetal Liver, and Morris Hepatomas 9618A and 3924A.** (Eng) Takami, H. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030); Busch, F. N.; Morris, H. P.; Busch, H. *Cancer Res* 39(6, part 1): 2096-2105; 1979.

The nuclear proteins of regenerating and fetal rat liver, slow-growing Morris hepatoma 8618A, and fast-growing Morris hepatoma 3924A were sequentially extracted from nuclei with 0.075 M NaCl/0.025 M EDTA, 10 mM Tris, 0.35 M NaCl, 0.6 M NaCl, and 3 M NaCl/7 M urea. The sequential protein fractions were analyzed by two-dimensional, isoelectric-focusing sodium dodecyl sulfate gel electrophoresis. Many of the protein spots were common to all tissues and corresponded to the proteins found previously in a Novikoff hepatoma and normal liver. The protein patterns of the regenerating liver and slow-growing 9618A hepatoma were more similar to those of normal liver than to those of other tissues. Many similarities were found between the fetal liver and the Morris 3924A and Novikoff hepatomas. Four protein spots, 64/5.9, 60/6.3, 51/5.3, and 38/7.3 (mol wt $\times 10^{-3}$ /plate), were found only in the fast-growing 3924A and Novikoff hepatomas. Proteins 79/6.4 and 61/7.2 were found only in the three hepatomas. Protein 37/6.3 was much more dense in the three hepatomas than in regenerating and fetal livers. Two proteins, 28/5.0 and 27/4.9, were detected only in the fast-growing 3924A and Novikoff hepatomas and fetal liver.

Proteins 125/8.2 and 98/8.4 were found in the fetal liver and the three hepatomas; these proteins may be oncofetal proteins. Three proteins, 61/5.5, 56/5.8, and 53/7.5, that were absent in other tissues were present in regenerating liver, fast-growing 3924A and Novikoff hepatomas, fetal liver, and slow-growing 9618A hepatoma. These proteins may be related to growth processes of the normal and tumor tissues studied. (31 refs)

- 79-4200 Estrogen Receptor in Hamster Kidney During Estrogen-induced Renal Tumorigenesis.** (Eng) Anderson, N. S. (Dept. Anatomy, Sch. of Medicine, Univ. Oregon Health Sciences Center, Portland, OR 97201); David, Y.; Fanestil, D. D. *J Steroid Biochem* 10(2): 123-128; 1979.

Specific binding of ^3H -estradiol to isolated cytosol and to nuclei in tissue slices was determined in the hamster kidney before and during estrogen-induced renal tumorigenesis and in the kidney of normal rats, a species that does not develop the tumor. At 10^{-9} M ^3H -estradiol, cytosol from normal hamster kidney bound 6.48×10^{-15} moles (mol)/mg protein, which was significantly less than the 15.8×10^{-15} mol/mg bound by the rat. The apparent dissociation constant for estradiol was not significantly different (0.50 nanometers (nM) in the hamster and 0.66 nM in the rat). Cytosol binding increased after hamsters were implanted with estrogen pellets for 6 mo (9.23×10^{-15} mol/mg) or had developed tumors (13.7×10^{-15} mol/mg). In tissue slices, the amount of ^3H -estradiol translocated to nuclei was greater in the rat (21.8×10^{-14} mol/mg nuclear protein) than in the normal hamster (3.28×10^{-15} mol/mg), and it was greater in tumor-bearing hamsters (6.70×10^{-15} mol/mg DNA) than in normal hamsters (3.19×10^{-15} mol/mg DNA). These results indicate that (1) the kidney of the tumor-prone hamster does not contain an estrogen receptor of excessive quantity or unusual affinity and (2) the estrogen-receptor complex in estrogen-induced tumors can be translocated to cellular nuclei. (17 refs)

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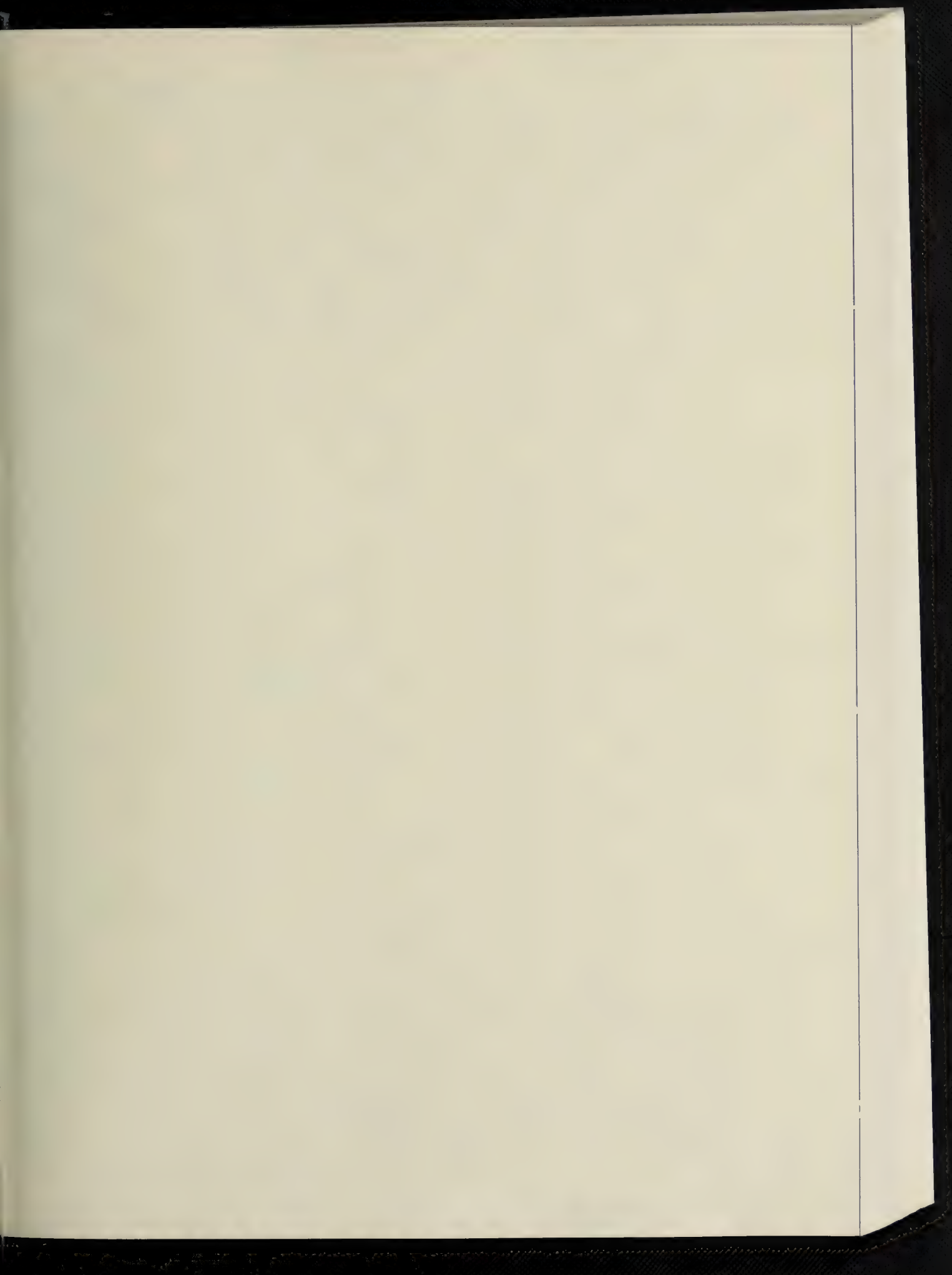
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ABBREVIATIONS

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LANGUAGE of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

ABBREVIATIONS used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED₅₀	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO₂	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD₅₀	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD₅₀	median lethal dose		
M	molar		
μM	micromolar		

REVIEW

- 79-4201 **Inauspicious Omens (Letter to Editor).** (Eng) Dunea, G. (Cook County Hosp., Chicago, IL 60612). *Br Med J* 1(6174): 1337-1338; 1979.

In the face of countless reports of adverse effects related to environmental pollution and the carcinogenic potentials of many industrial and domestic items, the public has begun to ignore the problem and the government has cut back on research and anti-pollution programs. (2 refs)

- 79-4202 **Some Guidelines for Determining Maximum Permissible Levels of Chemical Mutagens.** (Eng) Drake, J. W. (Dept. Microbiology, Univ. Illinois, Urbana, IL). *Adv Mod Toxicol* 5: 9-26; 1978.

The problem of determining maximum permissible levels for chemical mutagens is considered. Characteristics of the mutational response that are relevant in establishing guidelines for absolute mutagen limits are the existence (or absence) of thresholds below which no mutational damage occurs, the extent and nature of interactions among mutagens, and the uniformity of the response of different screening systems to a given agent. The question limiting human exposure to mutagens was studied by a committee of the Environmental Mutagen Society, which reached a consensus in favor of a system of absolute limits to be placed on environmental mutagens. The committee recommended that the total dose of environmental mutagens of artificial origin, including radiations but excluding medically administered mutagens, be limited to that which would produce an av 12.5% increase in the human mutation rate. The committee recommended that no single mutagenic agent should be allowed to exceed 10% of the total mutagenic budget. In order to measure exposure to mutagens, it will be necessary to monitor total production levels, distribution patterns, persistence in the environment, and metabolic disposition within the human body. Two of the main guidelines for selecting chemicals for mutagenicity testing are the extent to which an agent is present in the environment and the extent to which it is related to known mutagens. (12 refs)

- 79-4203 **Laboratory Design and Operation Procedures for Chemical Carcinogen Use.** (Eng) Barbeito, M. S. (Office of Res. Safety, Bldg. 13, Room 2E47, NCI, 9000 Rockville Pike, Bethesda, MD 20014). *ACS Symp Ser* (96): 191-214; 1978.

Interim safety standards developed by the National Cancer

Institute for intramural laboratories involved in research work with chemical carcinogens regulated by the Department of Labor are summarized. (20 refs)

- 79-4204 **Theoretical Prediction of Carcinogenicity: Quasi-Quantification by Quasi-Valence.** (Eng) Barnes, W. S. (Dept. Botany, Univ. Massachusetts, Amherst, MA 01003); Levin, D. E. *Experientia* 35(4): 565-567; 1979.

The carcinogenicity or noncarcinogenicity of chemicals cannot be predicted by their av quasivalence number (Z^*). The method misclassifies many compounds as false negatives or false positives and does not explain differences in their carcinogenic potency. As shown by a table listing the Z^* values and carcinogenicity of some organic compounds, there is no correlation between Z^* and carcinogenicity, and the difference in Z^* between a procarcinogen and the activated, ultimate carcinogen is only $\leq 10\%$. (11 refs)

- 79-4205 **The Detection and Hazards of Environmental Carcinogens/Mutagens.** (Eng) Ames, B. N. (Biochemistry Dept., Univ. California, Berkeley, CA 94720). *ACS Symp Ser* (94): 1-11; 1979.

Methods for the detection of environmental carcinogens/mutagens are reviewed. The Ames test detects such agents by their mutagenicity for special strains of *Salmonella* bacteria using tissue homogenates from rodents for metabolic activation. With this test, most mutagens are detected at nanogram dose levels. The test has been validated using approx 300 organic chemicals of many chemical classes. Of the 175 carcinogens tested, 157 were mutagenic, and 94/108 noncarcinogens were non-mutagenic. In almost all cases, the test discriminated very well between carcinogens and noncarcinogens. A concentration of very weak mutagens is found among the "false positives" of the *Salmonella* test. It is suggested that most of the false positives may be weak carcinogens which have not been detected in animal tests. Recent data indicates that there may be a rough correlation between carcinogenic and mutagenic potency. The *Salmonella* test has shown over a million-fold range of mutagenic potency among the carcinogens tested, significant numbers of mutagens having been observed throughout the entire potency range. Mutagenic impurities can play a significant role in mutagenesis testing. The *Salmonella* test can be used to monitor the introduction of mutagenic impurities during

industrial processing, as a bioassay when separating and concentrating mutagens/carcinogens from complex mixtures, and as part of a long-term toxic substances monitoring program. There are many other short-term tests for mutagenicity and, because each test detects a few carcinogens which the others do not, the use of a battery of such tests is favored. (32 refs)

79-4206 Genetic Diseases in Humans Versus Mutagenicity Test Systems. (Eng) Flamm, W. G. (Div. Cancer Cause and Prevention, Natl. Cancer Inst., Bethesda, MD). *Adv Mod Toxicol* 5: 3-8; 1978.

Four categories of human diseases that are of genetic origin are outlined and the types of available experimental systems for detecting and measuring the genetic events responsible for the diseases in each category are reviewed. The first category consists of chromosomal mutations, the most frequently encountered of which are mongolism, Klinefelter's syndrome, and Turner's syndrome. The second category of human genetic diseases comprises those occurring through mutations of single genes that are dominant. Nine forms of cancer are known to occur through mutations of dominant genes including bilateral retinoblastoma and polyposis coli. The other seven hereditary neoplasms are very rare. The third category of mutations consists of the recessive mutations and includes such diseases as sickle-cell anemia, phenylketonuria, and galactosemia. The only methods thought to be useful for detecting and measuring both dominant and recessive mutations are methods that measure heritable effects. Bacteria can be used for this and will measure a variety of mutational events. Genetic investigations combined with pharmacokinetic studies seems to be the best approach for assessing the genetic risk attributable to specific levels of exposure to a given substance. None of the current mutagenicity methods are relevant to the fourth category of genetic diseases which includes such disorders as diabetes, epilepsy, schizophrenia and essential hypertension, that are presumably transmitted as multiple gene disorders. (8 refs)

79-4207 DNA Repair as it Relates to Mutagenesis and Gene Expression in Mammalian Cells and Tissues. (Eng) Lieberman, M. W. (Dept. Pathology, Washington Univ. Sch. Medicine, St. Louis, MO 63110). *Adv Mod Toxicol* 5: 29-52; 1978.

The repair of specific types of DNA damage, adequacy of restoration of the damaged area, and functional consequences of DNA repair are reviewed and the usefulness of repair synthesis as a screen for genetically active agents in the environment is evaluated. A great variety of sequences are repairable in the mammalian genome, and at least one set of sequences that does not code for a specific protein is

also repairable. The informational content of an individual sequence is probably not a factor in determining whether that sequence will be repaired. The excision repair system appears to be at least error-correcting and perhaps error-free, but postreplication repair seems to be error-prone. Carcinogens may be removed from the genomes of mammalian cells, but their removal is rarely complete. Some data suggest that differential rates of removal of adducts occur in mammalian cells. Studies of repair in normal tissues and their neoplastic counterparts have demonstrated increased unscheduled DNA synthesis in the neoplastic tissue. Thus, loss of DNA repair capacity does not seem to be responsible for neoplastic progression. Repair synthesis and specific locus tests in mammalian cells may be about equally sensitive for detecting potentially hazardous agents. There is little evidence from cell culture work that aging affects repair. With increasing cellular age, increasing percentages of variant enzymes accumulate, presumably the result of accumulation of errors in the protein-synthesizing machinery. The relation of repair to this phenomenon is not clear. (116 refs)

79-4208 Human Lymphoblasts: Versatile Indicator Cells for Many Forms of Chemically Induced Genetic Damage. (Eng) Thilly, W. G. (Genetic Toxicology Group, Dept. Nutrition and Food Sciences, Massachusetts Inst. Technology, Cambridge, MA 02139); Deluca, J. G. *ACS Symp Ser* (94): 13-27; 1979.

Different approaches to the determination of genetic hazards are reviewed and attention is drawn to the relationship between the actual biochemistry of an animal and the probability that a particular chemical will cause genetic damage in that animal. The first processes considered are environmental production and distribution, and the probable modes of human exposure. Drug metabolism and chemical reactions with target molecules are also reviewed, as is DNA repair. The final consideration is physiological expression or suppression, and the long latent period between carcinogen treatment and tumor appearance. Four genetic endpoints are discussed in terms of assaying for genetic hazards: gene locus mutation, chromosome breaking (clastogenesis), recombination, and DNA repair. It is not true that a recombinogen is a clastogen, mutagen, or a carcinogen. The different genetic endpoints do seem to have different molecular etiologies, however, although it is not now known what they are. The diploid human lymphoblast can be used effectively in gene locus mutation assays, in the measurement of recombination, in enumerating chromosome aberrations, in measuring DNA repair by unscheduled DNA synthesis or other techniques, and in the direct measurement of cellular toxicity. (12 refs)

79-4209 The Problem of the Carcinogenic Risk by Furocoumarins. (Eng) Rodighiero, G. (Istituto

di Chimica farmaceutica dell'Università, Padua, Italy). *Prog Biochem Pharmacol* 14: 94-103; 1978.

The possible carcinogenic effects of the furocoumarins (FC) that may be introduced into the human body are reviewed. Two types of interaction can occur between FC and DNA: in the absence of light, a molecular complex is formed that involves very weak bonds, a relatively small association complex, and no biological effects; under irradiation with longwave UV light, a C₄-cyclo addition takes place between FC and the pyrimidine bases, especially thymine, of DNA. FC may form difunctional adducts with two pyrimidine bases or monofunctional adducts. Both mono- and difunctional adducts are very stable. The second type of interaction results in lethal, inhibitory, and mutagenic effects. A carcinogenic effect is correlated with the damaged DNA and with the possibility that the damage is repaired by the enzymatic systems in living cells. The initial properties of the DNA are less efficiently restored after the repair of difunctional adducts. In mice, epicutaneous application of FC and UV irradiation appear to produce cutaneous tumors. No tumors are seen after po administration. There is no evidence of cancer developing in humans after the therapeutic use of FC for vitiligo or psoriasis. (43 refs)

79-4210: Available Thyroid Protection (Letter to Editor). (Eng) von Hippel, F. (Center for Energy and Environmental Studies, Princeton Univ., Princeton, NJ 08540). *Science* 204(4397): 1032; 1979.

The absorption of radioactive iodide by the thyroid following a nuclear reactor accident could be blocked by the ingestion of large doses of nonradioactive potassium iodide. To be most effective in preventing radiation-induced thyroid tumors, potassium iodide pills would have to be taken before the cloud of radioiodine arrived. Stockpiling accompanied by a public information program and a rapid distribution system should, therefore, be organized without delay. (6 refs)

79-4211 Modes of Alcohol Administration Appropriate for the Study of the Role of Alcohol in Carcinogenesis. (Eng) Lester, D. (Center Alcohol Studies, Rutgers Univ., New Brunswick, NJ 08903). *Cancer Res* 39(7, part 2): 2891-2893; 1979.

The route of alcohol administration used in animal studies of the role of alcohol in carcinogenesis should mimic the human condition. The po route should, therefore, have major attention. This route is linked with epidemiological evidence of alcohol involvement in cancers of the upper digestive tract. The relative advantages of schedule-induced polydipsia, with an alcohol soln being used as the sole fluid source, and an alcohol-containing fluid diet are assessed, as

well as the intake of alcohol via the respired air. These routes allow a wide range of daily alcohol doses to be ingested, the largest resulting in continuous intoxication and the development of physical dependence. The equivocal results from various published experiments indicate the need for using a variety of appropriate dosages, dosage routes, and dosage forms in appropriate controls and for investigating the role of concentration and kind of alcoholic beverage in a variety of strains and species in both sexes and at various ages. (29 refs)

79-4212 Possible Relationships of Alcohol in Membranes to Cancer. (Eng) Freund, G. (Res. Service 151, Veterans Admin. Medical Center, Gainesville, FL 32602). *Cancer Res* 39(7, part 2): 2899-2901; 1979.

Ethanol-induced alterations in membranes and their relationships to cancer biology are explored. Ethanol alters membrane fluidity and/or composition and, as a result, it may affect the induction, growth, spread, or treatment of cancers. Ethanol rapidly equilibrates with total body water and enters all cell membranes. Ethanol molecules are intercalated between the lipids of the bilayer membranes. This expands membranes and increases their fluidity, which in turn affects cell agglutination, phagocytosis, membrane transport, membrane enzyme activities, and many other membrane functions. After 3-5 days of continuous ethanol administration, the original membrane fluidity is restored by the incorporation of "stiffening" lipids, such as cholesterol, into the bilayer and by an increase in the chain length and saturation of fatty acids. The desired membrane effects (increased fluidity or altered membrane composition) can be obtained by adjusting time-dose relationships of ethanol administration. There may be an important role of moderate alcohol consumption in cancer biology that is not presently recognized by epidemiological studies, because both cancer and moderate alcohol consumption are very prevalent in the general adult population. Moderate, social alcohol use could potentially suppress or enhance the induction, growth, spread, or therapy of cancers. Such potential roles of alcohol in cancer biology could easily be tested in animals by incorporating the feeding of alcohol-containing diets into experiments that follow standard cancer protocols. (36 refs)

79-4213 Etiological and Preventive Implications in Alcohol Carcinogenesis. (Eng) McCoy, G. D. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595); Wynder, E. L. *Cancer Res* 39(7, part 2): 2844-2850; 1979.

The current state of knowledge of the epidemiological association of alcohol and tobacco consumption with cancers of the head and neck in humans is reviewed. There are four possible mechanisms that can be envisioned for the

association between alcohol and cancer: alcohol as a solvent, alcohol-induced increases or decreases in liver metabolism, or alcohol-induced alterations in target tissue metabolism. The possible involvement of alcohol-associated nutritional deficiencies in the etiology of head and neck cancer is considered. Data are presented that indicate that the in vitro metabolism of the hepatocarcinogen N-nitrosopyrrolidine is increased in microsomal fractions isolated from ethanol-consuming hamsters. Relevant studies in experimental animals are discussed in the context of possible mechanisms that could account for the increased risk of cancer in heavy drinkers who smoke. (118 refs)

- 79-4214 Alcohol-related Diseases and Carcinogenesis. (Eng) Lieber, C. S. (Alcoholism Res. and Treatment Center, Bronx Veterans Admin. Medical Center, Bronx, NY); Seitz, H. K.; Garro, A. J.; Worner, T. M. *Cancer Res* 39(7, part 2): 2863-2886; 1979.

Some medical complications of alcoholism are described, especially with regard to their interrelationships with carcinogenesis. Alcohol may contribute to carcinogenesis via contact-related local effects, the induction of microsomal enzymes that activate procarcinogens, general mechanisms of tissue injury and regeneration (particularly in the liver), or associated nutritional disturbances. The relationship between alcohol-induced cirrhosis and hepatocellular carcinoma is also explored, and case histories of patients with hepatocellular carcinoma in the absence of cirrhosis are reviewed. Data are presented demonstrating the induction, by chronic ethanol consumption, of microsomal enzymes that convert procarcinogens to carcinogens. These data were derived from experiments in which the ability of microsomes isolated from liver, intestine, and lung tissues of ethanol-fed and control rats to activate several test carcinogens was examined in the Ames *Salmonella* mutagenicity test. It is hypothesized that the ethanol-mediated induction of enzyme systems that activate procarcinogens to carcinogens contributes to the enhanced incidence of cancer in alcoholics. (412 refs)

- 79-4215 Exposure to Leukemia (Letter to Editor). (Eng) Weber, F. E. (R.T. French Co., Rochester, NY). *Chem Eng News* 57(22): 48; 1979.

An article suggesting a link between leukemia and the chemical sterilant mixture 50-50 methyl formate (MF) and ethylene oxide (EO) is criticized. The authors had no data to demonstrate actual employee exposure to this mixture when the leukemia occurred in three employees. Several years later, they measured the EO concentration for an unspecified time and reported it at about 20 ppm time-weighted av. MF was dismissed as an unlikely coconspirator because it is a food additive. In addition, the authors made no distinction between the po and inhalation routes. (1 ref)

- 79-4216 Environmental Sources of Chemical Mutagens. II. Synthetic Mutagens. (Eng) Fishbein, L. (Natl. Cancer for Toxicological Res., Jefferson, AR). *Adv Mod Toxicol* 5: 257-348; 1978.

Comparative data are reviewed on the relative amounts, residues, and transport in the environment of primarily synthetic mutagenic and potentially mutagenic agents. The agents discussed include industrial mutagens (primarily the halogenated hydrocarbons, vinyl chloride, vinylidene chloride, trichloroethylene, tetrachloroethylene, chloroprene, carbon tetrachloride, fluorocarbons, polychlorinated biphenyls, chlorodioxins, haloethers, chloride, bromoalkanes, and fluorine, phthalate esters, pesticides (DDT, dichlorvos, formaldehyde, and ethylene oxide), and metals and metalloids (lead, mercury, and arsenic). Information is sparse concerning environmental reactions and interactions, transport, residence times, stability, and fates of these agents. Also, most environmental chemicals have not been adequately studied to permit evaluation of their relative mutagenicities for humans. (470 refs)

- 79-4217 The *Salmonella*/Microsome Mutagenicity Test: Predictive Value for Animal Carcinogenicity. (Eng) McCann, J. (Dept. Biochemistry, Univ. California, Berkeley, CA); Ames, B. *Adv Mod Toxicol* 5: 87-108; 1978.

The value of the Ames *Salmonella*/microsome mutagenicity test as a predictive tool for chemical carcinogens is reviewed. There is a >1-millionfold variation in the mutagenic potency of chemicals as detected by this test. Preliminary studies indicate that the results of the in vitro mutagenicity test are in good agreement with the results of animal carcinogenicity tests. The test has been validated with about 300 organic chemicals that had been tested in conventional animal carcinogenicity tests. The results showed a striking correlation between carcinogenicity and mutagenicity; 90% (157/175) of the carcinogens tested were mutagenic in the test, and 87% (94/108) of the noncarcinogens were nonmutagens. Most of the false positives and negatives are explainable by inadequate animal carcinogenicity tests or by inadequacies in the in vitro metabolic activation system. The sensitivity of the *Salmonella* test may make it useful for detecting chemicals that have weak carcinogenic activity. It is being used for setting priorities in selecting chemicals for carcinogenesis bioassay in animals. Environmental chemicals have been detected as mutagens in in vitro tests and subsequently identified as animal carcinogens, eg, furylfuramine, ethylene dibromide, 4-nitro-o-phenylenediamine, and 2-nitro-p-phenylenediamine. The work with mutagens in the *Salmonella* system supports the theory that radiation and chemical carcinogens cause cancer through damage to DNA (somatic mutation). (67 refs)

- 79-4218 **Cancer-Fluoridation Link (Letter to Editor).** (Eng) Burk, D. (Dean Burk Foundation, Washington, DC). *Chem Eng News* 57(22): 48-49; 1979.

Studies that contradict the statement that "recent reports that fluoridated drinking water causes cancer do not appear to stand up under close scrutiny" are documented. These studies indicate not a statistically insignificant 0.19% increase in cancer mortality among the 100 million Americans drinking water containing 1 ppm fluoride, as suggested in the previous report, but a highly significant 5% increase. (no refs)

- 79-4219 **Polarography and Voltammetry in Studies of Toxic Metals in Man and His Environment.** (Eng) Nurnberg, H. W. (Inst. Chemistry, Inst. 4 Applied Physical Chemistry, Nuclear Res. Centre, Juelich, W. Germany). *Sci Total Environ* 12(1): 35-60; 1979.

The use of advanced polarographic and voltammetric methods in studies of toxic metals affecting man and the environment is compared with the use of relevant nonelectrochemical alternatives. Representative applications are surveyed, including investigations of human body fluids and organs, all types of environmental compartments, and food chains to man. The potential application of electrochemical methods to speciation studies of dissolved toxic metals in natural waters is also discussed. (63 refs)

- 79-4220 **Target Organs: The Blood.** (Eng) Waldron, H. A. (TUC Centenary Inst. Occupational Health, London Sch. Hygiene and Tropical Medicine, Keppel St., London WC1E 7HT, England). *J Soc Occup Med* 29(2): 65-71; 1979.

The effects of toxic agents (including lead, arsine, benzene, trinitrotoluene, and radiation) on the blood are reviewed by considering each stage of RBC maturation (production of precursor cells, hemoglobinization and maturation, circulating RBC) separately. Some agents exert their effect at more than one stage. (60 refs)

- 79-4221 **Inducibility by Chemical Mutagens of Heritable Translocations in Male and Female Germ Cells of Mice.** (Eng) Generoso, W. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN); Cain, K. T.; Huff, S. W.; Gosslee, D. G. *Adv Mod Toxicol* 5: 109-129; 1978.

The inducibility by chemical mutagens of heritable translocations in various germ-cell stages in mice is discussed in relation to other end points of chromosome breakage effects (dominant-lethal mutations, sex chromosome loss, cytologically scored aberrations and heritable inversions).

Heritable translocations are the most reliable end point of chromosome breakage induced in postmeiotic stages in males. Triethylenemelamine (TEM) induced a significant increase in heritable translocations in mouse spermatids at a dose of 0.0125 mg/kg, far below the lethal dose. Results with 6-mercaptopurine gave the first evidence that a nonalkylating chemical can induce chromosome breakage in the germ line of mice. Chromosome breakage was induced only in late-differentiating spermatogonia and very early spermatocytes. In this case, dominant-lethal mutations and cytologically scored breaks appeared to be more sensitive end points than heritable translocations. One of the most striking differences between radiation and chemical mutagens is that radiation is highly effective in inducing chromosome breakage in spermatogonia stem cells, whereas alkylating chemicals are ineffective. Isopropyl methanesulfonate (IMS), which is an effective inducer of dominant-lethal mutations in dictyate oocytes, did not significantly increase the incidence of translocations in cells that were transmissible to male offspring. A sequential procedure for the detection of male translocation heterozygotes by fertility testing enables a large percentage of males to be declared fertile by counting the number of live births in, at most, three litters. The procedure is being used in both chemical and radiation experiments. (30 refs)

- 79-4222 **Drug-induced Diseases. Drug-induced Haematological Diseases.** (Eng) Dawson, A. A. (Dept. Medicine, Univ. Aberdeen, Foresterhill, Aberdeen AB9 2ZD, Scotland). *Br Med J* 1(6172): 1195-1197; 1979.

Drug-induced hematological diseases, including marrow hypoplasia, thrombocytopenia, neutropenia, acute myeloid leukemia, dyserythropoiesis (megaloblastosis and sideroblastic anemia), hemolytic anemia, chronic posthemorrhagic anemia, and hemostatic defects, are reviewed. There is considerable evidence that marrow rendered hypoplastic by a drug is much more liable to develop acute myeloid leukemia (AML) than normal marrow. There is a high index of suspicion about the induction of AML by melphalan. Immunosuppressants are associated with a high risk of lymphoma, and drugs used in Hodgkin's disease may be associated with the development of acute leukemia. (no refs)

- 79-4223 **Mutagenic, Clastogenic and Oncogenic Effects of 1- β -D-Arabinofuranosylcytosine.** (Eng) Benedict, W. F. (Dept. Pediatrics, USC Sch. Medicine, Los Angeles, CA 90027); Jones, P. A. *Mutat Res* 65(1): 1-20; 1979.

Current knowledge of the cytotoxic, teratogenic, clastogenic, mutagenic, and oncogenic actions of 1- β -D-arabinofuranosylcytosine (ara-C) are reviewed.

Ara-C inhibits viral, bacterial, and eukaryotic DNA synthesis, probably via varying degrees of inhibition of the different polymerase enzymes. The most likely explanation for its teratogenic effect is its ability to cause unusual amounts of cell death, particularly in localized regions. Its cytotoxic action appears to be closely associated with its ability to produce chromatid breakage. Ara-C can also produce point mutations and chromosome breaks and rearrangements via incorporation into DNA as a fraudulent nucleoside replacing cytosine. It is possible that ara-C could cause significant genetic changes in humans at the germ cell level. Ara-C produces both chromosome- and chromatid-type aberrations. There is considerable evidence that the mechanism(s) involved in the production of chromatid breaks are different from those causing sister-chromatid exchanges. Ara-C may be able to effect neoplastic changes via DNA damage. The specific chromosome aberrations produced by ara-C may derepress viral or nonviral oncogenic information, which subsequently may be expressed as a transformed cell. (130 refs)

- 79-4224 N-Nitroso Compounds in the Workplace. (Eng) Fine, D. H. (Cancer Res. Div., Therma Electron Corp., 45 First Ave., Waltham, MA 02154). *ACS Symp Ser* (94): 247-254; 1979.

The occurrence and measurement of N-nitroso compounds in the workplace are reviewed. Analysis of the N-nitroso compounds at the trace level is difficult, the most widely used technique involving the use of a nitrosamine specific detector interfaced to a gas chromatograph or a high pressure liquid chromatograph. High resolution mass spectrometry is used for structural confirmation of the N-nitroso compounds. Analysis of ambient air is best carried out using a mobile laboratory. The simplest dialkyl N-nitrosamines are relatively volatile and airborne N-nitrosamines are to be expected in the vicinity of chemicals which are contaminated with nitrosamines. These compounds have been reported in the atmospheres of rocket fuel factories and surroundings, amine factories, rubber and tire industrial facilities, leather tanneries, tobacco smoke, and a variety of other sources. They have not been found to any significant extent in the ambient air of New York City, Boston, or upstate New Jersey, and they appear to remain in cooking vapors for < 1 hr. (45 refs)

- 79-4225 Carcinogen Activation and the Biological Detection of Activated Metabolites. (Eng) Garner, R. C. (Cancer Res. Unit, Univ. York, Heslington, York YO1 5DD, England). *J Soc Occup Med* 29(2): 54-60; 1979.

Studies of the activation of organic carcinogens and several short-term mutagenicity/carcinogenicity assays are reviewed. Most organic chemicals do not initiate cancer directly

but require conversion to some other species in the body. It has been postulated that reactive chemical species are formed for a wide variety of chemical carcinogens, including nitrosamines, polycyclic hydrocarbons, aflatoxins, unsaturated halogenated hydrocarbons, etc. Metabolic activation for many carcinogens, however, is a minor route of metabolism. In compounds containing aromatic rings, the major routes of metabolism yield nontoxic derivatives. For a large number of compounds, organ sensitivity will depend on the balance between the ability of an organ to activate a particular compound and its ability to detoxify the chemical or its reactive metabolites. Bacterial mutagenesis assays have used either *Salmonella typhimurium* or *Escherichia coli* as detector strains. There are several objections to the methodology of such tests, but there is no doubt that bacterial mutagenicity does permit the screening of compounds for potential carcinogenicity. Other approaches that might be useful for the screening of chemicals for carcinogenic activity include mammalian cell transformation and mutation and the measurement of unscheduled DNA-repair synthesis in mammalian cells. (15 refs)

- 79-4226 Consequences of the AF-2 Incident in Japan. (Eng) Tazima, Y. (Natl. Inst. Genetics, Mishima, Shizuoka-ken, Japan). *Environ Health Perspect* 29: 183-187; 1979.

The significance of the discovery that the food preservative 2-(2-furyl)-3-(5-nitro-2-furyl) acrylamide (AF-2) is a potent mutagen is discussed. This discovery promoted the development of sensitive and reliable tester *Salmonella* strains and supported the hypothesis that there is a common mechanism between mutagenicity and carcinogenicity. Thus, preliminary screening for carcinogens has become feasible using mutagenicity as an index. The discovery also promoted legislation requiring mutagenicity testing of food additives in Japan. (36 refs)

- 79-4227 Simple Theoretical Criterion of Chemical Carcinogenicity - A Refutation. (Eng) Rosenblatt, D. H. (U.S. Army Medical Bioengineering Res. and Development Lab., Fort Detrick, Environmental Protection Res. Div., Frederick, MD 21701); Dacre, J. C. *Experientia* 35(4): 567-568; 1979.

The carcinogenicity of chemicals cannot be predicted by their quasi-valence number as stated in a previous article. According to this criterion, dimethyl sulfate and N-nitroso-N-methylurea, two known carcinogens, would be classed as noncarcinogens and water and ammonia would be classed as carcinogens. (5 refs)

79-4228 The Use of Mutagenicity Tests in Screening Chemical Carcinogens. (Eng) Montesano, R. (Unit Chemical Carcinogenesis, International Agency Res. Cancer, Lyon, France). *Prog Biochem Pharmacol* 14: 157-162; 1978.

The correlation between the mutagenicity and carcinogenicity of N-nitroso compounds and the relationships of in vitro tests to carcinogenic processes in vivo are reviewed. The 24 nitrosamines tested for mutagenicity were inactive in a variety of systems without metabolic activation, whereas 24/27 nitrosamides were active. When 23 nitrosamines were tested with mammalian metabolic activation, 17 carcinogenic nitrosamines were active. Of 47 N-nitroso compounds (23 nitrosamides and 24 nitrosamines), 38 were carcinogenic and mutagenic, 5 carcinogens were nonmutagenic, 1 noncarcinogen was mutagenic, and 3 noncarcinogens were nonmutagenic. The results demonstrate the validity of these tests as preliminary screens for potentially carcinogenic compounds, but they cannot substitute for long-term carcinogenicity tests in animals. Among the factors that may interfere with the determination of the mutagenicity of a chemical are the balance between activation and detoxification processes, chemical stability and differential reactivity of the ultimate metabolite(s), bacterial metabolic activation, the amount of microsomal enzymes, and mutagenic specificity. (17 refs)

79-4229 Overall Health Hazards of Environmental Chemicals. (Ger) Schulze, H. (Saarlauterner Strasse 109, 8000 Munich 50, W. Germany); Mucke, W. *ZFA (Stuttgart)* 55(10): 609-623; 1979.

Health hazards associated with environmental chemicals are surveyed. Sixty to ninety percent of all cancer cases can be attributed to environmental chemicals, with nitrosamines and aflatoxins being the most hazardous carcinogens. (13 refs)

79-4230 Cholesterol and Colon Cancer (Letter to Editor). (Eng) Lewis, B. (Dept. Chemical Pathology and Metabolic Disorders, St. Thomas's Hosp. Medical Sch., London SE1 7EH, England). *Lancet* 1(8126): 1136-1137; 1979.

In response to the recently offered hypothesis that dietary cholesterol is a co-carcinogen for colonic neoplasia, strong support is offered for the hypothesis that a high-fat diet increases fecal bile-acid concentrations, thus promoting colonic cancer. Dietary recommendations to reduce fecal bile-acid concentrations would include a reduction of fat, and hence cholesterol, intake. (18 refs)

79-4231 Food Toxins and Their Implication in Human Health. (Eng) Tulpule, P. G. (Natl. Inst. Nutrition, Hyderabad 500007, India); Bhat, R. V. *Indian J Med Res* 68(Suppl): 99-108; 1978.

Studies concerning mycotoxins, aflatoxin, enteroergotism, toxins in edible oils, and toxic alkaloids in contaminated weed seeds are reviewed with respect to human health implications. Aflatoxin has been unequivocally demonstrated to be a potent carcinogen in several species, and cholangiocarcinomas and hepatocellular carcinomas have been observed in monkeys given aflatoxin ip and po. (31 refs)

79-4232 Research at the Northern Regional Research Center. (Eng) Inglett, G. E. (Northern Regional Res. Center, SEA-USDA, Peoria, IL); Schaefer, W. C.; Tallent, W. H. *Cereal Foods World* 24(5): 186-188; 1979.

Some food quality and safety research carried out by the Northern Regional Research Center of the U.S. Department of Agriculture is outlined. This research has included the development of procedures for the detection of and protection against aflatoxins in foods. Detection methods include a rapid presumptive test to locate lots of contaminated corn, screening procedures to determine the presence or absence of toxin; and precise quantitative methods. Contaminated cereals can be detoxified by ammoniation. (12 refs)

79-4233 Relation Between Aflatoxin, Hepatitis-B Virus, and Hepatocellular Carcinoma. (Eng) Lutwick, L. I. (Div. Infectious Diseases, Dept. Internal Medicine, Univ. Iowa Hosp. and Clinics, Iowa City, IA 52242). *Lancet* 1(8119): 755-757; 1979.

Evidence is presented to support the hypothesis that aflatoxin (AF) is an indirect, rather than a primary, cause of liver cancer in humans. Hepatitis B virus (HBV) antigens and antibodies are significantly more common in hepatoma patients than in the general population. A human hepatoma cell line has been derived that produces HBV surface antigen (HBsAg) but not the internal (core) antigen of the virus. This suggests that only part of the HBV genome is present in the cell line and is integrated into the cell genome. In one study, some of the DNA extracted from a chronically infected liver, which was homologous with HBV DNA, had a much higher mol wt than small circular HBV DNA; ie, part of the viral genome was probably combined with the liver cell DNA. Oncogenesis by a DNA virus requires integration of viral DNA into cellular DNA. Data show that an increased intake of AF in areas of Africa is correlated with the hepatoma rate. If AF ingestion were a major factor in the predisposition to hepatoma, the risk of

cancer would be expected to be higher for HBV carriers from Mozambique (highest AF intake area of Africa) than those from the US (low AF intake). However, the risks of hepatoma development in HBV carriers in Mozambique and the US were found to be similar, suggesting that AF ingestion does not affect the development of liver cancer directly. Therefore, the difference between the hepatoma rates in the two countries is probably related to the HBV carrier rate in the population. AF may act primarily as an immunosuppressive agent, causing an increase in HBV carriers. If this hypothesis proves to be correct, a vaccine to prevent hepatitis B could be used to eradicate hepatoma. (34 refs)

79-4234 Saccharin plus Cigarettes Tied to Bladder Ca in Men. (Eng) Anonymous (No affiliation given). *Med World News* 20(11): 10-11; 1979.

The possibility that not saccharin (SC) alone but SC plus cigarette smoking increases the risk of bladder cancer (BIC) in men has emerged from a dispute over new and old interpretations of old epidemiological data. The author of a study of 260 men and 86 women with BIC and 2,235 men and 1,602 women without BIC had initially concluded that there was no association between SC use and BIC. However, the author's second look at his data showed that the cancer risk was almost triple for men who consumed SC and were also moderate cigarette smokers. This reversal, which was sent to the National Cancer Institute in November 1977, took 18 mo to surface. (no refs)

79-4235 Phenolics in Aquatic Ecosystems: A Selected Review of Recent Literature. (Eng) Buikema, A. L. (Center Environmental Studies, Virginia Polytechnic Inst. and State Univ., Blacksburg, VA 24061); McGinniss, M. J.; Cairns, J. *Marine Environ Res* 2(2): 87-181; 1979.

The extensive literature dealing with the fate and effects of phenolic compounds in aquatic ecosystems is reviewed. Approx 96% of the total phenolics produced in the US are synthetic and 4% are naturally occurring. The toxicity of phenolics has been studied on selected microbes, algae, duckweed, and numerous vertebrates and invertebrates. The toxicity of phenolics varies with the type, position, and number of substitutions on the parent molecule. Environmental factors affect the toxicity of phenolics, and these include photolytic action, microbial degradation, pH, and water hardness and temperature. The age and size of the test organisms influence their sensitivity to phenolics, as does the presence of oxygen. Studies of the biological effects of phenolics are limited and varied. Fish detoxify phenolics by forming conjugate glucuronides and sulfates. Body burdens vary with exposure time and concentration. (388 refs)

79-4236 Mutagenic Effect of Aromatic Epoxy Resins (2 Letters to Editor). (Eng) Granville, G. C. (Group Toxicology div., Shell International Petroleum Co. Ltd, Shell Centre, London SE1, England); Andersen, M.; Binderup, M. L.; Kiel, P.; Larsen, H.; Maxild, J. *Nature* 279(5711): 352; 1979.

In response to a recent article ascribing a genetic hazard and cancer risk for humans exposed to aromatic epoxy resins (AER), the preponderance of available evidence obtained in animals indicates that currently used bisphenol acetone-based resins present no carcinogenic hazard. The authors of the original article reply that the possibility of dangerous effects associated with AER are in no way disproved by animal carcinogenicity tests, which are insensitive and will identify only strong carcinogens. (11 refs)

79-4237 Trenimon: Biochemical, Physiological and Genetic Effects on Cells and Organisms. (Eng) Obe, G. (Institut für Genetik, Freie Universität Berlin, Arminiallee 5-7, D-1000 Berlin 33, W. Germany); Beek, B. *Mutat Res* 65(1): 21-70; 1979.

Some of the chemical and physicochemical properties of the mutagenic cancer chemotherapy drug Trenimon, its action on cellular metabolism, its cytostatic and therapeutic properties, and its carcinogenic and mutagenic activities are reviewed. Trenimon interferes with the genetic material of a variety of organisms and test systems, inducing point and chromosome mutations, sister-chromatid exchanges, recombination phenomena, and phage induction. DNA damage, especially the induction of cross-links, seems to be the common mechanism for most of the effects of the drug on cells and organisms. Treatment of 48 male rats with Trenimon (0.03 mg/kg/wk iv, for 52 wk) produced malignant tumors in 24% of the animals, vs 6% of the 89 controls, with an induction times being 16 and 23 mo, respectively, for the two groups. (187 refs)

79-4238 Tumor-specific Transplantation Antigens of Chemically Induced Tumors. (Eng) Lennox, E. S. (Salk Inst. Biological Studies, San Diego, CA); Sikora, K. *Adv Pathobiol* (6): 68-78; 1977.

Studies of tumor-specific transplantation antigens (TSTA) in chemically induced tumors are reviewed. Tumors induced in inbred mice and rats by polycyclic hydrocarbons, eg, methylcholanthrene (MC), each express a unique antigen. In transplantation assays, independently arising tumors show distinct non-cross-reacting antigens. Tumors arising in the same animal but at two different MC injection sites are not cross-reacting. Chemically induced sarcomas share antigens with embryos, but extensive experiments with rat sarcomas show that TSTA are not embryonic antigens. Genes in or near the major histocompatibility locus do not

appear to influence susceptibility to tumorigenesis by MC. TSTA are not synthesized or controlled by genes of the histocompatibility antigens (H-2) complex; their expression is influenced by chromosome 17, the chromosome that carries the locus for H-2. Some experiments have given results that suggest a cross-reaction between TSTA and H-2; however, others do not support this connection. Recent experiments demonstrate a predominant role played by the H-2 antigens as specific targets for immune attack by cytotoxic lymphocytes (CTL). If CTL are stimulated by a cell having a certain H-2 and a non-H-2 antigen, their specificity is determined by both the H-2 and the non-H-2 antigen. This finding may explain the diversity of TSTA. Furthermore, the TSTA are a result of the interaction of new proteins generated in carcinogen-induced transformation with H-2, and if these proteins could interact with each other as well as with H-2, a small number of them might produce a large number of non-cross-reacting specificities. (19 refs)

- 79-4239 Chromatographic Methods of Determining Polynuclear Aromatic Hydrocarbons in the Environment.** (Rus) Korol', A. N. (Inst. Physical Chemistry, Kiev, USSR); Lysiuk, L. S. *Zh Anal Khim* 34(3): 577-590; 1979.

Data on the efficacy of various chromatographic methods for determining levels of polynuclear aromatic hydrocarbons in the atmosphere are reviewed. The most reliable method was concluded to be a combination of gas chromatography and mass spectrometry. (150 refs)

- 79-4240 Estimating Maximum Limits to Mutagenic Potency From Cytotoxic Potency.** (Eng) Carver, J. H. (Lawrence Livermore Lab., Biomedical Sciences Div., Univ. California, P.O. Box 5507, Livermore, CA 94550); Hatch, F. T.; Branscomb, E. W. *Nature* 279(5709): 154-156; 1979.

The cytotoxic potency of 22 chemical mutagens was compared with their mutagenic potency in five rodent and human in vitro cell systems. The D_{37} unit of survival (the dose required to kill approx 63% of the initial cell population) was used to measure cytotoxic potency. The relative increase in cytotoxicity of the chemicals was accompanied by a proportional increase in mutagenicity, suggesting that a determination of the D_{37} dose of toxic agents can be used to estimate the maximum potential-induced mutation frequency. It is hypothesized that whereas mutagenicity and cytotoxicity range over six orders of magnitude, no agent can induce more than approx one forward mutation at a given locus per 100 cells surviving at the D_{37} dose. If this hypothesis is valid, this is a biological limit to mutagenic potency that is demonstrated by compounds whose cytotoxicity is due solely to mutational events. The

dynamic range for forward mutations in mammalian in vitro systems is approx four orders of magnitude. The relative mutagenicities of different compounds at a given cytotoxicity was not significantly related to the class of mutagen or the forward mutation marker scored. (43 refs)

- 79-4241 Health Factors in Urban Wastewater Master Plans.** (Eng) Bosker, C. M. (John Taylor & Sons, London, England); Kell, A. D. *Prog Water Technol* 11(1): 201-208; 1978.

The health effects of urban wastewater disposal in developing countries are considered. Human diseases that are transmitted by or are associated with water fall into two categories: (1) diseases caused by living organisms and (2) diseases caused by organic or inorganic substances carried in water, eg, cancer caused by organic halogens or polynuclear aromatic hydrocarbons in wastewater effluents. Group 2 diseases are of little significance in most developing countries, but they may become more so when improved sanitation and living conditions reduce the incidence of Group 1 diseases. The health aspects of the existing situation must be ascertained before viable alternative strategies can be determined. A review of several studies shows that the degree of health improvement that can be expected from wastewater improvements depends on the original health characteristics of the community, cultural habits, educational level, physical environment, and socioeconomic factors. Since incomes are generally low in developing countries, it is often necessary that wastewater facilities be subsidized by public funds. (5 refs)

- 79-4242 Inverse Correlation Between Species Life Span and Capacity of Cultured Fibroblasts to Metabolize Polycyclic Hydrocarbon Carcinogens.** (Eng) Schwartz, A. G. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA 19140); Moore, C. J. *Fed Proc* 38(6): 1989-1992; 1979.

Many investigators have hypothesized that aging may result from an accumulation of DNA damage. If valid, this hypothesis necessitates a means by which this accumulation can be related to the potential life-span of an organism. A cell-mediated mutagenesis assay was used to test diploid fibroblast strains from six mammalian species of widely differing life-spans (rat, guinea pig, rabbit, horse, elephant, and human). Very good inverse correlations were found between species life-span and ability to activate 7,12-dimethylbenz(a)anthracene (DMBA) to mutagenic forms and between species life-span and ability to activate DMBA to forms capable of covalent binding to DNA. Since polycyclic hydrocarbon carcinogens such as DMBA and benzo(a)pyrene (BP) must be metabolically activated by mixed-function oxidases to their biologically active forms, these data indicate that the capacity of fibroblasts to ac-

tivate polycyclic hydrocarbon carcinogens to DNA-damaging forms is a species property related to potential life-span. To determine the role of carcinogen metabolism in this phenomenon, the capacity of diploid fibroblasts from eight mammalian species to convert BP and DMBA to water-soluble metabolites was determined. This rate of conversion varied widely among different species and showed a very good inverse correlation with species life-span. As a whole, these findings suggest that the ability of cultured cells to metabolize polycyclic hydrocarbon carcinogens is related to species life-span and may be important in the occurrence of spontaneous cancer. (24 refs)

- 79-4243 Effects of Vitamin A and Related Retinoids on the Biochemical Processes Linked to Carcinogenesis.** (Eng) Boutwell, R. K. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706); Verma, A. K. *Pure Appl Chem* 51(4): 857-866; 1979.

Retinoids that inhibit skin tumors induced by 12-O-tetradecanoylphorbol-13-acetate (TPA) were studied for their effects on ornithine decarboxylase (OD) activity. When applied topically, retinoic acid [RA, 1.7 nanomoles (nmol)] inhibited the induction of OD and the promotion of epithelial tumors in mice by TPA (17 nmol). RA had no effect on S-adenosylmethionine decarboxylase activity or the incorporation of ³H-leucine by epidermal proteins. Mice were initiated with topical 7,12-dimethylbenz(a)anthracene (0.2 nmol) and promoted with TPA (17 nmol twice weekly). RA (17 nmol) applied several hours before or up to 3 hr after TPA application inhibited the induction of OD, max inhibition occurring when RA was applied 1 hr before TPA. No inhibitory effect was observed when RA was applied 16 hr or more before TPA. A similar time schedule was observed for the inhibition of TPA-induced tumors by RA. RA did not inhibit OD induction at a dose of 0.0034 nmol, but resulted in essentially complete inhibition at 3-4 nmol. RA given by stomach tube also effectively inhibited TPA-induced OD in a dose-dependent fashion between 0.034 and 0.34 micromole. There was a close parallel in the ability of several synthetic retinoids to inhibit OD activity and their ability to inhibit tumorigenesis. The data suggest that OD may play an obligatory role in carcinogenesis. (19 refs)

- 79-4244 Metabolic Activation/Deactivation Reactions During Perinatal Development.** (Eng) Lucier, G. W. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); Lui, E. M.; Lamariniere, C. A. *Environ Health Perspect* 29: 7-16; 1979.

The role of metabolic activation/deactivation reactions during development is evaluated in relation to developmental pharmacology and toxicology. Enzyme systems evaluated include the mixed-function oxidases (aryl

hydrocarbon hydroxylase and oxidative demethylation), epoxide hydration and conjugation (glutathione conjugation, sulfation, and glucuronidation). Placental transfer and milk secretion of chemicals are discussed in relation to maternal, placental, and fetal metabolism. Normal patterns of enzyme development can be modified in two ways: (1) enzyme induction and (2) enzyme imprinting. Postnatal induction of the mixed-function oxidases and glucuronyltransferase following treatment of pregnant rats with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is shown to be caused primarily by newborn exposure to TCDD in milk. Structure-activity relationships are defined for the perinatal induction of hepatic enzymes by the pure polychlorinated biphenyls (PCB's). PCB's are divided into two classes: P-450 inducers and P-448 inducers. Imprinting or programming of hepatic metabolism is a function of the sexual differentiation of enzyme activity; male and female activities are similar in prepubertal animals, whereas pronounced sex differences are evident in adults. Treatment of newborn rats (days 2-6) with diethylstilbestrol or testosterone resulted in a feminization (decrease) of mixed-function oxidation and glucuronidation in adult males. No changes were seen in immature males or females or adult females. This effect appears to be irreversible and is under pituitary-hypothalamic-gonadal control. In addition to the feminization of enzyme activity, neonatal exposure to hormonally active chemicals also feminizes the hepatic response to cadmium in resultant adult animals. (71 refs)

- 79-4245 Environmental Sources of Chemical Mutagens. I. Naturally Occurring Mutagens.** (Eng) Fishbein, L. (Natl. Center Toxicological Res., Jefferson, AR). *Adv Mod Toxicol* 5: 175-256; 1978.

Comparative data are reviewed on the relative amounts, residues, and transport in the environment of various naturally occurring mutagenic and potentially mutagenic agents. These include mycotoxins (aflatoxins, ochratoxins, sterigmatocystin, and *Penicillium* and *Fusarium* toxins), pyrrolizidine alkaloids, cycasin, polynuclear aromatic hydrocarbons (benzo(a)pyrene, benz(a)anthracene, and dibenzanthracene), nitrosamines, and atmospheric mutagens (sulfur oxides, nitrogen oxides, and ozone). Although the agents are considered individually, the environmental hazard to humans involves a multifaceted and often continuous exposure. There is little information regarding the additive, potentiating, and synergistic effects of the agent discussed. Information is also sparse regarding environmental reactions and interactions, transport, residence times, stability, fate, and relative mutagenic hazards for humans. (534 refs)

- 79-4246 Toxicity of Oil Shale Chemical Products. A Review.** (Eng) Kahn, H. (Eksperimentaalse ja Kliinilise Meditsiini Instituut, Hiiu 42, Neuvostoeesti,

Tallinn 200015, USSR). *Scand J Work Environ Health* 5(1): 1-9; 1979.

The toxicity and carcinogenicity of oil shale chemical products to animals and humans are reviewed. All primary products of the thermal processing of Estonian oil shale appear to have a carcinogenic effect, with the activity increasing with processing temperature. The dephenolization of chamber tar increases the carcinogenicity of this product. In one study, the various oil shale tars produced skin tumors in 35%-48.5% of CC₅₇BR mice. Water-soluble oil shale phenols have been shown to be cocarcinogens. Studies of the work environments in oil shale processing plants indicate that benzo(a)pyrene is permanently present in the air, although the concentrations do not exceed the max allowable concentration (15 µg/100 m³). According to a recent investigation, workers employed in the oil shale industry for 10 yr or more show an excess skin cancer morbidity compared with that in the general population ($p < 0.05$). (42 refs)

79-4247 Ecotoxicology of Dyestuffs--A Joint Effort by Industry. (Eng) Anliker, R. (P.O. Box CH-4005, Basel, Switzerland). *Ecotoxicol Environ Saf* 3(1): 59-74; 1979.

The primary objectives of the Ecological and Toxicological Association of the Dyestuffs Manufacturing Industry (ETA) are to coordinate and unify the efforts of dyestuff manufacturers to minimize environmental damage arising from use of their products, to protect the users of the products, and to aid public institutions concerned with the ecotoxicological impact of the products. The organization and activities of ETAD are discussed. The organization provides users with safety data sheets that provide information relating to safe handling of the products. It is also involved in the development and standardization of analytical, ecological, and toxicological methods. High priority is given to the identification of particularly hazardous products and to setting up appropriate precautionary measures. However, it is important to recognize that not all compounds within a given class may be equally dangerous. For example, many azo compounds are carcinogenic, but many others are not. In setting priorities for full-scale testing programs, the important factors in risk assessment are exposure pattern, ecotoxicological profiles, and production volume. At present, unless epidemiological data are available, only long-term tests in animals give sufficiently reliable results to serve as a basis for assessing carcinogenic risks. ETAD is currently studying the behavior of different classes of dyestuffs in the Salmonella/mammalian microsome test. (30 refs)

79-4248 Cigarette Smoking--Does It Carry a Genetic Risk? (Eng) Bridges, B. A. (Medical-

Biological Lab. TNO, P.O. Box 45,, 2280 AA Rijswijk, Netherlands); Clemmesen, J.; Sugimura, T. *Mutat Res* 65(1): 71-81; 1979.

The possibility of a genetic risk associated with cigarette smoking is reviewed. Evidence indicates that cigarette smoke contains many mutagens and that persons who inhale the smoke probably absorb significant quantities. At least some of the mutagens are distributed systematically and are likely to reach the gonads. Three investigations suggest the existence of genetic damage to the circulating lymphocytes and spermatozoa of smokers. The only available study of heritable effects in man indicated a significant correlation between paternal smoking and both the rate of perinatal mortality and the frequency of congenital abnormalities. It is hypothesized that cigarette smoking may prove to be a significant genetic hazard for the offspring of smokers and for subsequent generations. (34 refs)

79-4249 Biological Activity of Tobacco Smoke and Tobacco Smoke-related Chemicals. (Eng) Kouri, R. E. (Dept. Biochemical Oncology, Microbiological Associates, 5221 River Road, Bethesda, MD 20016); Rude, T. H.; Curren, R. D.; Brandt, K. R.; Sosnowski, R. G.; Schechtman, L. M.; Benedict, W. F.; Henry, C. J. *Environ Health Perspect* 29: 63-69; 1979.

The role of tobacco smoke or smoke-related chemicals in cancer susceptibility in humans is reviewed. Exposure to whole cigarette smoke from reference cigarettes results in the prompt (peak activity is 6 hr), but fairly weak (approx twofold), induction of murine pulmonary microsomal monooxygenase (PMO) activity. This activity can be detected by using benzo(a)pyrene (BP) or ethoxyresorufin as substrates, and it can be inhibited by treatment with cycloheximide or actinomycin D. Unlike the induction of PMO's following intratracheal administration of 3-methylcholanthrene, these cigarette smoke-induced increases were not unequivocally linked to the Ah locus. Whole smoke condensate and fractions derived from these condensates can (1) induce PMO activity, (2) inhibit BP metabolism in vitro, (3) be metabolized to forms mutagenic to *Salmonella typhimurium* tester strains TA1538 or TA98, (4) transform C3H 10T1/2 cells in vitro, and (5) enhance the carcinogenicity of BP in murine pulmonary tissue. A potentially important observation is that whereas hepatic tissue is capable of activating whole cigarette smoke condensate to mutagenic forms in vitro, murine pulmonary tissue does not seem capable of such activation. Although these pulmonary-derived tissue homogenates have significant AHH activity and can metabolize aflatoxin B₁, 2-aminofluorene, and 7,8-dihydro-7,8-dihydroxybenzo(a)pyrene to mutagenic forms, they fail to activate both cigarette smoke condensate and the promutagen 6-aminochrysene. These results are discussed with reference to the concept that whole cigarette smoke may be both a

potential "initiator" and "promotor" of lung cancer in mice and that this latter property may be the most important in determining cancer risk. (35 refs)

- 79-4250 **Bronchial Carcinoma.** (Ger) Friedel, H. (Bezirkskrankenhaus für Lungenkrankheiten und Tuberkulose, DDR-3271 Löstau b. Magdeburg, E. Germany); Preisler, J. *Z Gesamte Inn Med* 34(4): 25-29; 1979.

Problems in diagnosing bronchial carcinoma are reviewed with special regard to cigarette smoking. A diagnosis of primary chronic bronchitis can be misleading, especially in smokers, in whom the disease can mimic symptoms of lung cancer. The successful antibiotic treatment of pneumonia does not rule out lung carcinoma as the primary cause, because antibiotics can temporarily alleviate the bronchial obstruction. Endoscopy is of great significance in the differential diagnosis of bronchial carcinoma and pneumonia. (25 refs)

- 79-4251 **Cancer Risk of Chemists.** (Dut) Beekmans, W. (No affiliation given). *Chemisch Weekblad Magazine*: 311-313, April; 1979.

Cohort studies conducted among chemists (scientists, engineers, technicians, and chemical workers) in the US, Sweden, and the Netherlands indicated that they had a significantly increased cancer mortality rate compared with nonchemists. Lymphoma and tumors of the bone marrow and pancreas were most frequent. (13 refs)

- 79-4252 **Scientific Basis for Interpretation of Delaney Clause.** (Eng) Zimbelman, R. G. (Upjohn Company, Kalamazoo, MI 49001). *J Anim Sci* 48(4): 986-992; 1979.

The involvement of the American Society of Animal Science in the assessment of hormones as carcinogens is reviewed, and the implications of residues in food-producing animals are discussed. Dose selection is important in animal tests, and the effects of hormonal or physiologic alterations on tumor incidence should be taken into account. Statistics should be used to interpret differences between groups. The concept that statistical extrapolation procedures are sound and in accord with either mathematical considerations or biological mechanisms can be challenged, especially in the case of substances that alter the incidence of cancer in an indirect fashion. The emphasis should be on the biological assessment of all data rather than mathematical manipulation of certain data, particularly when the biological data suggest certain modes of action. (no refs)

- 79-4253 **Hormones and Hormonomimetic Compounds in the Etiology of Cancer.** (Eng) Lingeman, C. H. (Div. Cancer Cause and Prevention, NCI, Bethesda, MD 20014). *Recent Results Cancer Res* (66): 1-48; 1979.

The carcinogenic effects of gonadal hormones and hormonomimetic compounds are discussed. The chemistry, biology, and pharmacology of estrogens, progesterones and other progestins, androgens, prolactin, adrenal cortical steroids, gonadotropic- and hypothalamic-releasing hormones, and hormone receptors are reviewed. The relationship of hormones to cancers of the following specific sites is also discussed: breast, ovary, endometrium, cervix, vagina, prostate, testes, pituitary, thyroid, and nongenital organs. In addition to endogenous hormones, humans and animals are also exposed to a variety of hormonomimetic compounds in foods, cosmetics, and drugs. More information is needed to determine the precise roles of hormones and hormonomimetic compounds in carcinogenic processes in humans and animals. In the meantime, exposure to all of these compounds should be reduced or eliminated whenever possible except for valid medical indications. (213 refs)

- 79-4254 **Cellular Basis of Prolactin Action: Involvement of Cyclic Nucleotides, Polyamines, Prostaglandins, Steroids, Thyroid Hormones, Na/K ATPases and Calcium: Relevance to Breast Cancer and the Menstrual Cycle.** (Eng) Horrobin, D. F. (Clinical Res. Inst. Montreal, 110 Pine Ave. West, Montreal H2W 1R7, Canada). *Med Hypotheses* 5(5): 599-620; 1979.

The cellular basis of the actions of prolactin and its relevance to breast cancer and the menstrual cycle are discussed. It appears that prolactin and prostaglandin E₁ (PGE₁) at low concentrations tend to raise cytoplasmic calcium levels by enhancing its release from intracellular stores. High concentrations have the opposite effect. This biphasic response may depend on the activation of two distinct types of receptors. At physiologic levels, cortisol appears to block the effects of prolactin and the synthesis of PGE₁ but not the effects of dihomogamma-linolenic acid (DGLA) or PGE₁; progesterone blocks the effects of prolactin, PGE₁, and DGLA; thyroid hormones may enhance the effects of prolactin but not PGE₁; and estradiol and testosterone have no apparent effects on prolactin or PGE₁. Second messengers of prolactin other than PGE₁ are all probably dependent on calcium, and many prolactin effects may involve both PGE₁ and another second messenger. It is suggested that the effects of prolactin on the growth and development of the mammary glands are dependent on the activation of polyamine and cyclic nucleotide/protein kinase related mechanisms. Prolactin given early in breast tumor growth may enhance the formation of PGE₁ from adequate stores of DGLA, thereby lowering cytoplasmic calcium stores and nullifying the growth-promoting effects of prolactin operating via other

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second messengers. However, later in tumor growth when DGLA stores are depleted, prolactin may exert its growth-promoting effects via the other second messengers inhibited by PGE₁. It is possible that a partial essential fatty acid deficiency might predispose to breast cancer development and that gamma-linolenic acid might have a growth inhibiting effect. (100 refs)

- 79-4255 Breast Cancer and Treatment with Neuroleptics (2 Letters to Editor).** (Eng) Thompson, W. D. (Dept. Epidemiology, Yale Univ. Sch. Medicine, New Haven, CT 06519); Weissman, M. M.; Overall, J. E. *Arch Gen Psychiatr* 36(5): 604-605; 1979.

Conclusions of a previous report on the possible association between breast cancer (BC) and treatment of schizophrenia with neuroleptic drugs are reviewed critically. The major problem was that the study sample size was inadequate to detect anything less than an extremely large relative risk. The conclusion that increased BC risk from neuroleptic therapy is not a clinically substantial problem when schizophrenia is the alternative is questioned. It is also pointed out that knowledge of the risk arising from treatment with neuroleptics would aid in assessing the overall level of screening appropriate for individual schizophrenic women. Another problem with the study, arising in part from the small sample size, concerns the infeasibility of stratifying the samples to control for potentially confounding variables in addition to age; eg, age at first intercourse and age at first child. It is suggested that comparison of cases and controls with a history of psychiatric treatment with respect to amount of exposure to neuroleptic drugs would be of interest. The original author replies that of 3,278 women who were treated for schizophrenia over a 15-yr period only 6 returned for diagnosis and/or treatment of BC. Patients who were previously treated for schizophrenia were not overly represented relative to other psychiatric disorders in the BC group. There was no intention that the data should be considered conclusive with regard to the scientific question of whether neuroleptic-induced elevation of prolactin levels can cause BC in humans. (7 refs)

- 79-4256 Mammary Neoplasia in Animals: Pathologic Aspects and the Effects of Contraceptive Steroids.** (Eng) Casey, H. W. (Dept. Veterinary Pathology, Armed Forces Inst. Pathology, Washington, DC 20306); Giles, R. C.; Kwapien, R. P. *Recent Results Cancer Res* (66): 129-160; 1979.

Some major aspects of spontaneous mammary tumors in mice, rats, cats, dogs, and nonhuman primates are reviewed with emphasis on the pathologic aspects of these neoplasms and the use of these species in testing contraceptive steroids for carcinogenic activity. Murine mammary

tumors are primarily virus induced and provide an excellent model for studying the genetic, immunologic, and hormonal interactions of virus-induced mammary tumors. Rats have been extensively used to test a variety of chemicals for carcinogenicity. Although mammary neoplasms in cats most closely resemble those in women, this model has not been extensively studied. Canine mammary neoplasms, which generally occur in older animals, have been exploited in testing contraceptive steroids for carcinogenicity. Dogs develop a significant number of proliferative mammary lesions after 2-4 yr of drug administration. Studies on the effects of contraceptive steroids on the mammary glands of nonhuman primates are incomplete. Early animal studies indicated that some contraceptive steroids have carcinogenic properties. Further epidemiologic studies among women should permit the evaluation of their carcinogenic potential without reliance on animal data. (78 refs)

- 79-4257 Hepatic Neoplasms Associated with Contraceptive and Anabolic Steroids.** (Eng) Ishak, K. G. (Dept. Hepatic Pathology, Armed Forces Inst. Pathology, Washington, DC 20306). *Recent Results Cancer Res* (66): 73-128; 1979.

The differences between hepatocellular adenoma and focal nodular hyperplasia are discussed as is the current state of knowledge regarding their association with oral contraceptives (OC). The occasional occurrence of nodular regenerative hyperplasia in patients on OC or anabolic steroids (AS) is also discussed in light of other cases which are not associated with steroids. The occurrence of malignant liver tumors in patients using OC or AS is reviewed. (155 refs)

- 79-4258 Nutrition and Drug Metabolism.** (Eng) Krishnaswamy, K. (Natl. Inst. Nutrition, Hyderabad 500007, India). *Indian J Med Res* 68(Suppl): 109-120; 1978.

Studies concerning the effect of malnutrition on drug metabolism are reviewed. A study in which drug/carcinogen metabolism was evaluated in humans is briefly summarized. In albino rats, vitamin A deficiency and undernutrition significantly impaired aminopyrine N-demethylase activity, but aniline hydroxylase activity was not altered. Benzo(a)pyrene hydroxylase activity was impaired in vitamin A-deficient rats. (20 refs)

- 79-4259 Hepatobiliary Complications of Estrogen Therapy.** (Fre) Darnis, F. (Clinique Hepato-gastro-enterologique, Hopital Saint-Antoine, 75012 Paris, France); Poupon, R. *Rev Prat* 29(21): 1783-1784; 1979.

Studies of the hepatobiliary complications of estrogen therapy in general and of oral contraceptives in particular are reviewed. Although there is no definite evidence that progestogens induce benign hepatic tumors, they promote the growth of existing ones (mainly through vascularization) and they contribute to their rupture. The increase in the incidence of benign hepatocellular tumors since 1975 parallels the increasing use of oral contraceptives. There is an abrupt increase in the incidence of benign hepatic tumors after 60 mo continuous use of oral contraceptives. In one series, 93% of all patients with benign hepatic tumors had used mestranol, and 52.5% of the tumor-free women had used ethinyl estradiol. Benign hepatic adenomas may undergo malignant transformation. (6 refs)

- 79-4260 Pathologic Effects of Oral Contraceptives.** (Eng) Hilliard, G. D. (Dept. Gynecologic and Breast Pathology, Armed Forces Inst. Pathology, Washington, DC 20306); Norris, H. J. *Recent Results Cancer Res* (66): 49-71; 1979.

The pathologic effects of oral contraceptives (OC) on different organ systems are reviewed. No evidence indicates that OC users are more likely to develop more cervical neoplasms, although dysplasias may progress more rapidly in such women. Endometrial hyperplasia and carcinoma have been found more commonly among women taking sequential-type OC's, but not among those taking combined-type OC's. OC's may affect leiomyomas in two ways: by producing nuclear atypism in some instances; and by producing hemorrhagic degeneration of abnormally cellular foci. An association between the use of OC's and pituitary adenomas has not been demonstrated. In patients with vaginal adenosis who receive OC's, microglandular hyperplasia may develop in areas of stenosis. Atypical epithelial proliferation and carcinomas occur in women taking OC's, but there is no evidence to indicate that the drugs cause these lesions. However, in rare instances, steroids have been shown to modify the histologic appearances of mammary cancers. The relationship of OC use to pathology of the fallopian tubes and ovary is also discussed, as is the relationship to thrombosis, thromboembolic disease, hypertension, and carbohydrate metabolism. A national registry should be formed to record and investigate the cases of women who die or have adverse reactions while taking OC's. (133 refs)

- 79-4261 Cancer and Other Lesions in Mice Receiving Estrogens.** (Eng) Dunn, T. B. (Registry Experimental Cancers, NCI, NIH, Bethesda, MD 20014). *Recent Results Cancer Res* (66): 175-192; 1979.

The occurrence of cancer and other lesions in mice receiving estrogens is reviewed. Newborn male mice given

diethylstilbestrol (DES) develop epididymal cysts but no other lesions. Similarly treated females, especially those of the BALB/c strain, often developed epidermoid carcinomas of the cervix and vaginal wall, with persistent vaginal cornification and changes in the endometrium, adrenals, ovaries, and other organs. These mice appeared to be under a continuous estrogen stimulation. Enovid (mestranol and norethynodrel) produced similar, but less severe effects when injected. It also effectively induced sterility when given in a liquid diet, but not when incorporated in the solid feed. The sterile mice also showed signs of continuous estrogen stimulation, and when they were older they developed nonmetastasizing small cancers of the uterine cervix. When Enovid was incorporated into food pellets, only BALB/c mice developed cervical cancer and only C57BL mice developed pituitary tumors. No increase in mammary or ovarian tumors occurred. Although the carcinogenic effects of estrogen have been shown to be unpredictable, these compounds were always carcinogenic when adequately tested. (12 refs)

- 79-4262 Abnormalities of the Genital Tract Following Stilbestrol Exposure in Utero.** (Eng) Kurman, R. J. (Dept. Pathology, Georgetown Univ. Sch. Medicine, 3800 Reservoir Road, Washington, DC 20007). *Recent Results Cancer Res* (66): 161-174; 1979.

The effects of in utero exposure to diethylstilbestrol (DES) on the genital tract and the embryology of the female genital tract are reviewed. Evidence indicates that DES inhibits the replacement of the müllerian epithelium, thereby resulting in the aberrant location of glandular epithelium in the vagina and cervix. DES was also carcinogenic in female mice exposed in utero. The occurrence of clear-cell adenocarcinoma (CCA) in human females born after 1940 was studied. Approx two-thirds of the patients with this tumor had been exposed to DES, diethylstilbestrol, or hexestrol in utero, and an additional 10% had received an unspecified medication for bleeding in pregnancy or previous miscarriage. A progestational agent was used in conjunction with DES in 7% of the patients with tumors. There was no instance of CCA associated with a naturally occurring estrogen. In all instances in which CCA was associated with in utero DES exposure, DES treatment was initiated during the first half of pregnancy. DES exposure has never been associated with neoplasms of the endometrium or ovary. Almost one quarter of the women with CCA of the vagina and cervix have died or developed recurrent tumor, but this figure must still be regarded as tentative in view of the relatively short follow-up period in many of the cases. CCA appears to have a greater propensity for metastasis to the lungs and supraclavicular lymph nodes than does squamous carcinoma of the vagina and cervix. Almost all women exposed to DES developed vaginal adenosis, cervical ectropion, and/or transverse vaginal and cervical ridges. All females with a history of DES exposure should be examined; this does not appear necessary for exposed males. (51 refs)

79-4263 Cancer Risk in DES-exposed Offspring. (Eng) Powell, J. L. (Foundation Gynecologic-Oncology, Inc., Ste. 100, 5669 Peachtree-Dunwoody Rd., NE, Atlanta, GA 30342). *J Med Assoc Ga* 68(5): 403-406; 1979.

Cases of clear-cell adenocarcinoma (CCA) in women born during or after 1940 are reviewed on the basis of information gathered in the Registry for Research on Hormonal Transplacental Carcinogenesis. Of 341 cases analyzed by 1977, approx two-thirds were associated with intrauterine exposure to diethylstilbestrol (DES) or a chemically related estrogen. The risk of carcinomas developing in DES-exposed females through the age of 24 yr was estimated to be 0.14-1.4/1,000. The peak frequency of CCA is at 19.5 yr, and it has been suggested that the hormonal events accompanying puberty may play some role in carcinogenesis. Nonneoplastic changes are more common than CCA. These include cervical ectropion, transverse cervical and vaginal ridges, and adenosis. Three invasive squamous cell carcinomas have been identified in DES-exposed progeny. Malignancies have not been found in DES-exposed males, although epididymal cysts, hypoplastic testes, cryptorchidism, and semen abnormalities occur more frequently in these subjects. A woman exposed to DES in utero should have an examination once she starts to menstruate or at about age 14 if menstruation has not begun by that time. Colposcopy and iodine staining, although not essential for such an examination, permit accurate assessment of the extent of epithelial change. After a normal initial examination, annual pelvic examinations with cervical and vaginal cytology are recommended. (14 refs)

79-4264 Perinatal Period and Pregnancy: Intervals of High Risk for Chemical Carcinogens. (Eng) Rice, J. M. (Lab. Experimental Pathology, NCI, Bethesda, MD 20014). *Environ Health Perspect* 29: 23-27; 1979.

Groups of individuals who may be at high risk for chemical carcinogens are reviewed. If human prenatal susceptibility is like that of the lower primate, the human fetus may be most susceptible to at least some transplacental carcinogens before the mother may know she is pregnant. Most carcinogens will probably affect the offspring to some extent when given during pregnancy. The evidence from both rat and primate studies, although limited, suggests that the pregnant female is at higher risk to at least certain carcinogens than nonpregnant females or males of comparable age. It is reasonable to infer a hormonal component in the pathogenesis of the tumors caused by these agents. The available evidence is also sufficiently convincing to warrant consideration of early postnatal life as a period of generally enhanced susceptibility to chemical carcinogens. (17 refs)

79-4265 US Academy Denies Threshold for Radiation Damage. (Eng) Dickson, D. (No affiliation given). *Nature* 279(5709): 90-91; 1979.

A committee of the US National Academy of Sciences confirmed its support for the hypothesis that the effect of ionizing radiation on the human body is directly proportional to dose and that there is no threshold below which such radiation can be ignored. This conclusion is contested by some committee members on the grounds that too little theoretical information exists to serve as a reliable guide for extrapolation. (no refs)

79-4266 The Scientific Basis for the ICRP's Use of Linear Extrapolation to Obtain Best Estimates of the Risk of Radiation-induced Cancer at Low Doses (Meeting Abstract). (Eng) Brown, J. M. (MRC Unit Clinical Oncology, Radiotherapeutics, Hills Road, Cambridge, England). *Br J Radiol* 52(617): 427; 1979 (1 ref)

79-4267 The Impact of Nuclear Technology on the Natural Environment and Human Life. (Eng) Latarjet, R. (Fondation Curie, Institut du Radium, Section de Biologie, 26 rue d'Ulm, F-75005 Paris, France). *Prog Biochem Pharmacol* 14: 28-35; 1978.

Studies concerning the impact of diffuse and permanent pollution by small amounts of ionizing radiation on the environment and human life are reviewed. Radiation has been shown to be lethal to cells and to be mutagenic and carcinogenic. The author feels that the argument that radiation carcinogenesis in humans is a nonthreshold phenomenon is invalid. A realistic max dose in widespread radiation pollution would be about 1/20 of the dose that would double the frequency of natural mutations. (2 refs)

79-4268 Skin and Eye Irradiations--Examples of Some Problems of Implementing International Recommendations in Radiological Protection (Meeting Abstract). (Eng) Charles, M. W. (CEGB Berkeley Nuclear Labs., Berkeley, Gloucestershire GL13 9PB, England). *Br J Radiol* 52(617): 427; 1979 (5 refs)

79-4269 Carcinogenic Risk of Mammography. (Rus) Osipova, V. N. (I. M. Sechenov First Medical Inst., Moscow, USSR). *Med Radiol (Mosk)* 24(5): 74-78; 1979.

Controversial data on the carcinogenic risk of mammography are reviewed. Mammography increases the detectability of breast cancer to 13.2/1,000, compared with 0.38-2.0/1,000 by palpation. X-irradiation of Sprague-Dawley rats with a single dose of 10, 100, 200, 400, or 600 R induced mammary gland tumors 12 mo later in 2.2%, 9.4%, 13%, 9.7%, and 30% of the rats, respectively.

Although the literature lacks valid clinical confirmation of breast cancer development following mammography, the carcinogenic risk of this technique cannot be excluded. (19 refs)

- 79-4270 Photodynamic Therapy for Herpes Simplex: A Critical Review.** (Eng) Bockstahler, L. E. (Bureau Radiological Health, Food and Drug Admin., Rockville, MD 20857); Coohill, T. P.; Hellman, K. B.; Lytle, C. D.; Roberts, J. E. *Pharmacol Ther [A]* 4(2): 473-499; 1979.

The use of photodynamic therapy for herpes simplex virus (HSV) infection is reviewed. Herpesviruses are associated with tumors in many animal species, and they may be associated with human cervical carcinoma (HSV-2) and carcinoma of the lip (HSV-1). The association between HSV-2 and human carcinoma is based on viral and seroepidemiological studies. There is no single, generally effective treatment for herpetic lesions of the skin and mucous membranes or a method of preventing recurrences. Fundamental principles of photodynamic action that provide a basis for antiviral photodynamic therapy and for potential long-term side effects are reviewed. The treatment consists of applying a photosensitizing dye to HSV lesions and then exposing them to visible light. Some human clinical trials show a reduction of HSV infectivity, whereas others throw doubt on the efficacy of the procedure. Possible side effects from the photodynamic treatment of virus include unmasking of the oncogenic potential of HSV, induction of endogenous C-type RNA tumor viruses, and mutation of HSV. Possible side effects from the photodynamic treatment of cells include cellular mutation, enhanced cellular susceptibility to transformation by tumor viruses, and the induction of latent tumor virus. (245 refs)

- 79-4271 Radiation Hazards in Connection with the Use of ^{125}I in In Vitro Tests.** (Cze) Husak, V. (Klinika nuklearní medicíny, Fakultní nemocnice s poliklinikou, I.P. Pavlova 6, 775 20 Olomouc, Czechoslovakia); Budikova, M. *Cas Lek Cesk* 118(4): 117-119; 1979.

The hazards of contamination of laboratory personnel with ^{125}I used in in vitro tests, especially in radioimmunoassays, are discussed. An ^{125}I concentration corresponding to an equivalent of 20 roentgen-equivalents-man was found in the thyroid gland of one laboratory technician. (20 refs)

- 79-4272 Aging, Carcinogenesis and Radiation Biology.** (Eng) Smith, K. C. (Dept. Radiology, Stanford Univ. Sch. Medicine, Stanford, CA 94305). *Prog Biochem Pharmacol* 14: 70-75; 1978.

The relationships between aging, carcinogenesis, and radiation biology are reviewed. Mutagenesis appears to be due primarily to errors in the repair of damaged DNA. Presumably, chemical carcinogenesis is also the result of the error-prone repair of chemically damaged DNA. This latter concept is supported by the fact that nearly all chemical carcinogens are mutagens and by observations that patients with heritable syndromes that greatly increase their chances of cancer have deficiencies in DNA repair. The various theories of aging can be combined to form a unified theory based on alterations in the structure and function of DNA. What the cell does or does not do to this DNA damage determines its biological effect. Most current knowledge concerning the chemical nature of the damage that can be produced in DNA, its biological consequences, and the mechanisms by which it can be repaired comes from the field of radiation biology. (22 refs)

- 79-4273 Radioruthenium in the Environment.** (Jpn) Iwashima, K. (Inst. Public Health, 4-6-1, Shioganedai, Minato-ku, Tokyo 180, Japan); Morita, S. *Radioisotopes* 28(3): 184-193; 1979.

Environmental contamination by ^{103}Ru and ^{106}Ru due to nuclear tests and nuclear waste from reprocessing plants can result in the internal bombardment of human tissues by Ru through food and breathing air containing particulate Ru. Nuclear tests contaminate directly via breathing and treatment plants contaminate indirectly via their discharge into water and the subsequent consumption of food nurtured in such water. In one test, the ^{103}Ru dose absorbed by workers harvesting porphyra from water contaminated by the Windscale treatment plant was calculated by combining the max allowed visceral absorption dose [roentgen-equivalent-man/yr (rem/yr)], the max daily absorbed dose (Ci/day), and the max radiation found in the water (Ci/cm³ 73 liters). The absorption rate was $1.8 \times 10^{-3} \mu\text{Ci/cm}^3/\text{day}$. In 1975, the exposure of the general population to ^{106}Ru around reprocessing plants was estimated to be 16-18.7 millirem/yr for the digestive organs and 0.3-0.35 millirem/yr whole body by combining the ^{106}Ru concentration in foods consumed daily and taken from contaminated seawater (5 km from source 3.6×10^{-15} Ci/ml, 1 km 1.9×10^{-14} Ci/ml): shellfish (10 g/day/person), sea vegetation (30-35 g/day), young sardines (20-30 g/day), mature fish (180-200 g/day), and shrimp (20 g/day). The calculations showed that rate of radiation absorption in the digestive tract and whole body was due to high ^{106}Ru concentrations in shellfish and sea vegetation, but field data showed mature fish to be the primary contributors to absorbed radiation levels. (86 refs)

- 79-4274 Biological Fate of Inhaled Transuranic Elements.** (Fre) Metivier, H. (Laboratoire de Toxicologie expérimentale, Commissariat à l'Énergie

atomique, B.P. no. 561, 92542 Montrouge Cedex, France). *Radioprotection* 14(1): 19-39; 1979.

Studies of the biological fate of inhaled transuranic elements are reviewed. Lung clearance and translocation are greatly affected by the chemistry of these elements. However, studies show that the kinetics of some chemical reactions, especially those involving plutonium, is a function of element concentration. Therefore, caution should be exercised in attempts to extrapolate to biological concentrations. Lung clearance increases with solubility, and it may slow down as the inhaled dose of particles in the micron range increases. On the other hand, the residual fraction is eliminated from the lungs very slowly: the half-life ranges from 500 days for $^{238}\text{PuO}_2$ and $^{239}\text{PuO}_2$ to 250 days for $^{238}\text{Pu}(\text{NO}_3)_4$, $^{239}\text{Pu}(\text{NO}_3)_4$, $^{241}\text{AmO}_2$, $^{241}\text{Am}(\text{NO}_3)_3$, and $^{244}\text{Cm}(\text{NO}_3)_3$. Most of the radioactivity is eliminated in 8-120 days. Translocation of ^{239}Pu into the thoracic lymph nodes, liver, and skeleton was found in dogs 56 days after the inhalation of plutonium oxides. (77 refs)

- 79-4275 Theoretical Mechanisms for Synthesis of Carcinogen-induced Embryonic Proteins: IV. The Viruses.** (Eng) Hancock, R. L. (High River Inst. Theoretical Cancer Study, High River, Alberta, Canada); McDuffie, N. G.; Sinclair, D. B. *Med Hypotheses* 5(3): 383-401; 1979.

Mechanisms by which oncogenic viruses induce cell alterations that permit the expression of embryonic genes are suggested. It is proposed that deheterochromatization occurs when a viral DNA becomes inserted, directly or via RNA-directed DNA polymerase, at particular euchromatin-heterochromatin junctions of quasi-differentiated stem cells. The deheterochromatization derepresses the genes for acid protein phosphokinases, leading to phosphorylation reactions that also cause derepressions by altering acid proteins that act as specific repressors (eg, of fetal-type histone methylase). This leads in turn to additional chromatin alterations and the activation of repressed fetal genes. Thus, viruses induce the embryonic phenotype through processes similar to those effected by chemicals such as the hepatocarcinogen ethionine. (66 refs)

- 79-4276 Cell-surface Antigens Induced by RNA Tumor Viruses.** (Eng) Kurth, R. (Friedrich Miescher Lab., Max Planck Inst., Spemannstrasse 37-39, 74 Tübingen, W. Germany); Fenyo, E. M.; Klein, E.; Essex, M. *Nature* 279(5710): 197-201; 1979.

The properties of virus-induced cell-surface antigens (CSA's) of the avian, murine, and feline retrovirus systems were compared. Host species respond with immunity against the cell surface of RNA tumor virus-infected cells.

Although antiviral immunity may abrogate infection, resistance against tumors is primarily due to the responses against CSA's. Recognition of antigens is influenced by host genetics and may show wide variations. Gross cell surface antigen (GCSA) cannot be distinguished from viral (*gag*) precursor proteins. Part of the Moloney cell surface antigen (MCSA) and feline oncornavirus-associated cell membrane antigen (FOCMA) reactive molecules also contain the antigenic determinants of viral 5' terminal *gag* protein(s). Thus, these CSA's all correspond to precursors of the viral *gag* gene. The relationship of CSA to transformation seems to be different in the various systems. FOCMA is specific for transformation by both feline leukemia virus and feline sarcoma virus. Tumor-specific cell surface antigen (TSSA) is specific for transformation of fibroblasts by avian sarcoma virus, but is probably not expressed on avian lymphatic leukemia virus-transformed lymphoma cells. MCSA is similar to GCSA in that both are expressed on preleukemic, nonmalignant thymus cells. It is not yet clear whether the expression of functional TSSA and FOCMA is sufficient to establish the malignant character of correspondingly infected cells. (110 refs)

- 79-4277 Lymphoma Development in Mice and Humans: Diversity of Initiation Is Followed by Convergent Cytogenetic Evolution.** (Eng) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S 104 01 Stockholm 60, Sweden). *Proc Natl Acad Sci USA* 76(5): 2442-2446; 1979.

The role of cytogenetic changes in the development of human B-cell lymphoma and murine T-cell leukemia is reviewed. The available data suggest that transformation in vitro is not synonymous with tumorigenicity in vivo. If transformation in vitro reflects a "built-in" ability to grow in the absence of exogenous stimulation, tumorigenicity in vivo must imply additional resistance to negative feedback regulations of the host. Determinants on human chromosomes 9, 14, and 22 and on murine chromosome 15 appear to be of crucial importance for the responsiveness of different cell types to growth control. Host cell controls can apparently modify the expression of transformation in vitro, and they can reverse tumorigenic to nontumorigenic phenotypes in vivo. It is suggested that like chemical or physical carcinogens, viruses essentially play the role of initiators in tumor progression, their major effect being the establishment of long-lived preneoplastic cells. Specific genetic changes are then responsible for the transition of preneoplastic to frankly malignant cells. The cytogenetic changes act by shifting the balance between genes that favor progressive growth in vivo and genes that counteract it. (82 refs)

- 79-4278 New Oncogenic Human Papovaviruses.** (Eng) Mantyjarvi, R. A. (Dept. Clinical

Microbiology, Univ. Kuopio, P.O.B. 138, SF-70101 Kuopio 10, Finland). *Med Biol* 57(1): 29-35; 1979.

Characteristics of two new oncogenic human papovaviruses (BKV and JCV) are reviewed. BKV replicates in several types of human fetal cells and Vero cells, but only human fetal glial cells support the growth of JCV. Both viruses agglutinate human and guinea-pig RBC. There are five or six polypeptides in the BKV virion. Antigenically, BKV and JCV are distinct from each other and from simian virus 40, but they share nucleotide sequences; and a minor cross-reactivity associated with a virion surface antigen has been demonstrated. The nucleic acid of BKV and JCV is a double-stranded circular DNA. Sc inoculation of BKV into newborn hamsters induced fibrosarcomas, and intracranial inoculation induced papillary ependymomas and choroid plexus papillomas. JCV also induced brain tumors in newborn hamsters and two owl monkeys. Primary cultures of hamster, rat, mouse, rhesus monkey, African green monkey, and rabbit cells, and a continuous line of hamster cells have been transformed in vitro by BKV or viral DNA. Even defective BKV may induce transformation. Tumor-specific T and transplantation antigens have been found in papovavirus-transformed cells. Although a high percentage of the human population has antibodies against BKV, no correlation has been shown between the prevalence or titers of antibodies and the presence of tumors. If human papovaviruses cause tumors in man, it is a rare event. However, BKV infection is activated in patients receiving immunosuppressive therapy and it is possible that this activation could lead to tumor induction. (99 refs)

- 79-4279 **Role of Macrophages in Natural Resistance to Virus Infections.** (Eng) Mogensen, S. C. (Inst. Medical Microbiology, Univ. Aarhus, Aarhus, Denmark). *Microbiol Rev* 43(1): 1-26; 1979.

The role of macrophages (MP's) in natural resistance to primary virus infections is reviewed. On the basis of morphology, function, and kinetics of development, all highly phagocytic cells and their precursors can be grouped in one cell lineage called the mononuclear phagocyte system. Virus-MP interactions have been studied in vitro either directly or, more often, indirectly using techniques such as infectious center assays, cytopathology, immunofluorescence, electron microscopy, and tracer techniques. In vivo studies comprise those in which (1) MP restriction of virus growth has been shown to be important for the difference in animal resistance to closely related virus strains or virus types; (2) variation in innate resistance to a virus infection displayed by members of the same animal species (genetically determined resistance) has been correlated with the ability of the virus to replicate in MP's from the animals; (3) an age-related increase in resistance to a virus infection has been found to be caused by a maturation of MP's during the first few weeks of life; and

(4) restricted virus replication in nonspecifically activated MP's accounts for augmented resistance to virus infections. The basic nature of the capacity of MP's to restrict virus replication is not known. Evidence is accumulating, however, of the key role of MP's as a first line of defense against the establishment of viral infection during the first few days of infection or in target organs of crucial importance for the outcome of infections. (168 refs)

- 79-4280 **Perinatal Viral Infections and the Risk of Certain Cancers.** (Eng) Munoz, N. (International Agency Res. Center, 150 Cours Albert Thomas, F-69008 Lyon, France). *Prog Biochem Pharmacol* 14: 104-108; 1978.

Evidence that perinatal viral infections may play a role in the development of certain tumors is reviewed. Several epidemiological studies have suggested an association between prenatal influenza and childhood cancer, especially leukemia, although other studies have shown no such association. There is also evidence that influenza virus may be teratogenic. Varicella virus may also be teratogenic, and chicken pox infection during pregnancy has been associated with an increased cancer risk in the offspring. An increased risk of cancer, particularly neural tumors, has been reported for the offspring of women who were immunized with killed polio vaccine contaminated with simian virus 40. There are also data suggesting that intrauterine or perinatal infection with hepatitis B virus (HBV) might be one of the risk determinants for hepatocellular carcinoma (HCC) in adults. The hepatitis B surface antigen carrier rate is much higher among HCC patients than among controls and in high- vs low-risk areas. A very high proportion of carriers (80%-90%) has also been found among mothers of HCC patients from Senegal. The data suggest that perinatal infections with HBV develop more readily into chronic persistent hepatitis and that this infection is a high risk condition for HCC. (22 refs)

- 79-4281 **Interactions and DNA Transfer Between *Agrobacterium tumefaciens*, the Ti-Plasmid and the Plant Host.** (Eng) Schell, J. (Lab. Genetics, State Univ., Ledeganckstraat 35, B-9000 Ghent, Belgium); Van Montagu, M.; De Beuckeleer, M.; De Block, M.; Depicker, A.; De Wilde, M.; Engler, G.; Genetello, C.; Hernalsteens, J. P.; Holsters, M.; Seurinck, J.; Silva, B.; Van Vliet, F.; Villarroel, R. *Proc R Soc Lond [Biol]* 204(1155): 251-266; 1979.

Studies concerning the oncogenicity of *Agrobacterium tumefaciens*, a gram-negative bacterium with the ability to induce neoplastic transformation in dicotyledonous plants, are reviewed. The large extrachromosomal DNA plasmid, the Ti plasmid, is responsible for the oncogenicity. A segment of the Ti plasmid, which contains information deter-

mining tumor growth pattern and opine synthesis, is transferred and stably maintained and expressed in transformed plant cells. (39 refs)

- 79-4282 Type C Retrovirus Activation and Possible Functions in the Normal and Tumor-bearing Host.** (Eng) Hellman, A. (NCI, Frederick Cancer Res. Center, Building 538, Room 200D, Frederick, MD 21701); Weislow, O. S.; Twardzik, D. R.; Fowler, A. K. *Cancer Res* 39(7, part 2): 2902-2907; 1979.

Current information on the role of C-type viruses in processes such as cellular differentiation, immune recognition, embryogenesis, and tumor induction is reviewed. To participate in normal physiological processes, viral products should be inducible at the appropriate time by physiological means. Multipotent factors, such as hormones, are likely candidates for such a function. One study showed that the activation of viral information by estrogens is under genetic control. More recently, investigators have also used steroids to increase viral production in vitro. Examination of virus expression in normal mice during the estrous cycle and gestation showed that the level of viral protein (p30) was highly correlated with the rhythm of endogenous estrogen levels during both periods. Immunological activation of C-type retroviruses has also been demonstrated. Retroviral activation occurs in vivo during graft-vs-host disease and allogeneic graft rejection and in vitro during mixed lymphocyte reactions and mitogen-induced lymphocyte blastogenesis. The significance of viral activation by immune reactions is unclear. C-type retroviruses and the immune responses they generate can have a primary and often profound effect on normal and tumor-bearing host-immune recognition processes that, in turn, may secondarily influence other organ systems and host functions. Several approaches applied to the study of retrovirus interaction with the immune system are described. It is speculated that activated, viral-induced modifications of the cell surface might lead to necessary changes in the recognition patterns required for cellular differentiation, cell-to-cell interactions, and self-nonsel distinctions. (57 refs)

- 79-4283 Review of Results of the USSR "Revertase" Project.** (Rus) Kiselev, L. L. (Inst. Molecular Biology, Moscow, USSR); Engelhardt, V. A. *Mol Biol (Mosk)* 13(2): 245-265; 1979.

The results of a USSR cooperative research program termed "Revertase" that was carried out during 1974-1978 are reviewed. The scientific problems studied within the framework of the project included the reverse transcription of DNA on RNA templates, the use of complementary DNA to analyze the genome of viruses and eukaryotes, enzymatic synthesis of structural genes, enzymology of

reverse transcription, and the role of reverse transcriptase in the virus-induced transformation of cells. (109 refs)

- 79-4284 Immunity in Malignant Disease.** (Eng) Woodruff, M. (MRC Clinical and Population Cytogenetics Unit, Western General Hosp., Crewe Road, Edinburgh EH4 2XU, Scotland). *Br J Surg* 66(5): 297-301; 1979.

The role of immunology in malignant disease is discussed. It is generally accepted that the primary event in the development of a tumor is a heritable change. Carcinogenic chemicals, ionizing radiation, and UV radiation play an etiologic role in human cancer; viruses may also play a part although their role is unclear. There is evidence that many tumors are of multifocal origin, although it has become increasingly apparent that many are, to a large extent, monoclonal. Dissemination leading to metastasis can occur quite early, often before patients present for treatment. Although it was once accepted that malignant tumors are autonomous, some tumors have been shown to be hormone dependent and there is evidence that homeostatic mechanisms exist for controlling carcinogenesis and tumor growth. A tumor is a complex ecosystem consisting of living and dead neoplastic cells, nonneoplastic cells such as lymphocytes and macrophages, and vascular connective tissue. The tumor-associated transplantation antigens of many tumors form the basis of the immunologic surveillance hypothesis. Experimental data suggests that macrophages and unidentified natural killer cells play an important role in surveillance. Both specific and nonspecific immunologic procedures for augmenting natural homeostatic mechanisms are currently being investigated. Such forms of treatment are needed to deal with residual disease following the surgical excision of primary tumors. (16 refs)

- 79-4285 Subversion of the Immune System by Tumors as a Mechanism of Their Escape from Immune Rejection.** (Eng) Plescia, O. J. (Waksman Inst. Microbiology, Rutgers, The State Univ. New Jersey, New Brunswick, NJ 08903); Grinwich, K.; Sheridan, J.; Plescia, A. M. *Prog Biochem Pharmacol* 14: 123-138; 1978.

This review concerns subversion of the immune system by tumors as a possible reason why an autochthonous or syngeneic tumor fails to elicit an effective immune response. There is evidence that tumors can act aggressively against certain types of immune cells, subverting them and, thereby, escaping from immunological rejection. A generalized depression of the antibody response has been observed in syngeneic tumor-bearing mice. T cells appear to be the targets of immunosuppressive syngeneic tumor cells in such cases. Phagocytic cells are not depressed by the action of syngeneic tumors. The prostaglandins, particular-

ly prostaglandin E, appear to have some role as mediators of immunosuppression by tumor cells. There is also some meager and indirect evidence suggesting a role for cyclic AMP as a messenger of prostaglandin activity. (21 refs)

- 79-4286 Effect of Age on Immune Function in Terms of Chemically Induced Cancers.** (Eng) Bennett, M. (Dept. Pathology, Boston Univ. Sch. Medicine, Boston, MA 02118). *Environ Health Perspect* 29: 17-22; 1979.

Data on the effect of age on immune function are reviewed along with how this effect may relate to age-associated susceptibility to chemically induced cancers. Neonatal, fetal, and very old animals are particularly sensitive to chemical carcinogenesis. This increased sensitivity could be due to increased susceptibility of "target" organs or cells, peculiar hormonal levels at these age groups, relatively deficient immune functions, or combinations of these and/or other factors. During the late fetal and first 3 wk of neonatal life, the immune system is maturing rapidly and is relatively incompetent, and its diverse components are developing at different rates. For example, T alloreactive cells capable of proliferating in mixed lymphocyte reactions (T helper cells) develop by 7 days of age, but precursors of T killer cells are not competent until approx 14 days of age. B cells capable of generating antibody responses are present in fetal liver, but they are extremely sensitive to tolerance induction until 10-14 days of age, when IgD cell-surface receptors are detectable. Marrow-dependent (M) cells responsible for the regulation of suppressor cells and for natural cytotoxicity to transformed tumor cells do not mature until 3 wk of age. In very old animals, the thymus is atrophic and cell-mediated immunity is moderately suppressed. Natural cytotoxicity against tumor cells is less than normal, but antibody formation (B-cell function) is adequate. Gonadotrophic hormones of the pituitary or placenta are high during pregnancy, the early neonatal period, after menopause, and in a large fraction of men >60 yr of age. These and other hormones are immunosuppressive and could theoretically facilitate carcinogenesis. The particular immune cell type, if any, responsible for resistance to chemically induced tumors has not been determined. One can only state that susceptibility to chemical carcinogenesis is associated with a relative dysfunction of the immune system and that age is an important factor. (74 refs)

- 79-4287 Chronic Lymphocytic Leukemia (CLL) Terminating in Multiple Myeloma: Report of Two Cases (2 Letters to Editor).** (Eng) Crowley, J. P. (Div. Clinical Hematology, Rhode Island Hosp., Providence, RI); Kough, R. H.; Makary, A. Z. *Blood* 53(3): 523; 1979.

The difficulties of establishing the common origin of

chronic lymphocytic leukemia and multiple myeloma by surface markers alone are pointed out. The only definitive way to establish the similarity of origin of the two diseases would be to demonstrate an idiotypic chromosomal abnormality in both the lymphocytes and plasma cells of a patient with the coexisting disorders. (4 refs)

- 79-4288 Cell-mediated Immunity, Epstein-Barr Virus and Nasopharyngeal Carcinoma.** (Eng) Levine, P. H. (NCI, NIH, Bethesda, MD 20014); Lamelin, J. P.; Stevens, D. A. *IARC Sci Publ* 20: 483-494; 1978.

Cell-mediated immunity (CMI) to Epstein-Barr virus (EBV) and its relation to nasopharyngeal carcinoma (NPC) are reviewed. Clinical observations and experimental models suggest that CMI plays a central role in host defenses against human herpesviruses. When the primary EBV infection is manifested as infectious mononucleosis (IM), there is an increase in T cells following an increase in B cells. Lymphocytes from IM patients showed lymphoproliferation and increased cytotoxicity in response to EBV-infected cells. EBV capsid antigen- and S antigen-induced blastogenesis have been reported in both seropositive and seronegative donors; it appears that these indices of CMI develop slowly in IM patients. Depressed blastogenesis of peripheral blood lymphocytes in response to phytohemagglutinin and concanavalin A and negative Mantoux tests have been reported in NPC patients. Skin tests on NPC patients showed 55% reactivity to an NPC lymphoblastoid cell line and $\leq 10\%$ reactivity to other antigens. The NPC patients were significantly more reactive to the NPC line than patients with leukemia, lymphoma, or other solid tumors, although approx 20% of non-NPC patients also showed positive reactions. Increased production of lymphocyte migration inhibitory factor in response to Raji cells and pooled biopsy extracts was significantly more common among NPC patients than among those with other tumors. CMI responses to virus- and tumor-associated antigens may have implications for the etiology, diagnosis, and treatment of NPC. (25 refs)

- 79-4289 Neurofibromatosis and Malignancy (2 Letters to Editor).** (Eng) Hecht, F. (Genetics Center, Southwest Biomedical Res. Inst., 123 E. University Drive, Tempe, AZ 85281); McCaw, B. K.; Lanzkowsky, P.; Shanske, A.; Shende, A.; Karayalcin, G. *J Pediatr* 94(6): 1010-1011; 1979.

Neurofibromatosis is a prototype of an autosomal dominant phenotype associated with a growing list of malignant tumors. Understanding neurofibromatosis will aid in determining the process that starts with a mutant gene and results in a malignant cell. The occurrence of rhabdomyosarcoma in 2 of a series of 10 patients with

neurofibromatosis highlights the fact that neurofibromatosis carries an increased risk of this neoplasm. (5 refs)

79-4290 Genetic Determination of Retinoblastoma (Pattern of Inheritance). (Rus) Artemov, A. V. (V. P. Filatov Res. Inst. Eye Diseases and Tissue Therapy, Odessa, USSR). *Vestn Akad Med Nauk SSSR* (5): 87-90; 1979.

Various hypotheses concerning the inheritance of retinoblastoma are reviewed. It is suggested that all individuals who are predisposed to the development of retinoblastoma have a specific oncogene incorporated into their cellular DNA. The activation of this oncogene cannot be explained in terms of epigenetic regulation. The ability of the oncogene to be destroyed is probably determined by the conformation of the DNA molecule. (17 refs)

79-4291 Recent Developments in Understanding the Pathogenesis of Aplastic Anemia. (Eng) Fitch, J. H. (Div. Hematology-Oncology, Dept. Medicine, Univ. California Sch. Medicine, Los Angeles, CA 90024); Cline, M. J. *Am J Hematol* 5(4): 365-372; 1978.

Bone marrow failure in aplastic anemia (AA) could result from abnormalities of hematopoietic stem cells, abnormal control of hematopoiesis, or abnormalities of the hematopoietic environment. A review of the results of 11 bone marrow transplants between identical twins for treatment of AA indicates that the provision of normal hematopoietic stem cells corrects aplasia in most cases and suggests that the defect in AA resides in the stem cells. However, it is possible that the cells critical to hematopoietic recovery are regulatory or "helper" cells instead. Similarly, whereas the effectiveness of allogeneic bone marrow transplantation in AA has been cited as evidence that the disorder is the result of a stem-cell abnormality, the data do not exclude the possibility that transplantation provides necessary stromal or helper cells. Evidence from studies applying in vitro bone marrow culture techniques to patients with AA suggests that AA does not result from abnormalities in the humoral control of hematopoiesis or in critical cellular interactions. A series of reports in the literature has suggested that cellular immune mechanisms may mediate AA. Studies of radiation- or drug-induced marrow failure in mice have suggested that marrow failure may be the result of inappropriate differentiation during periods of stem cell depletion. Studies of congenitally anemic mice suggest that the defect involves a regulatory cell as well as a stem cell. It seems likely that AA will prove to be many diseases that share common clinical and morphologic features. Cell fractionation techniques should enable the separation of AA resulting from abnormalities in hematopoietic stem cells from those caused by abnormal regulation and those resulting from immune suppression of hematopoiesis. (45 refs)

79-4292 Dental Diseases and Oral Malignant Disease. (Eng) Zehm, S. J. (Hals-Nasen-Ohren Clinic, Allgemeines Krankenhaus Heidelberg, Tangstedter Landstrasse 400, 2 Hamburg 62, W. Germany). *Otolaryngol Clin North Am* 12(1): 21-27; 1979.

Dental diseases associated with the development of malignant oral tumors are reviewed, with emphasis on processes that cause clinical alterations, hyperplasia, ulcerations, or disfiguration of the gingiva and adjacent mucosa. (38 refs)

79-4293 Histogenetic, Functional, and Morphological Properties of Medullary Carcinoma of the Thyroid Gland. (Ser) Hadzic, B. (Dept. Pathology, Faculty Medicine, Novi Sad, Yugoslavia); Budakov, P.; Stajnic, S. *Srp Arh Celok Lek* 106(4): 415-420; 1978.

Recent studies of the histogenetic, functional and morphological aspects of medullary carcinoma of the thyroid gland are reviewed. Medullary carcinoma originates from the parafollicular or C cells of the thyroid gland. Calcitonin formation is an essential characteristic of medullary carcinoma, which explains the paraneoplastic syndrome seen with these tumors. Medullary carcinoma is associated frequently with pheochromocytoma, neurofibroma, and carcinoid tumor. (25 refs)

79-4294 α_1 -Antitrypsin Deficiency and Susceptibility to Lung Disease. (Eng) Evans, H. E. (Dept. Pediatrics, Jewish Hosp. and Medical Center Brooklyn, 555 Prospect Place, Brooklyn, NY 11238); Bognacki, N. *Environ Health Perspect* 29: 57-61; 1979.

The nature and properties of α_1 -antitrypsin (AAT) are reviewed together with the relationship between α_1 -antitrypsin deficiency and pulmonary disease. AAT deficiency occurs in persons with the homozygous Z phenotype, and it is estimated that about 80% of those with this phenotype will develop lung disease. (32 refs)

79-4295 Chronic Gastritis and Cancer of the Stomach. (Rus) Butov, Iu. L. (Dept. Pathological Anatomy, Inst. Advanced Training Physicians, Kharkov, USSR). *Arkhl Patol* 41(3): 71-76; 1979.

Current data on the role of chronic gastritis in the pathogenesis of stomach cancer are reviewed. Histological examination of 178 stomach specimens obtained at surgery for Stages I-III stomach cancer and 22 stage IV stomach cancer specimens obtained at autopsy showed that chronic gastritis had been present in 73.5% of the cases, adenomatous polyps in 16.5% of the cases, and chronic gastric ulcer in 10% of the cases. It was found that diffuse

or regional atrophic or hypertrophic gastritis can develop into all histological variants of stomach cancer, but follicular gastritis predisposes to the development of poorly differentiated squamous carcinoma. (72 refs)

- 79-4296 **Cancer as a Problem in Development.** (Eng) Braun, A. C. (Lab. Plant Biology, Rockefeller Univ., New York, NY 10021). *Cancer Outlaw Cell* 47-59; 1978.

The possibility that all carcinogens affect a common cellular mechanism that, once deranged, causes tumor cells to continue abnormal growth is considered. An epigenetic mechanism could account for the inheritable cellular changes found in tumor cells. With this mechanism, the DNA is undamaged, but its expression is altered, perhaps reversibly. Some tumors can develop without changes in cell genes, as demonstrated in experiments with plant tumor cells, plant crown gall disease cells, mouse teratocarcinoma cells, and squamous epidermoid carcinoma cells, all of which can give rise to normal organisms/cells if treated appropriately. In another study, nuclei from frog Lucke adenocarcinoma cells were implanted in enucleated frog eggs. Frog eggs that contained a cancer nucleus developed into normal healthy tadpoles. This indicates that cytoplasmic factors in the normal frog eggs reprogram the cancer nuclei, and it suggests that the cancer cells may not be irreversibly altered. Neuroblastomas occasionally change spontaneously into normal nerve cells and human acute myelocytic leukemia cells growing in culture revert to normal when treated with MGI protein. Substances effective in returning these cells to normal act by removing a block for differentiation in the cancer cells. In some cases, virus infection results in tumor formation; in others, the viral information is suppressed and only normal cells develop. The difference does not involve mutations in the genes but changes in how they are used. These examples demonstrate that any explanation for cancer must recognize the potential reversibility of the tumor state. (16 refs)

- 79-4297 **The Adenoma-Carcinoma Sequence in the Experimental Animal.** (Eng) Cole, J. W. (Comprehensive Cancer Center, Yale Univ., New Haven, CT). *Major Probl Pathol* 10: 119-125; 1978.

Evidence resulting from basic and clinical research suggests that many, if not most, adenocarcinomas of the colon are the terminal stage in the polyp-cancer sequence. The evidence, however, remains circumstantial. Review of the various experimental approaches to this sequence indicates that the actual cause of a neoplastic growth in the colonic epithelium will be multifactorial. (21 refs)

- 79-4298 **Epidemiology of Polyps and Cancer.** (Eng) Correa, P. (Charity Hosp., New Orleans, LA). *Major Probl Pathol* 10: 126-152; 1978.

The epidemiology of colon cancer (CC) and polyps of the large bowel is compared, and similarities are used as a basis for drawing inferences concerning the polyp-cancer sequence. Both CC and adenomatous polyps are strongly related to geography, anatomic localization (sigmoid tumors predominate in countries at high risk for CC and adenomatous polyps), socioeconomic class, migration experience, and time trends. Wealthy western societies are at high risk for adenomatous polyps and CC, and Japan and Latin American countries are at low risk. Migrants from countries where the risk of CC cancer and adenomatous polyps is low to countries where such risk is high acquire the high risk of the host country, usually after the second decade of migration. There is a socioeconomic gradient of similar magnitude for adenomatous polyps and CC in patients of different socioeconomic classes. The strength of the association between adenomatous polyps and CC favors a direct, positive correlation between multiple tumors, size and atypia of polyps, and cancer risks, equivalent to a 'dose-effect.' Adenomatous polyps are a good epidemiologic indicator of CC risk. There is no epidemiological evidence of a premalignant role for juvenile polyps. The epidemiology of hyperplastic polyps suggests environmental factors independent of those associated with CC. (56 refs)

- 79-4299 **The Adenoma-Carcinoma Sequence.** (Eng) Day, D. W. (Dept. Pathology, St. Mark's Hosp., London, England); Morson, B. C. *Major Probl Pathol* 10: 58-71; 1978.

The adenoma-carcinoma sequence and factors associated with the malignant transformation of adenomas are reviewed. In a series of 157 patients with synchronous carcinomas, 75% had associated adenomas. In several other series, contiguous benign tumors were found in a carcinoma. Of 323 synchronous malignant tumors, 87 showed evidence of benign tumor adjacent to the invasive cancer. The disproportionate prevalence of adenomas compared with adenocarcinomas in western populations indicates that malignant transformation is a relatively rare event. Three factors that are important in determining the malignant potential of adenomas are their size, growth pattern, and degree of epithelial atypia. The malignant potential is increased in large adenomas compared with small ones. Generally, the adenoma with a villous pattern has a higher malignant potential than the one with a tubular or mixed tubular-villous pattern. In one series, the malignancy rate was 5% for tubular adenomas and 40% for villous adenomas. The grading of adenomas into those showing mild, moderate, or severe dysplasia has shown that, irrespective of histologic growth pattern, their malignant potential increases with increasing degree of atypia. The

demonstration of grades of epithelial atypia in adenomas supports the concept of the adenoma-carcinoma sequence and suggests a gradual transition from a benign to a malignant tumor. The life history of this sequence is never < 5 yr; it averages 10-15 yr, but it may even cover a normal adult life-span. (31 refs)

- 79-4300 Juvenile and Peutz-Jeghers Polyps.** (Eng) Gibbs, N. M. (Univ. Surrey, Guildford, Surrey, England). *Major Probl Pathol* 10: 21-32; 1978.

The histology and malignant potential of juvenile (J) polyps and colonic Peutz-Jeghers (PJ) polyps are compared. Approx three-quarters of J polyps occur in the rectum, and there is no evidence that isolated polyps undergo malignant transformation. Malignancy within PJ polyps is difficult to substantiate, since a large proportion of colon carcinomas develop from adenomas, which may coexist with the polyps in the same colon. However, the epithelial dysplasia that has been noted in an occasional PJ polyp is indicative of potential malignant change. (15 refs)

- 79-4301 Hyperplastic Polyps (Synonym: Metaplastic Polyp).** (Eng) Gibbs, N. M. (Dept. Pathology, Univ. Surrey, Surrey, England); Katz, D. *Major Probl Pathol* 10: 14-20; 1978.

The hyperplastic polyp, previously confused with adenomas and sometimes termed "metaplastic", can be defined as a separate entity representing an alteration in maturation of normal mucosal epithelium. The basal colonic epithelial cell acquires absorptive features lower in the crypt. DNA synthesis is less rapid. Maturation characteristics are accentuated at the apex of the crypt cells. The factors that can produce this change in maturation are not known. (8 refs)

- 79-4302 Cancerogenesis, an Ubiquitous Hazard.** (Eng) Stock, C. C. (Sloan-Kettering Inst. Cancer Res., Donald S. Walker Lab., 145 Boston Post Road, Rye, NY 10580). *Prog Biochem Pharmacol* 14: 1-5; 1978.

Carcinogenesis is a ubiquitous hazard, between 75% and 90% of cancers being caused by factors in water, air, soil, food, and medicines. Nevertheless, studies in the field of chemical carcinogenesis have been neglected. Carcinogenesis studies in progress at the Memorial Sloan-Kettering Cancer Institute are reviewed briefly. (no refs)

- 79-4303 Firefighter Exposure to Environmental Carcinogens.** (Eng) Bendix, S. (Dept. City

Planning, City and County San Francisco, San Francisco, CA 94102). *J Combust Toxicol* 6: 127-135; 1979.

Two recent studies found steady increases in cancer among Toronto firefighters between 1945-1949 (15.4%) and 1965-1970 (38.4%) and excess cancer and leukemia deaths among firefighters. It is suggested that this increase results from exposure to carcinogens such as benzene, asbestos, chlorinated hydrocarbons, acrylonitrile, 4,4-methylene-bis(2-chloroaniline), benzidine, plastics, pesticides, and carcinogens from wood and fossil fuels. Recommendations to protect firefighters from such exposures are given. (44 refs)

- 79-4304 Surgical Management and Epidemiology of Lip Cancer.** (Eng) Lore, J. M. (2121 Main St., Buffalo, NY 14214); Kaufman, S.; Grabau, J. C.; Popovic, D. N. *Otolaryngol Clin North Am* 12(1): 81-95; 1979.

Literature concerning the epidemiology, natural history, diagnosis, treatment, and prognosis of lip cancer is reviewed. Lip cancer accounts for 10% of the 40,000 cases of head and neck cancer diagnosed annually in the US. The etiology may involve exposure to the sun or the use of tobacco. Squamous cell lip carcinoma is often accompanied or preceded by leukoplakia, recurrent or persistent ulceration, warty outgrowths, or erythroplasia. Generally, lip carcinomas remain localized for long periods and grow slowly. (25 refs)

- 79-4305 Epidemiology of Oral Cancer.** (Eng) Sellars, S. L. (Dept. Otolaryngology, Medical Sch., Univ. Cape Town, Cape Town, S. Africa 7925). *Otolaryngol Clin North Am* 12(1): 45-55; 1979.

Worldwide incidences and etiological factors associated with oral cancer (OC) are reviewed. In most Western countries, the incidence of OC in men has declined over the past 20 yr, a trend attributable to changes in smoking and chewing habits, improved oral hygiene, eradication of dietary deficiencies, and effective treatment of syphilis. In some of these countries, the incidence has increased in women. There is a 30-fold variation in the OC incidence rate among different countries. Eighty percent of patients are > 50 yr old and, except in India, 70%-80% are men. The high incidence of OC among Indian women has been attributed to the habit of betel chewing: 70% of Indian women but only 10% of the men chew betel. The traditional list of etiological factors includes syphilis, sepsis, alcohol, and tobacco. Recent additions are trauma, infection, and dietary deficiencies. Under conditions of poor oral hygiene, secondary infection occurs at traumatized sites, followed by chronic inflammatory mucosal hyperplasia. This tissue reaction also results from dentures. In one age-

matched series, OC was twice as common in the edentulous. Two infections, candidiasis and herpes, have been linked to OC. Iron deficiency has been specifically related to cancer of the mouth, pharynx, and esophagus. Although long-standing malnutrition is related to mucosal atrophy and to leukoplakia, OC is not more common among the chronically malnourished peoples of the world. (3 refs)

- 79-4306** Re: "A Study of Diet and Breast Cancer" (2 Letters to Editor). (Eng) Bross, I. D. (Roswell Park Memorial Inst., Buffalo, NY 14263); Miller, A. B.; Howe, G. R. *Am J Epidemiol* 109(5): 619-620; 1979.

A study implicating increased nutrient intake in the etiology of breast cancer is criticized on the grounds that this claim was contradicted by the evidence in the paper itself and by other studies. The authors of the paper point out that to the extent that mammary cancer in rodents is a reflection of mammary cancer in humans, a series of studies have shown an association between cancer and diet, particularly total fat intake. In addition, a number of investigators have correlated human breast cancer incidence and mortality with dietary factors. (3 refs)

- 79-4307** Aetiology of Breast Cancer: A Brief Review. (Eng) Roe, F. J. (4 Kings Road, Wimbledon, London SW19 8QN, England). *Invest Cell Pathol* 2(1): 45-53; 1979.

Current knowledge of the cause of human breast cancer (BC) is reviewed in light of the two-stage concept of carcinogenesis. Human BC is probably a group of diseases that have different causes. Changes in hormonal status that increase BC risk probably do so by "promoting" tumor development rather than by "initiating" it. Exogenous estrogens seem to act as tumor promoters in this context, but there is no evidence that oral contraceptives, some of which contain estrogens in low dosage, increase BC risk. On the contrary, they appear to reduce the incidence of benign breast tumors. Prolactin release is associated with increased mammary tumor incidence in rats but not in humans. There is no evidence that viruses or exposure to hair dyes increases BC risk. The fact that a slight dietary restriction can dramatically reduce mammary tumor incidence in rats suggests that dietary factors should be looked at more closely in the search for etiological factors in humans. (28 refs)

- 79-4308** Epidemiological Research on the Relationship Between Tobacco, Alcohol and Cancer. (Eng) Flamant, R. (Departement de Statistique Medicale de l'Institut Gustave Roussy, Villejuif, France). *Prog Biochem Pharmacol* 14: 36-46; 1978.

Epidemiologic studies of the relationship between tobacco, alcohol, and cancer are reviewed. Cancers associated with smoking are those of the lung, larynx, pharynx, oral cavity, esophagus, and bladder. All except esophageal cancer are related to the amount smoked, and lung, laryngeal, and bladder cancer are related only to the inhalation of cigarette smoke. Two studies show that the risk of lung cancer is reduced in men smoking filter-tipped cigarettes and in men who have quit smoking. Based on studies in different countries, it is hypothesized that the cancer risk varies with the kind of tobacco smoked. Alcoholism is associated with increased mortality from cancers of the esophagus, hypopharynx, and other sites in the upper respiratory and alimentary tracts. The effect of alcohol is limited to certain geographic areas and is less important than that of tobacco. Upper respiratory and alimentary cancers are related nearly equally to the use of tobacco and alcohol. To a certain extent, the higher the correlation between the sites and the use of tobacco and alcohol, the higher the male:female sex ratio. (10 refs)

- 79-4309** Epidemiology of Alcohol and Cancer. (Eng) Tuyns, A. J. (Unit Epidemiology and Biostatistics, International Agency Res. Cancer, 150 Cours Albert Thomas, 69372 Lyon Cedex 2, France). *Cancer Res* 39(7, part 2): 2840-2843; 1979.

Both prospective and retrospective epidemiological studies indicate that alcohol consumption is a cancer hazard. Prospective studies of excessive drinkers have shown an increased risk for cancer of the mouth, pharynx, larynx, esophagus, liver, and lung. Retrospective studies have confirmed this excess risk. For cancers of the buccal cavity, pharynx, larynx, and esophagus, the effect of drinking has been shown to be associated with the effect of smoking. In the case of esophageal cancer, these two effects are independent, and the observations made are consistent with a multiplicative model. Primary liver cancer is also associated with alcohol consumption, probably by a less direct action; the importance of the impact of alcohol on primary liver cancer is probably underestimated. Animal experiments have not shown that ethanol alone has a carcinogenic effect, and the mechanisms by which alcoholic beverages act on humans remain unknown. The proportion of cancer cases at sites known to be associated with alcohol consumption is approx 8% in most population groups in the US. This indicates that a sizeable proportion of cancers is potentially preventable if appropriate action is taken. (29 refs)

- 79-4310** Epidemiological Opportunities in Alcohol-related Cancer. (Eng) Fraumeni, J. F. (Environmental Epidemiology Branch, NCI, Bethesda, MD 20205). *Cancer Res* 39(7, part 2): 2851-2852; 1979.

Epidemiological studies have clearly demonstrated that alcohol interacts with tobacco smoke in the development of cancers of the oropharynx, esophagus, and larynx. It should be possible to clarify further the role of alcohol itself, the modifying effects of tobacco, dose-response relationships, and nutritional cofactors. Studies are also needed to delineate the steps by which alcohol consumption leads to liver cancer and to resolve the suggestion that certain beverages may predispose to other cancers, including those of the pancreas and rectum. Epidemiological investigations should be combined with experimental work to identify hazardous fractions in alcoholic beverages and to delineate the mechanisms by which alcohol promotes carcinogenesis. Epidemiologists and biometricians may also contribute toward the development of programs aimed at primary prevention and early detection of cancers related to alcohol and tobacco. Incorporation of research questions into data collection systems deserves serious consideration as a means of obtaining additional valuable information for etiological studies. (no refs)

- 79-4311 Etiologic Factors in Lung Cancer.** (Eng) Weiss, W. (Philadelphia County Medical Society, 2100 Spring Garden St., Philadelphia, PA 19130). *Phila Medicine* 75(5): 202-204; 1979.

Possible etiologic agents in lung cancer that have been studied so far include smoking, asbestos, radiation in uranium miners, chloromethyl ethers, and ambient air pollution. The evidence linking cigarette smoking with lung cancer is strong: there is a striking dose-response relationship, the incidence of the disease tends to diminish when smokers stop smoking, the causal relationship has been confirmed in animal studies, and the hypothesis that smoking is related to lung cancer is coherent with current knowledge of the natural history and epidemiology of the disease. (no refs)

- 79-4312 Cancer in Patients on Hemodialysis (Letter to Editor).** (Eng) Jacobs, C. (Groupe Hospitalier Pitie-Salpetriere, Paris, France); Reach, I.; Degoulet, P. *N Engl J Med* 300(22): 1279-1280; 1979.

Malignant tumors were detected in 115 (67 male and 48 female) uremic patients on maintenance dialysis in 168 centers. The incidence of malignant tumors among such patients in these centers was 9.31/1,000 in 1977. The pathogenesis of tumors in the 115 patients was more closely related to age than to duration of chronic renal failure. The occurrence of malignant tumors was closely associated with a history of analgesic nephropathy but not with polycystic renal disease. (5 refs)

- 79-4313 Nutrition, Carcinogenesis, and Mutagenesis.** (Eng) Newberne, P. M. (Dept. Nutrition and

Food Science, Massachusetts Inst. Technology, Cambridge, MA); Zeiger, E. *Adv Mod Toxicol* 5: 53-84; 1978.

The relationship between nutrition and cancer of various sites is reviewed. There is highly suggestive evidence, generally from epidemiological studies, that nutrition is related to gastric cancer in some human populations; but the specific agents are still unknown, and in the case of animal experiments, the evidence is equivocal. There is also conflicting evidence on the role of protein and fat in relation to colon cancer. Refined foods that are low in fiber have been connected with colon cancer. In animal studies, vitamin A deficiency increased the number of colon tumors induced by some chemicals. Intestinal aryl hydrocarbon hydroxylase (AHH) can be modified by diet and can, in turn, modify chemical carcinogenesis. Studies in Africa suggest that carcinogenic food contaminants probably interact with nutritional deficits to result in liver cancer. In the US, hepatocarcinoma is associated with alcoholic cirrhosis. Liver cancer has also been associated with increased dietary lipids, lipotrope deficiency, protein deficiency, and high levels of vitamin B₁₂. Breast cancer appears to be associated with increased body mass (both height and wt). Some potential carcinogens present in the diet or water appear to be associated with urinary bladder cancer. A dietary survey of men with lung cancer revealed a negative association with an index of vitamin A intake. This was not confirmed in animal studies. Hydrocarbon carcinogenesis in the animal lung can be affected by dietary modification of pulmonary AHH activity. It is concluded that nutrition offers the most acceptable and direct means of attacking the cancer problem in humans. (133 refs)

- 79-4314 Diverticulosis and Carcinoma of the Large Intestine as Fiber Deficiency Diseases: Fact or Hypothesis?** (Ger) Miller, B. (Medizinische Klinik und Poliklinik D, Universitat Dusseldorf, Moorenstrasse 5, D-4000 Dusseldorf, W. Germany); Strohmeyer, G. *Internist (Berlin)* 20(4): 195-200; 1979.

Etiological and epidemiological studies of the possible relationships between diet and diverticulosis and colon carcinoma (CC) are reviewed. Although environmental factors play an important role in CC, the effects of carcinogenic food additives in particular are negligible. The carcinogens and cocarcinogens responsible for CC are formed from normal food components or from endogenous substances, such as digestive juices. Tryptophan and tyrosine can be transformed into mutagenically active metabolites by methods of food preparation or by intestinal bacteria. Dehydroxylating intestinal bacteria can form cocarcinogens from bile acids and cholesterol. Because of their water-binding capacity, dietary fibers reduce the concentration of carcinogens and cocarcinogens in the contents of the large bowel, and thus, may exert a certain protective effect against CC. However, the acceleration of intestinal transit time by dietary fibers has no

effect on cancer risk. Epidemiological studies have demonstrated a positive correlation between the incidence of CC and a high-fat or a high-protein diet, but at best a slight protective effect of a high-fiber diet. (43 refs)

- 79-4315 Etiology of the Adenoma-Carcinoma Sequence.** (Eng) Hill, M. (Bacterial Metabolism Res. Lab., Central Public Health Lab., Colindale, London, England). *Major Probl Pathol* 10: 153-162; 1978.

The epidemiology of intestinal adenomas is briefly considered, and an etiological hypothesis is postulated. The proportions of persons carrying adenomas and the number of adenomas per carrier are lower in Africa, Asia, and South America than in North America and Northwest Europe. The incidence of adenomas in Japanese living in Japan is lower than that in Japanese born in Hawaii, and the incidence is lower in black South Africans than in black Americans. Thus, the incidence rates seem to differ because of some environmental factor. Adenomas in Japan and Colombia, which have very low incidences of large bowel cancer, are small compared with those in Sweden, US, and Britain. Thus, there appears to be a direct relationship between the size of the adenomas and the incidence of large bowel cancer. There is some evidence of a genetic predisposition to large bowel cancer and, by inference, to adenomas. If it is assumed that in the West all adenoma-prone persons actually develop adenomas, then the gene is extremely widespread and is as common in Japanese and Negroes as in Caucasians. An environmental factor, E_1 , is postulated to account for the difference in incidence of adenomas between the Japanese living in Japan and those living in Hawaii. A second environmental factor, E_2 , is postulated to explain why so few of the Japanese or Colombian adenomas grow to a large size. A third environmental factor, C, which is able to produce malignancy in adenomatous tissue, is also postulated. Since the proportion of large adenomas progressing to carcinoma is the same in Japan as in Britain and Sweden, factor C must be fairly uniformly distributed. Although factor C is potentially able to induce malignancy in any cell, the large adenoma is postulated to be extremely sensitive to C because of the large mass of adenomatous tissue involved. (18 refs)

- 79-4316 N-Acetyltransferase Phenotype and Risk in Urinary Bladder Cancer: Approaches in Molecular Epidemiology. Preliminary Results in Sweden and Denmark.** (Eng) Lower, G. M. (Dept. Human Oncology, Univ. Wisconsin Center Health Sciences, Madison, WI 53706); Nilsson, T.; Nelson, C. E.; Wolf, H.; Gamsky, T. E.; Bryan, G. T. *Environ Health Perspect* 29: 71-79; 1979.

Possible correlations between N-acetyltransferase phenotype and urinary bladder cancer risk was studied among 115 patients from the rural area surrounding Lund, Sweden, and 71 patients from Copenhagen, Denmark. The slow acetylator phenotype was found in 46/71 Copenhagen patients and 38/74 controls from the same area ($p = 0.065$). Smoking histories were similar for rapid and slow acetylators. The slow acetylator phenotype was found in 80/115 Lund patients and 79/118 controls from the same area; the difference was not statistically significant. The results suggest that arylamines may play a role in disease etiology in Copenhagen and that slow acetylators may be at higher risk for arylamine-induced bladder cancer. (54 refs)

- 79-4317 Current Problems in Sexually Transmitted Diseases.** (Eng) Sparling, P. F. (Dept. Medicine, Univ. North Carolina, Chapel Hill, NC). *Adv Intern Med* 24: 203-228; 1979.

Topics discussed in this review of problems in sexually transmitted diseases (STD) include homosexuality and STD, gonorrhea, nongonococcal urethritis, genital chlamydia and ureaplasma in other syndromes, syphilis, and genital herpetic infections. Preliminary results suggest that there is an increased risk of cervical cancer in women with a genital Herpesvirus hominis infection. (130 refs)

- 79-4318 Measurement of Chemical Carcinogens in the Human Environment: The Objectives and Problems Encountered.** (Eng) Griecute, L. (Unit Environmental Carcinogens, IARC, 150 Cours Albert Thomas, F-69008 Lyon, France). *Prog Biochem Pharmacol* 14: 57-69; 1978.

The measurement of chemical carcinogens in the human environment and problems encountered in doing so are reviewed. With regard to establishing priorities for analysis, the only realistic approach would be analysis of environmental substrates for known chemical carcinogens and estimation of the total load. The regions most suited to this type of study would be those where traditional ways of life have been maintained and where peculiar patterns of cancer morbidity have been found. The problems that arise when known chemical carcinogens are measured fall into two categories: inadequate techniques and difficulties in the interpretation of the acquired data. Progress to date in the standardization of analytical techniques for volatile N-nitrosamines, benzo(a)pyrene, and aflatoxin is reported. (27 refs)

- 79-4319 Toward Less Hazardous Cigarettes (2 Letters to Editor).** (Eng) Warner, K. E. (Univ. Michigan Sch. Public Health, Ann Arbor, MI); Gori, G. B.; Lynch, C. J. *JAMA* 241(20): 2143-2144; 1979.

In a recent article, critical levels of selected cigarette smoke constituents expressed in terms of pre-1960 cigarettes were translated into equivalent numbers of present-day low-tar and -nicotine cigarettes to help smokers wean themselves to less-hazardous cigarettes. The analysis in this article was based on some highly questionable assumptions that, if wrong, challenge the validity of the conclusions. Also, the article did not take into account relative risks of certain high-risk groups. Thus, the article did not provide any scientifically meaningful evidence that certain present-day smoking behaviors will result in statistically nondetectable health risks. In reply, the authors of the original article state the basis for their assumptions and evidence supporting them. The entire thrust of the article was to suggest a method for changing present-day smoking behaviors through progressively less hazardous and less addictive smoking stages. (2 refs)

79-4320 Socially Tolerable Cigarette Smoke (2 Letters to Editor?) (Eng) Homburger, F. (Bio-Res. Inst., Cambridge, MA); Gori, G. B.; Lynch, C. J. *JAMA* 241(20): 2142-2143; 1979.

An article that gave the impression that some cigarettes are relatively safe to smoke in limited quantity is criticized. The number of current cigarettes that would expose the smoker to amounts of tar, nicotine, carbon monoxide, nitrogen oxides, hydrogen cyanide, and acrolein that were tolerated without demonstrable effect with pre-1960 cigarettes was calculated. However, such extrapolation from pre-1960 cigarette smoke to today's smoke would be justified only if the composition of the diluted smoke remained basically the same. The manipulations necessary to obtain lower yields of the six substances under consideration, alter the proportions of other ingredients and may change the biologic activity of the smoke. Only a valid and practical in vivo bioassay of the smoke itself, as it is inhaled by human smokers, can discriminate between hazardous and less-hazardous smoke. The authors of the original article replied that tests with >130 types of experimental cigarettes confirm that today's cigarette processing reduces known toxic constituents quantitatively and that there is no evidence of an increase in other suspect toxic constituents. The authors agreed on the need for an in vivo bioassay of the smoke itself. (4 refs)

79-4321 Measurements of Drinking Patterns in the General Population and Possible Applications in Studies of the Role of Alcohol in Cancer. (Eng) Room, R. (Social Res. Group, Sch. Public Health, Univ. California, Berkeley, CA 94720). *Cancer Res* 39(7, part 2): 2830-2833; 1979.

The kinds of measurements used to study drinking practices in the general population and their application to

studies of the role of alcohol in cancer development are described. Quantitative information on alcohol consumption patterns derives from four possible sources: indirect measures, observational studies, aggregate consumption statistics, and sample surveys of general populations. The potentials and problems of each method are briefly discussed, with primary attention being given to the various traditions of survey questioning and data analysis. Although medically oriented epidemiologists have often used only an overall drinking volume measure, social scientists have pointed to the importance also of variability in characterizing drinking, particularly in relation to social and casualty problems as opposed to chronic health problems with drinking. The dimensions of drinking patterns that might be relevant to hypothesized linkages of alcohol and cancer are discussed. It is suggested that measurements will need to extend beyond the volume of drinking and may indeed involve studies of new kinds of dimensions in drinking patterns. (21 refs)

79-4322 Preventing Occupational Cancer. (Eng) Bates, R. R. (National Inst. Environmental Health Sciences, NIH, Bethesda, MD 20014). *Environ Health Perspect* 28: 303-310; 1979.

The prevention of occupational cancer is discussed. The development of cancer is dependent on the level of exposure to carcinogenic agents and on multiple environmental and genetic factors that affect individual susceptibility. Prevention of cancer depends on identifying the causative factors and controlling them whenever possible. Animal experiments are needed to identify chemical carcinogens before they cause cancer in humans. However, there is much debate about the interpretation of animal experiments designed to detect chemicals that may be carcinogenic for humans. The experiments are usually conducted at considerably higher levels of exposure than those commonly occurring in humans, the reason being to enhance the sensitivity of the bioassay. It is assumed that there is no threshold below which exposure to a chemical carcinogen entails no risk, but it is not possible to decide unequivocally whether or not thresholds exist. Another debated issue is the significance of benign tumors as an index of the carcinogenicity of a chemical. There is often no sharp distinction between benign and malignant tumors, and a number of studies have suggested that the presence of benign tumors indicates that their inducing agents are capable of causing malignancy. Prevention of cancer requires that action be taken on the basis of reasonable evidence of a potential hazard, even if this sometimes results in what may be shown later to be unnecessary controls. (53 refs)

79-4323 Predictive Carcinogenicity Bioassays in Industrial Oncogenesis. (Eng) Maltoni, C. (Inst.

Oncology and Tumour Center, Bologna, Italy). *Prog Biochem Pharmacol* 14: 47-56; 1978.

The use of carcinogenicity bioassays to predict the oncogenic risks associated with industrial chemicals is reviewed. The goal is to avoid human exposure to carcinogenic chemicals. The four most important cases of environmental and occupational cancers discovered since 1970 were directly or indirectly predicted experimentally. The choice of agents to be tested should follow a list of priorities based on diffusion of the agent, number of people potentially exposed, and direct and/or indirect data suggesting a possible risk. Long-term carcinogenicity bioassays include first-level bioassays in which the route of administration of the agent and affected tissues are not the same as those for humans; second-level bioassays in which the route of administration of the agent is different but the affected tissue is the same as that for humans; and third-level bioassays in which both the route of administration and affected tissues are the same as those for humans. Preliminary results of bioassays of several inorganic compounds and vinyl chloride in Sprague-Dawley rats are given. (9 refs)

79-4324 Health Problems of Anaesthetists and Their Families (Letter to Editor). (Eng) Tomlin, P. J. (Univ. Dept. Anaesthetics, Queen Elizabeth Hosp., Birmingham B15 2TH, England). *Br Med J* 1(6173): 1280-1281; 1979.

Critics of a previous article on health problems of anesthetists and their families apparently missed the key points of that article: there are more health problems in the families of anesthetists than occur in the general population and conventional statistical assumptions are biased against the findings of a health hazard in investigations dealing with safety. (8 refs)

79-4325 Historical Background to the Asbestos Problem. (Eng) Lee, D. H. (Nazareth 5-1, Deer Hill Road, St. Thomas, Virgin Islands 00801); Selikoff, I. J. *Environ Res* 18(2): 300-314; 1979.

The history of the use of asbestos and its medical hazards are reviewed. The pulmonary effects of asbestos were recognized as early as 1898; in 1927, the term "asbestosis" was used to describe lung disease caused by asbestos inhalation. The association between asbestosis and pleural and pericardial mesothelioma (MT) was reported in 1947, but only in 1960 was the existence of MT established. Lung carcinoma in asbesto-silicosis was first reported in 1935. Reduction of heavy exposures that led to early death revealed slowly developing diseases such as MT and bronchogenic carcinoma with increasing clarity. (95 refs)

79-4326 Functional Role of Coagulative and Anticoagulative Blood System in the Development of Malignant Tumors. (Rus) Kudriashov, B. A. (Dept. Physiology Humans and Animals, M. V. Lomonosov State Univ., Moscow, USSR); Kalishevskaya, T. M.; Kolomina, S. M. *USP Fiziol Nauk* 10(2): 3-23; 1979.

Current data on the role of the blood coagulation system in carcinogenesis are reviewed. The high incidence of thrombotic complications in cancer patients indicates that the development of a malignant tumor is associated with an increase in the functional activity of the coagulative system (CS) and a simultaneous inhibition of the activity of the anticoagulative system (ACS). Administration of anticoagulants or fibrinolytic agents to tumor-bearing animals could inhibit the development of hematogenic and lymphogenic metastases and decrease the number of tumor cells circulating in the blood. (189 refs)

CHEMICAL CARCINOGENESIS

79-4327 HLA-A and B Antigen Frequencies in an Asbestos Exposed Population with Normal and Abnormal Chest Radiographs. (Eng) Darke, C. (Tissue Typing Lab., Blood Transfusion Centre, Cardiff CF5 6XF, Wales); Wagner, M. M.; Grant McMillan, G. H. *Tissue Antigens Histocompat Immunogenet* 13(3): 228-232; 1979.

Previous studies of histocompatibility (HLA) antigen frequencies in asbestos-related pulmonary fibrosis have suggested some weak associations both with susceptibility to the disease (B12 and B27) and protection from the disease (B18 and Bw35). HLA typing was performed on a further series of 64 asbestos workers with no chest abnormality and 166 workers with various radiographic changes, 78 of whom had pulmonary fibrosis. The results failed to give statistical confirmation of these associations, although B27 was twice as frequently associated with pulmonary fibrosis and diffuse pleural thickening. Analysis of the combined data from this and four other studies failed to show any consistent associations. (11 refs)

79-4328 The Cytotoxic, Mutagenic and Clastogenic Effects of Chromium-containing Compounds on Mammalian Cells in Culture. (Eng) Newbold, R. F. (Inst. Cancer Res., Pollards Wood Res. Station, Chemical Carcinogenesis Div., Nightingales Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, England); Amos, J.; Connell, J. R. *Mutat Res* 67(1): 55-63; 1979.

The cytotoxic, mutagenic, and clastogenic effects of various chromic and chromate salts on cultured Chinese hamster cells (line V79/4) were studied. Potassium dichromate (PDC: highly water-soluble) and zinc chromate (ZC: less soluble) were cytotoxic and mutagenic in a dose-dependent manner. PDC was about fivefold more effective than ZC. Chromic acetate and lead chromate were inactive in this system at 200 and 10 times, respectively, the max dose at which cell survival was measurable with PDC. The dose-response curve exhibited by PDC was the type usually associated with agents having a general toxic effect on cells, rather than with agents that tend to attack a more specific cell target, such as DNA. Mutation frequency increased linearly with increasing lethality only when subthreshold (minimally lethal) doses of PDC were used. At all doses, PDC was a very powerful clastogenic agent, producing predominantly chromatid breaks. (42 refs)

79-4329 Preliminary Study on the Carcinogenic Activity of the Fungicide Manganese Ethylenebisdi-

thiocarbamate in the Adult Newt, *Triturus Cristatus Carnifex*. (Eng) Zavanella, T. (Istituto di Zoologia, Via G. Celoria 10, 20133 Milano, Italy); Arias, E.; Zaffaroni, N. *P. Tumori* 65(2): 163-167; 1979.

The carcinogenic effects of percutaneous manganese ethylenebisdithiocarbamate (maneb: 0.5-5.0 ppm in the ambient water for 19-23 wk) in adult newts (*Triturus cristatus carnifex*) were studied. No tumors were found in any animal, although maneb-treated newts showed some soft tissue edema; marked splenic enlargement, blood stasis, and lymphocyte depletion; diffuse vascular congestion; and, in some cases, the presence of proteinaceous material in the renal tubular lumen. Hepatic glycogen levels were increased in males, a finding that is indicative of a disturbance in carbohydrate metabolism. The negative results of this study may be due to the low doses of maneb tested. (13 refs)

79-4330 Mutagenic Effects of Ozone on Human Cells Exposed In Vivo and In Vitro Based on Sister Chromatid Exchange Analysis. (Eng) Guerrero, R. R. (Pasadena Foundation Medical Res., Pasadena, CA 91101); Rounds, D. E.; Olson, R. S.; Hackney, J. D. *Environ Res* 18(2): 336-346; 1979.

The frequency of sister chromatid exchanges (SCE) in the circulating lymphocytes of 31 young adult volunteers exposed to 0.5 ppm ozone for 2 hr (simulating conditions in severe air pollution episodes) were determined. There was no significant difference in SCE frequency between control and ozone-exposed lymphocytes. In contrast, human diploid fetal lung cells (WI-38) exposed to ozone in vitro showed a linear dose-related increase in SCE over an ozone concentration range of 0 to 1.00 ppm. Even the lowest ozone concentration (0.25 ppm) produced a level of SCE (0.158/chromosome) which was significantly higher than the control value (0.129/chromosome) ($p < 0.05$). Although the number of chromosome deletions, dicentric chromosomes, and chromosome fragmentations in the ozone-treated cells (0%-2%) did not differ from control (1%), the frequency of chromatid deletions following treatment with 1.0 ppm ozone showed a 7-fold increase over the control value; there was no significant increase in the latter type of aberration following lower ozone exposures. It is concluded that lack of evidence for genetic damage in lymphocytes does not preclude the potential for mutagenic changes in cells which are subject to direct ozone exposure. (53 refs)

79-4331 Absence of a Mutagenic Effect of Dichlorvos in *Drosophila melanogaster*. (Eng) Sobels, F.

H. (Dept. Radiation Genetics and Chemical Mutagenesis, Univ. Leiden, Sylvius Labs., Wassenaarseweg 72, Leiden, Netherlands); Todd, N. K. *Mutat Res* 67(1): 89-92; 1979.

The possible mutagenic activity of dichlorvos (2,2-dichlorovinyl dimethyl phosphate; DDVP) was tested in *Drosophila melanogaster* using a standard assay for detecting sex-linked recessive lethal mutations. Male flies were fed 0.2 ml of 6.0×10^{-10} M DDVP, 0.2 ml of 3.0×10^{-9} M DDVP, 0.2 ml of 6.0×10^{-8} M DDVP, or 0.2 ml of 6.0×10^{-7} M DDVP plus 0.2 ml 1% sucrose soln. After a 24-hr feeding period, the males were mated to virgin females. The F_2 progeny were assessed for the presence of wild-type male flies, and all presumed lethals were checked in F_3 matings. DDVP treatment did not raise the frequencies of sex-linked recessive lethal mutations significantly over those in controls. Thus, the results provide no evidence of any mutagenic activity of DDVP in adult *Drosophila*. (8 refs)

- 79-4332 **Platinum-induced Mutations to 8-Azaguanine Resistance in Chinese Hamster Ovary Cells.** (Eng) Taylor, R. T. (Biomedical Sciences Div., Lawrence Livermore Lab., Livermore, CA 94550); Carver, J. H.; Hanna, M. L.; Wandres, D. L. *Mutat Res* 67(1): 65-80; 1979.

The cytotoxicities and mutagenicities of six related platinum compounds were compared in suspension cultures of Chinese hamster ovary (CHO-S) cells. The toxicity of these compounds could be ranked according to concentrations that decrease suspension growth/cloning efficiency by 50%: $\text{cis-Pt}(\text{NH}_3)_2\text{Cl}_2$ ($0.9/1.5 \mu\text{M}$) > $\text{Pt}(\text{SO}_4)_2$ + methylcobalamin (MeB-12) methylation product ($20/10 \mu\text{M}$) > K_2PtCl_4 ($32/50 \mu\text{M}$) = K_2PtCl_6 ($34/50 \mu\text{M}$) = MePtCl_3^{2-} ($60/50 \mu\text{M}$) > $\text{Pt}(\text{SO}_4)_2$ ($66/105 \mu\text{M}$). Following 20-hr exposures to concentrations that resulted in relative survivals of 2%-80%, none of the compounds consistently increased the frequency of ouabain-resistant (OUA-r) mutants above the spontaneous frequency (6.0×10^{-6}). Parallel treatments with $800 \mu\text{M}$ ($100 \mu\text{g/ml}$) ethyl methanesulfonate (EMS) increased the OUA-r mutant frequency 10- to 12-fold. Using 8-azaguanine (8-AG) for mutant selection, dose-dependent increases that were five- to sevenfold above the spontaneous frequency (3.8×10^{-5}) were obtained with $\text{cis-Pt}(\text{NH}_3)_2\text{Cl}_2$, $\text{Pt}(\text{SO}_4)_2$, and the product from $\text{Pt}(\text{SO}_4)_2$ + MeB-12. Identical 20-hr exposures to varying amounts of K_2PtCl_4 , K_2PtCl_6 , and MePtCl_3^{2-} did not induce 8-AG-r mutants. Optimal detection of Pt-induced 8-AG-r mutants required seven posttreatment, population doublings in suspension culture. Under the selection conditions used, 8/8 spontaneous and 24/24 Pt-induced 8-AG-r variants contained reduced hypoxanthine-guanine phosphoribosyltransferase (HGPRT) activities (means ranging from 3% to 11% of the parental CHO-S cells). When linear plots of 8-AG-r frequency were compared with initial medium concentration, $\text{cis-Pt}(\text{NH}_3)_2\text{Cl}_2$

was 134 times and $\text{Pt}(\text{SO}_4)_2$ was 3.5 times more mutagenic than EMS. However, on a cell-survival basis, EMS was 8- to 10-fold more mutagenic than these two Pt compounds. The sensitivity of the CHO-S HGPRT locus for detecting mutagenesis by Pt complexes could be increased severalfold by continuous subculture of the cells in the presence of these agents for 10-25 population doublings. By this procedure, K_2PtCl_6 was weakly mutagenic and $20 \mu\text{M}$ $\text{Pt}(\text{SO}_4)_2$ produced 8-AG-r mutants at frequencies requiring seven- to eightfold higher concentrations when a fixed 20-hr exposure was used. (36 refs)

- 79-4333 **N-Formyliminodiacetic Acid, a New Compound from the Reaction of Nitrilotriacetic Acid and Chlorine.** (Eng) Spanggord, R. J. (Life Sciences Div., SRI International, Menlo Park, CA 94025); Tyson, C. A. *Science* 204(4397): 1081-1082; 1979.

The trisodium salt of the monohydrate of nitrilotriacetic acid was mixed at room temperature and pH 11 with NaOCl solution. The reaction products included formate, bicarbonate, formaldehyde, N-(formyl)-N-(carboxymethyl)-glycine (FIDA), and iminodiacetic acid. FIDA, which was also formed at pH 5, was nonmutagenic with and without metabolic activation for *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA1538. (10 refs)

- 79-4334 **Mechanism of the Lethal and Mutagenic Effects of Phenoxyacetic Acids in *Saccharomyces cerevisiae*.** (Eng) Zetterberg, G. (Dept. General Genetics, Univ. Uppsala, S-750 07 Uppsala, Sweden). *Mutat Res* 60(3): 291-300; 1979.

2-Methyl-4-chlorophenoxyacetic acid (MCPA) and salicylic acid, two compounds with similar structures and almost the same dissociation pattern, were tested for lethal and mutagenic effects on, and uptake by, cells of *Saccharomyces cerevisiae* strain *rad18*. The two compounds produced similar results, suggesting a common mechanism of action. It is proposed that they act by increasing the concentration of hydrogen ions within the cell, so that killing and mutation occur. Mutations were induced only when killing reached 95%-99%. The compounds are considered weak mutagens for yeast cells. The methyl ester of MCPA also induced killing and reverse mutation, but only at concentrations about 100 times higher than that for the undissociated acid. MCPA methyl ester did not increase the number of revertants in the *Salmonella*/liver microsome test. It is suggested that the effects of the methyl ester of MCPA depend on the ester being hydrolyzed to the acid by yeast cells and the liver microsome preparation. (14 refs)

- 79-4335 **Mutagenicity Assay of an *Agaricus bisporus* Extract.** (Eng) De Flora, S. (Inst. Hygiene,

Sch. Medicine, Univ. Genoa, Genoa, Italy); Cajelli, E.; Brambilla, G. *IRCS Med Sci (Cancer)* 7(4): 185; 1979.

A methanolic extract of *Agaricus bisporus* mushrooms was weakly mutagenic toward *Salmonella typhimurium* strain TA100 in the *Salmonella*/microsome test, at concentrations just below the toxicity level. The extract was negative in the plate incorporation assay, even at concentrations up to 16 mg/plate, and no significant increase in revertants could be detected after preincubation of the extract with human gastric juice. (4 refs)

79-4336 Carcinomas of the Liver in Osborne-Mendel Rats Ingesting Methoxychlor. (Eng) Reuber, M. D. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701). *Life Sci* 24(15): 1367-1371; 1979.

The induction of liver carcinomas by technical methoxychlor (0, 10, 25, 100, 200, 500, or 2,000 ppm incorporated in the diet for 104 wk) was studied using male and female Osborne-Mendel rats. Ten of 52 treated females (19%) developed hyperplastic nodules or carcinomas of the liver as compared with $\leq 1/50$ usually observed in control rats ($p = 0.000478$). Eight of 32 males (25%) given methoxychlor developed hyperplastic nodules or carcinomas of the liver as compared to 0% of control males ($p = 0.00189$). The hyperplasia in the control rats was classified as 0-1+, whereas that in treated rats increased from 2-3+ in animals given 25 ppm methoxychlor to 5+ in those given 2,000 ppm. Five of 10 females given 500 ppm methoxychlor and 1/10 given 100 ppm developed carcinomas of the ovary. The liver tumors were undifferentiated to well-differentiated hepatocellular carcinomas. (29 refs)

79-4337 Alcoholism and Cancer. I. Effects of Long-Term Exposure to Alcohol on Spontaneous Mammary Adenocarcinoma and Prolactin Levels in C3H/St Mice. (Eng) Schrauzer, G. N. (Dept. Chemistry, Reville Coll., Univ. California, San Diego, La Jolla, CA 92093); McGinness, J. E.; Ishmael, D.; Bell, L. J. *J Stud Alcohol* 49(3): 240-246; 1979.

The effect of simulated alcoholism on the development of spontaneous mammary adenocarcinomas in female inbred C3H/St mice was studied. Immediately after weaning 15 animals received 12% ethanol and 15 animals received red wine (11.5% alcohol content) as the sole fluid. The total tumor incidence did not differ significantly between the control (82%) and 12%-ethanol (73%) groups, but the median latency time was 8 mo in the ethanol group as compared with 14.2 mo in the controls ($p < 0.001$). The growth rates of the tumors in the two groups were similar. The growth rate, and fluid and food intakes did not differ significantly between the control and 12%-ethanol groups.

In the wine group, 2/5 animals developed slowly growing tumors at 12 mo. All of the wine-exposed animals were underweight and in poor condition. There were no histologic abnormalities in the livers or in esophageal tissues of the tumor-free animals of any group. The mean serum prolactin concentration was significantly lower in the alcohol-exposed animals (23 nanograms (ng)/ml) than in controls (52 ng/ml) ($p < 0.001$). (15 refs)

79-4338 Carcinogenic Bioassay of the Herbicide, 2,4,5-Trichlorophenoxyethanol (TCPE) with Different 2,3,7,8-Tetrachlorodibenzo- p-dioxin (Dioxin) Content in Swiss Mice. (Eng) Toth, K. (Res. Inst. Oncopathology, Budapest, Hungary); Sugar, J.; Somfai-Relle, S.; Bence, J. *Prog Biochem Pharmacol* 14: 82-93; 1978.

The carcinogenic effects of the herbicide 2,4,5-trichlorophenoxyethanol (TCPE) and its contaminant dioxin were studied in male and female outbred Swiss-H/Riop mice. The acute po LD₅₀ for TCPE contaminated with 0.1 ppm dioxin was 1,320 mg/kg, and the max tolerable dose was 70 mg/kg. In male mice given 70 mg/kg TCPE, the incidence of liver tumors at the end of the second year of the experiment was twice that in controls. The tumor frequency was not affected by increased levels of dioxin. A decreased TCPE dose resulted in a decreased liver tumor incidence, even when the concentration of dioxin was increased. Cirrhosis never preceded the development of tumors. The liver tumors were classified histologically as hepatomas with eosinophilic globular bodies and no cellular atypia or metastasis; hepatomas without inclusions; and trabecular-type hepatocellular carcinomas that were able to metastasize. The histologic pattern was not related to drug administration. Mice treated with dioxin (10⁻³ mg/kg) alone developed postnecrotic liver cirrhosis or focal necrosis in some cases, but the tumor incidence was not elevated above control levels. The use of TCPE as a herbicide in accordance with factory regulations and the consumption of TCPE-treated products does not appear to pose a carcinogenic risk for the consumer. (12 refs)

79-4339 Comparative Evaluation of the Frequency of Chromosomal Aberrations and the SCE Numbers in Peripheral Lymphocytes of Workers Occupationally Exposed to Vinyl Chloride Monomer. (Eng) Kucerova, M. (Pediatric Dept. Postgraduate Medical Inst., Inst. Hygiene and Epidemiology, Prague, Srobarova 48, Czechoslovakia); Polivkova, Z.; Batora, J. *Mutat Res* 67(1): 97-100; 1979.

Three blood samples from each of nine workers occupationally exposed for 10-27 yr to vinyl chloride monomer (VCM: mean annual dose 50-20-150 ppm) were analyzed for chromosome aberrations. The third blood sample was

also analyzed for sister-chromatid exchanges (SCE's). The frequency of chromosome aberrations over a 2-yr period was nonhomogeneous, ranging from 0%-11% aberrant cells. Chromatid and chromosome breaks were detected generally; chromatid and chromosome exchanges occurred only sporadically. The SCE frequency was more homogeneous, and it was significantly higher in the cells of VCM workers than in those of controls, ranging from 9.56 to 17.50 SCE's/cell. Smoking and other habits appeared to have no effect on the frequency of any chromosome changes. It is concluded that the routine and SCE cytogenetic analyses are equally suitable for determining high-dose VCM mutagenicity in vivo. The sensitivities of the two methods seem to be the same. (14 refs)

- 79-4340 Formation and Inactivation of a Reactive Metabolite of Vinyl Chloride (Meeting Abstract). (Fre) Pessayre, D. (Unite de Recherches de Physiopathologie Hepatique, INSERM, Hopital Beaujon, F92110 Clichy, France); Wandscheer, J. C.; Artigou, J. Y.; Descatoire, V.; Degott, C.; Benhamou, J. P. *Gastroenterol Clin Biol* 3(1): 100; 1979 (no refs)

- 79-4341 Determination of Vinyl Chloride in Polymer Materials, Model Media and Food Products. (Rus) Tarasova, N. A. (Technological Inst. Meat and Dairy Industry, Moscow, USSR); Kataeva, S. E. *Gig Sanit* (3): 48-50; 1979.

Gas chromatography was used to determine vinyl chloride levels in plastic packages and bottles and in samples of cheese that were packaged in polyvinyl chloride. The use of a vapor-air phase in the chromatographic procedure increased the reliability of the assay by 3%-10%. (1 ref)

- 79-4342 Mutagenicity of Inhalation Anaesthetics: Trichloroethylene, Divinyl Ether, Nitrous Oxide and Cyclopropane. (Eng) Baden, J. M. (Anesthesiology Service--112A, Veterans Admin. Hosp., 3801 Miranda Ave., Palo Alto, CA 94304); Kelley, M.; Mazze, R. I.; Simmon, V. F. *Br J Anaesth* 51(5): 417-421; 1979.

The mutagenic potential of the volatile anesthetics trichloroethylene and divinyl ether and the gaseous anesthetics nitrous oxide and cyclopropane was assessed in vitro by microbial assay employing two histidine-dependent strains of *Salmonella typhimurium*, TA1535 and TA100. Various concentrations of the agents were incubated with bacteria in the presence or absence of an enzyme system prepared from enzyme-induced (500 mg/kg Aroclor 1254, ip) Sprague-Dawley rat liver. Nitrous oxide and cyclopropane were not mutagenic, whereas divinyl ether gave a strongly positive response. The results for

trichloroethylene were equivocal. These and previous studies with the *Salmonella* system, together with mutagenicity studies using different test systems, indicate that modern inhalation anesthetic agents are unlikely to be mutagenic. (12 refs)

- 79-4343 Flame Retardants in Textiles. Another Hazard (Letter to Editor)? (Ger) Habs, M. (Institut für Toxikologie, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany). *Munch Med Wochenschr* 121(19): 644-645; 1979.

Tris-(2,3-dibromopropyl)phosphate (Tris), a flame retardant for textiles and a structural analog of 1,2-dibromomethane, a known carcinogen, showed mutagenic activity in *Salmonella typhimurium*. After po administration to mice and rats for 2 yr, Tris induced malignant tumors, mainly in the kidney. Therefore, it must be considered a potential carcinogen in humans as well. Although there has been no increase in cancer incidence among exposed workers, this finding cannot be considered evidence of a lack of a carcinogenic effect of Tris in humans in view of the long latent period for cancer in general. (no refs)

- 79-4344 S-Vinyl Homocysteine, an Analog of Ethionine That Is Highly Mutagenic for *S. typhimurium* TA100. (Eng) Leopold, W. R. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Center, Madison, WI 53706); Miller, J. A.; Miller, E. C. *Biochem Biophys Res Commun* 88(2): 395-401; 1979.

The recent finding that vinyl carbamate had a much higher carcinogenicity than ethyl carbamate prompted the synthesis of S-vinyl homocysteine (vinthionine) as a possible proximate carcinogenic metabolite of ethionine. Unlike ethionine, vinthionine and its N-acetyl methyl ester were directly mutagenic for *Salmonella typhimurium* TA100; they induced 15-20 and 0.2 revertants/nanomole, respectively. Liver Microsome-cytosol preparations from Aroclor 1254-treated Fischer rats increased the mutagenicity of the amide ester derivative but not that of vinthionine. Methionine inhibited the mutagenic action of vinthionine, but other inhibitors of ethionine carcinogenesis did not. Neither ethionine analog was mutagenic for *S. typhimurium* TA98. Neither vinthionine nor ethionine significantly increased the number of A/JAX mice that developed lung adenomas or the av number of lung adenomas per mouse. (27 refs)

- 79-4345 The Development of a 'Microtitre' Fluctuation Test for the Detection of Indirect Mutagens, and Its Use in the Evaluation of Mixed Enzyme Induction of the Liver. (Eng) Gatehouse, D. G. (Pathology Div.,

Glaxo-Allenburys Ltd., Priory St., Ware, Herts, England); Delow, G. F. *Mutat Res* 60(3): 239-252; 1979.

The 'Microtitre' fluctuation test was adapted for the detection of indirect mutagens through the incorporation of an S9 mix activation system. The technique was used to evaluate mixed enzyme induction using a combination of phenobarbital (PB) and β -naphthoflavone (BNF: benzoflavone) as a substitute for Aroclor-1254. The postmitochondrial preparations from rats induced with PB + BNF were as effective as those derived from Aroclor-induced animals in metabolism activation. This combination would therefore provide a useful alternative to Aroclor-1254 for routine screening. The level of S9 mix in the metabolism system greatly affected the quantitative mutagenic response. This response varied considerably from chemical to chemical and underlined the need for such preliminary investigations in routine screening. (37 refs)

- 79-4346 **Functionally Impaired tRNA from Ethionine Treated Rats as Detected in Injected *Xenopus* Oocytes.** (Eng) Ginzburg, I. (Dept. Neurobiology, Weizmann Inst. Science, Rehovot, Israel); Cornelis, P.; Giveon, D.; Littauer, U. Z. *Nucleic Acids Res* 6(2): 657-672; 1979.

The effect of DL-ethionine (EN) treatment on rat liver transfer RNA (tRNA) was assayed in microinjected *Xenopus laevis* oocytes and in a cell-free wheat germ system. tRNA was prepared from the livers of female Wistar rats injected with EN (25 mg/100/day ip for 3 or 5 days). tRNA from animals injected for 3 days was more undermethylated than tRNA from animals treated for 5 days in experiments using oocyte extracts as a source of tRNA methylases. tRNA from EN-injected rats showed a decreased level of phenylalanine aminoacylation, an increased level of histidine aminoacylation, and no difference in leucine aminoacylation compared with tRNA from untreated rats. tRNA from EN-exposed animals showed an initially faster methionine aminoacylation rate than tRNA from control animals in the in vivo oocyte assay. With more prolonged incubation periods, however, the methionyl-tRNA from EN-treated rats was deacylated at an accelerated rate, but the level of normal methionyl-tRNA remained almost constant. In the in vitro assay, there were no differences in aminoacylation capacity between normal tRNA and tRNA from EN-treated animals. The participation of aminoacyl-tRNA in protein synthesis was also tested in the presence of exogenous globin messenger RNA (mRNA), myeloma mRNA, or carnation mottle virus RNA. In both assay systems, a decreased incorporation of amino acids into proteins was observed when tRNA from EN-treated rats was used compared with untreated rats. These results demonstrate that EN severely impairs the aminoacylation capacity of tRNA and its ability to sustain protein synthesis. (36 refs)

- 79-4347 **Transplacental Carcinogenicity of p-Hydroxyphenyllactic Acid.** (Rus) Zharova, E. I. (Lab. Systemic Blood Diseases, Cancer Res. Center, Moscow, USSR); Sergeeva, T. I.; Makhlova, N. V.; Romanenko, V. I.; Chitiridi, N. G.; Raushenbakh, M. O. *Biull Eksp Biol Med* 87(1): 39-41; 1979.

The transplacental carcinogenicity of p-hydroxyphenyllactic acid (HPLA) was studied in CC57BR and C57BL mice. Pregnant females were inoculated sc with 5 mg HPLA/day either during the first 10 days of pregnancy (total dose, 50 mg) or during the last week of pregnancy (total dose, 20-30 mg). The progeny were followed for 22 mo. Of 160 CC57BR mice, 141 developed tumors (compared with 10/26 controls). There were 101 hemoblastoses (85 lymphoid leukemias), 62 adenomas, 9 lung carcinomas, 2 bronchial papillomas, 37 hepatomas, 22 hemangiomas (13 in the liver), 16 papillomas of the urinary bladder, 9 carcinomas of the urinary bladder, 1 myosarcoma, 3 sarcomas, and 1 unspecified tumor. Of 74 C57BL mice, 58 developed tumors (compared with 4/29 controls). There were 42 lympholeukemias, 12 lymphosarcomas, 5 lung adenomas, 2 bronchial papillomas, 4 hepatomas, 3 papillomas of the urinary bladder, 1 myosarcoma, 1 sarcoma, and 3 unspecified tumors. (13 refs)

- 79-4348 **Chemical and Toxicological Studies on Bracken Fern, *Pteridium aquilinum* var. *latiusculum*. III. Further Characterization of Pterosins and Pterosides, Sesquiterpenes and Glucosides Having 1-Indanone Skeleton, from the Rhizomes.** (Eng) Kuroyanagi, M. (Natl. Inst. Hygienic Sciences, Kamiyoga-1-chome, Setagaya-ku, Tokyo, Japan); Fukuoka, M.; Yoshihira, K.; Natori, S. *Chem Pharm Bull (Tokyo)* 27(3): 592-601; 1979.

Four pterosins (sesquiterpene compounds having a 1-indanone nucleus) and nine pterosides (glucoside compounds) were isolated from the rhizomes of bracken fern, *Pteridium aquilinum* var. *latiusculum*. The stereochemistry of the compounds was elucidated by chemical and physical methods. (14 refs)

- 79-4349 **Correlation of Fluorescence Intensity and Carcinogenic Potency of Synthetic and Natural Petroleum in Mouse Skin.** (Eng) Holland, J. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Whitaker, M. S.; Wesley, J. W. *Am Ind Hyg Assoc J* 40(6): 496-503; 1979.

After standardized topical exposure to male C3Hf/Bd mouse skin synthetic and natural petroleum, the penetration, persistence, and carcinogenicity of the fluorescent components was quantitated. Penetration was extremely rapid, fluorescence being detectable in the sebaceous

glands within 5 min. The degree of initial fluorescence was positively correlated with persistence, and in some cases fluorescence was still detectable after 14 days. The petroleum showed marked differences in their capacity to induce necrotizing and inflammatory dermatotoxic changes, and this capacity was negatively correlated with carcinogenic efficiency. A composite blend of natural petroleum showed a virtual lack of overt effects on mouse skin. Fluorescence intensity measured in sebaceous glands or in vitro was not well correlated with carcinogenicity and there was no simple relationship between in vivo and in vitro fluorescence. (6 refs)

- 79-4350 Deposition of Gasoline in Tissues and Organs of Pregnant Rubber Industry Workers.** (Rus) Lipovskii, S. M. (Inst. Obstetrics and Gynecology, Leningrad, USSR); Tomaeva, L. V.; Varfolomeev, D. I.; Fedoseev, Ia. E.; Karganova, E. V. *Gig Tr Prof Zabol* (3): 25-28; 1979.

Serum gasoline levels were determined in 85 pregnant female workers of the rubber industry. The women were 20-40 yr old, and they had been occupationally exposed to gasoline for 1-22 yr (concentration of gasoline vapors was 300 mg/m³). The women were divided into two groups: Group 1 consisted of 46 women who underwent abortion and Group 2 consisted of 39 women who were seen at the end of pregnancy. Gasoline levels in the fetal tissues were significantly greater than those in the blood of Group 1 women (0.00329 and 0.00127 mg/ml, respectively). Correspondingly, in Group 2, gasoline levels in blood samples taken from the umbilical cord were significantly greater than those in samples taken from the maternal elbow vein (0.0035 and 0.0025 mg/ml, respectively). To verify these findings that gasoline levels in embryo tissues exceed those in the mother, female Wistar rats were exposed to gasoline vapor (300 mg/m³) for 48 days. They were mated on days 25-30 of exposure and sacrificed after termination of the exposure. The av level of gasoline in the fetus was 0.00489 mg/g, compared with 0.00433 mg/g in the placenta, 0.00389 mg/g in the uterus, 0.00287 mg/g in the maternal brain tissue, and 0.00207 mg/ml in the maternal blood. (7 refs)

- 79-4351 Formation of Carcinogenic N-Nitroso Compounds in Corn-Bread Inoculated with Fungi.** (Eng) Mingxin, L. (Cancer Inst., Acad. Medical Sciences, Lin Xian, Henan, Republic China); Shixin, L.; Chuan, J.; Mingyao, W.; Shujun, C.; Changlian, J. *Scientia Sinica* 22(4): 471-477; 1979.

The formation of carcinogenic N-nitroso compounds in corn bread inoculated with some common species of fungi from Lin Xian County in China was studied. Dimethylnitrosamine (DMNA), diethylnitrosamine (DENA), and methylbenzyl nitrosamine (MBNA) were formed in corn bread inoculated with *Fusarium*

moniliforme, and DENA and DMNA were formed in corn bread inoculated with *Aspergillus flavus*. DMNA and DENA were observed in experiments with *Aspergillus niger* and mixed fungi. The amounts of DMNA, DENA, and MBNA in the samples were 0.1-0.2 ppm. The following compounds were also identified in samples inoculated with *F. moniliforme*: tetramethylpyrazine, O-methoxyphenol, benzyl alcohol, m- or p-ethylphenol, nicotinic acetate, 2,4- or 3,4-dimethylacetylbenzene, 3-methylbutanol, and 4-methyl-p-quinone. (7 refs)

- 79-4352 Sister Chromatid Exchanges Induced by Inhaled Anesthetics.** (Eng) White, A. E. (Dept. Anesthesia, Univ. California, San Francisco, CA 94143); Takehisa, S.; Eger, E. I.; Wolff, S.; Stevens, W. C. *Anesthesiology* 50(5): 426-430; 1979.

The sister-chromatid exchange (SCE) assay was used to examine the carcinogenic potential of 10 inhaled anesthetics. Chinese hamster ovary cells were exposed to the test chemicals for 1 hr in the presence of a metabolism-activating system (S-9 rat liver extract prepared from Aroclor 1254-treated male rats and fortified with NADPH). With this test system, exposure to a single dose of the max allowable concentration of nitrous oxide, diethyl ether, trichloroethylene, halothane, enflurane, isoflurane, methoxyflurane, or chloroform did not increase SCE values. Divinyl ether, fluroxene, and ethyl vinyl ether increased SCE values significantly above those of control cultures. These three anesthetics are older types that are not used at present. The results of this study of mammalian cells suggest that no currently used anesthetic is a mutagen-carcinogen. They also suggest that anesthetics containing a vinyl moiety may be mutagen-carcinogens. (19 refs)

- 79-4353 Nitrite Promotes Lymphoma Incidence in Rats.** (Eng) Newberne, P. M. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139). *Science* 204(4397): 1079-1081; 1979.

The effects of nitrite given in various concentrations in food or water on the lymphatic system were studied in Sprague-Dawley CRCD rats. The rats were exposed to sodium nitrite at concentrations of 0, 250, 500, 1,000 or 2,000 ppm and were killed at 6, 12, 18, 24, or (terminally) 26 mo. Malignant lymphomas were increased in all groups fed nitrite. The combined incidence of lymphomas in control groups was 5.4% compared with 10.2% in the combined nitrite-treated groups. The pattern of tumors suggests that the carcinogenic effect of nitrite was through a mechanism other than formation of nitrosamines. Nitrite is known to cause changes in the size and shape of RBC, which are readily detectable by the immune system. The animals' immune systems could have become depressed to maintain RBC life. It is speculated that this decreased ac-

tivity might eventually have led to overcompensation, to the observed proliferation of the immune tissues, and ultimately to tumors. (20 refs)

79-4354 Ultrastructural Study of the Preneoplastic Changes in the Colon Induced by 1,2-Dimethylhydrazine. (Fre) Delapierre, F. (Laboratoire de Cancerologie Experimentale et de Pathologie, Centre Henri Becquerel, 76038 Rouen Cedex, France); Laumonier, R.; Tayot, J. *Ann Anat Pathol (Paris)* 23(4/5): 309-332; 1978.

The induction of preneoplastic changes in the colon by 1,2-dimethylhydrazine (DMH: injections of 1.5-15 mg/kg/wk for 1-28 wk, total dose 15-420 mg/kg) was studied in 100 Wistar rats. Changes in mucin secretion were observed during week 2 and dysplastic lesions, from week 4 onward. The first tumors appeared during week 19. Ultrastructural study of the preneoplastic changes showed enlargement of the mitotic zones, secretory changes, and mutagenic effects. These changes were similar to the histochemical characteristics of the human fetal colon. The epithelial and mesenchymal changes were not accompanied by an inflammatory reaction. Early vascular changes, loss of the epithelial membrane, and dissociation of the basal membrane and its abnormal attachment to epithelial cells, collagen, and fibroblasts led to disorganization of the regenerating system of the colonic mucosa. This loss of equilibrium between the parenchyma and mesenchyma may provide conditions favorable for the excessive and disorganized growth of the mucosa. (32 refs)

79-4355 Effect of Age, Castration, and Pregnancy on 1,2-Dimethylhydrazine-induced Carcinogenesis in CBA Mice. (Rus) Turusov, V. S. (Lab. Carcinogenic Compounds, Cancer Res. Center, Moscow, USSR); Bazlova, L. S.; Krutovskikh, V. A. *Biull Eksp Biol Med* 87(5): 458-460; 1979.

The effect of factors such as age, castration, and pregnancy on 1,2-dimethylhydrazine (DMH)-induced carcinogenesis was studied in CBA mice. The animals were divided into four groups: Group 1 consisted of young (3-mo-old virgin) mice, Group 2 consisted of old (12- to 13-mo-old) mice, Group 3 consisted of mice subjected to bilateral ovariectomy 2 wk prior to initiation of DMH, treatment, and Group 4 consisted of mice that had two to six pregnancies during DMH administration. DMH was administered sc in a dose of 8 mg/kg/wk for 30 wk. Group 4 mice had a significantly lower incidence of uterine sarcomas (10.3%, compared with 48.3% in Group 1, 45.6% in Group 2 and 41.4% in Group 3). However, pregnancy had no effect on the incidence of tumors at other sites. The incidence of tumors of the anal region was 79.3% in Group 1, 74% in Group 2, 75.8% in Group 3, and 86.2% in Group 4. The

incidence of liver tumors was 38% in Group 1, 34.7% in Group 2, 48.2% in Group 3, and 51.7% in Group 4. The latency time for the uterine sarcomas and anal tumors was shorter in Group 2 mice than in Group 1 mice. Castration had no effect on tumor latency or incidence. (12 refs)

79-4356 Changes in the Adrenal Cortex During Experimental Carcinogenesis in the Rat Intestine. (Rus) Faustov, L. A. (Dept. Pathoanatomy, Medical Inst., Kursk, USSR). *Arkiv Patol* 41(3): 25-29; 1979.

The morphological and histochemical changes that occur in the adrenal cortex during 1,2-dimethylhydrazine (DMH)-induced carcinogenesis were studied in rats. The rats received weekly sc injections of DMH at doses of 21 or 40 mg/kg, and they were sacrificed at 1-mo intervals within 6 mo of the initiation of the experiment. The intestinal carcinogenesis could be divided into three periods: a latent period with a max duration of 3 mo after 40 mg/kg and 5 mo after 21 mg/kg; a period of tumor growth within the intestinal mucosa; and a tumor dissemination period. The latent period was associated with a decrease in adrenal wt (26 mg/100 g after 21 mg/kg and 25 mg/100 g after 40 mg/kg, compared with 30 mg/100 g in controls), a narrowing of the cortex zone (734 μ m in males and 747 μ m in females after 21 mg/kg, 755 μ m in males and 762 μ m in females after 40 mg/kg, compared with 825 and 851 μ m in controls), and a decrease in the level of ascorbic acid in all zones of the cortex. The period of tumor growth was associated with an increase in adrenal wt (34 mg in males and 34 mg in females after 21 mg/kg, 34 and 35 mg after 40 mg/kg), a widening of the cortex zone (926 mm in males and 888 in females after 21 mg/kg, 876 and 896 mm after 40 mg/kg) and a homogeneous distribution of ascorbic acid in the cytoplasm. The dissemination period was associated with more pronounced hypertrophic changes in the adrenal cortex: the wt of the adrenals increased to 38-40 mg after 21 mg/kg and 38-39 mg after 40 mg/kg, and the width of the cortex zone increased to 1,033-1,039 mm and 948-1,101 mm, respectively. (13 refs)

79-4357 Diet, Liver Function and Dimethylhydrazine-induced Gastrointestinal Tumours in Male Wistar Rats. (Eng) Castleden, W. M. (Univ. Western Australia, Nedlands, Western Australia 6009, Australia); Shilkin, K. B. *Br J Cancer* 39(6): 731-739; 1979.

The relationship between hepatocellular injury and gastrointestinal tumor development was investigated by comparing the effect of an all liquid diet with the standard diet in male Wistar rats treated with 1,2-dimethylhydrazine (DMH). Rats fed a normal laboratory pelleted diet, when treated sc, with DMH 10 mg/kg/wk survived the 24-wk experiment, showed no signs of chemical toxicity or macroscopic liver damage, and developed mainly large-

bowel tumors. Conversely, rats treated with 20 mg/kg/wk DMH did not survive the full term of the experiment and developed ascites, pleural effusions and nodular livers. They also developed more small-bowel tumors than large-bowel tumors. The relationship between the predominant site of tumor development and dosage of DMH was highly significant ($p < 0.001$). Rats fed with an all-liquid diet (Vivonex) and treated with 20 mg/kg/wk DMH behaved quite differently both in terms of survival and site of tumor development. These rats survived the full term of the experiment, showed no signs of chemical toxicity, experienced minimal liver damage and developed predominantly large-bowel tumors. The protection afforded by the all-liquid diet against DMH toxicity and small-bowel tumor induction was statistically highly significant. A series of blood tests with special reference to liver function confirmed the highly significant degree of protection against liver damage afforded by the all-liquid diet. Sections of liver from treated rats were examined, and a simple pathological scoring system was devised that showed a highly significant difference in liver histology between standard diet and liquid-diet rats treated with 20 mg/kg/wk DMH. The results strongly suggest an association between severity of liver damage from DMH and the subsequent development of small-bowel tumors. The all-liquid diet protected rats from liver damage, and these rats developed significantly fewer small bowel tumors. (19 refs)

79-4358 Cytotoxicity of *p*-Chloroamphetamine in Dimethylhydrazine-induced Carcinomata of Rat Colon. (Eng) Tutton, P. J. (Dept. Anatomy, Monash Univ., Clayton, Victoria 3168, Australia); Barkla, D.H. *Cancer Chemother Pharmacol* 2(2): 137-138; 1979.

Significant increases in the percentages of necrotic cells were seen in dimethylhydrazine (DMH)-induced rat colon tumors examined 24-48 hr following the sc injection of the serotonin-related compound *p*-chloroamphetamine (1 or 10 mg/kg), in comparison with DMH-induced tumors from controls. (14 refs)

79-4359 Protective Effect of Oral *Salmonella enteritidis* 11RX Infection Against Colon Tumor Induction by 1,2-Dimethylhydrazine in Mice. (Eng) Ashman, L. K. (Dept. Microbiology and Immunology, Univ. Adelaide, G.P.O. Box 498, Adelaide, South Australia 5001); Cook, M. G.; Kotlarski, L. *Cancer Res* 39(7): 2768-2771; 1979.

Infection of mice with *Salmonella enteritidis* 11RX has been shown previously to cause nonspecific immune stimulation and, consequently, resistance to subsequent challenge with a variety of transplantable tumors. The present study examined the effect of infection with this organism in a chemical carcinogenesis system. Colonic

tumors were induced in LACA and BALB/c x C57BL/6J F₁ mice by weekly sc injection of 1,2-dimethylhydrazine (15 mg/kg) for 28 wk. Infection of mice po with live *S. enteritidis* 11RX at 8-wk intervals during 1,2-dimethylhydrazine administration protected both strains against colon tumorigenesis. Significantly fewer infected than control BALB/c x C57BL/6J F₁ mice had colonic tumors (adenomas, adenocarcinomas) at or before termination of the experiment (34 or 40 wk) ($p < 0.001$ in all cases). Comparable results were obtained with both male and female mice. The difference in tumor incidence between control and infected LACA mice was not statistically significant. However, the number and the size of the lesions were greater in the control mice ($p < 0.02$). Although it has not been proven that the protective effect is mediated by the immune system, the results are consistent with the operation of a macrophage-mediated surveillance system. It is suggested that high standards of hygiene and, consequently, a low frequency of enteric infections contribute to the high incidence of colon cancer in developed countries. (28 refs)

79-4360 Mutagenicity of 7-Iodo and 7-Fluoro Derivatives of N-Hydroxy and N-Acetoxy-N-2-Acetylaminofluorene in the *Salmonella typhimurium* Assay. (Eng) Santella, R. M. (Inst. Cancer Res., Columbia Univ. Coll. Physicians and Surgeons, New York, NY); Fuchs, R. P.; Grunberger, D. *Mutat Res* 67(1): 85-87; 1979.

The mutagenicities of the 7-iodo and 7-fluoro derivatives of N-hydroxy-N-2-acetylaminofluorene (N-OH-AAF) and N-acetoxy-N-2-acetylaminofluorene (N-AcO-AAF) were compared in the *Salmonella typhimurium* assay using strain TA98. The assays were performed in the presence of an S9 mix prepared from 3-methylcholanthrene-induced C57BL/6N mice. There were no significant differences in the activation and mutagenicity of N-OH-AAF and its fluoro derivative (N-OH-AAFF), although the latter was slightly more toxic. The iodo derivative, N-OH-AAIF, was more mutagenic than N-OH-AAFF and N-OH-AAF; at 40 nanomoles (nm)/plate, N-OH-AAIF produced 8,000 revertants/plate, whereas N-OH-AAF and N-OH-AAFF produced approx 3,000/plate. The similar activity of N-OH-AAFF and N-OH-AAF and the difference between this activity and that of N-OH-AAIF agree with the different effects of these compounds on the conformation of DNA. In the absence of S9, N-AcO-AAFF and N-AcO-AAIF were two to five times more mutagenic than N-AcO-AAF at concentrations < 15 and < 6 nm/plate, respectively. Above these concentrations, N-AcO-AAFF and N-AcO-AAIF were much more toxic than N-AcO-AAF, making their mutagenicity difficult to evaluate. (16 refs)

79-4361 Reactivity of Antibodies to Guanosine Modified by the Carcinogen N-Acetoxy-N-2-

acetylaminofluorene. (Eng) Guigues, M. (Centre de Biophysique Moléculaire, C.N.R.S., 1A, Avenue de la Recherche Scientifique, 45045 Orleans Cedex, France); Leng, M. *Nucleic Acids Res* 6(2): 733-744; 1979.

The interactions of antibodies to N-(guanosin-8-yl)acetylaminofluorene (Guo-AAF) and several ligands (DNA-AAF, modified nucleosides, and oligonucleotides) were studied by radioimmunoassay (RIA), UV absorption, and circular dichroism. The antibodies were elicited in rabbits by immunization with a bovine serum albumin (BSA)-Guo-AAF conjugate, and they were purified by affinity chromatography on a Sepharose-Guo-AAF column. The antibodies were IgG as shown by ultracentrifugation, gel filtration, and immunodiffusion against goat anti-IgG serum and anti-IgM serum. In the RIA, the presence of a phosphate group [guanosine monophosphate (GMP)-AAF] and the removal of the acetyl group (GMP-AF decreased the affinity compared with Guo-AAF. The affinity of the antibodies toward modified DNA was independent of DNA conformation. The same results (6.7%) were found with double-stranded (ds) and single-stranded (ss) DNA-AAF. Therefore, the geometry of the regions with covalently bound AAF residues in ds DNA is identical to that of the regions with covalently bound AAF residues in ss DNA. Antibodies to Guo-AAF have much less affinity for DNA-AAF than for Guo-AAF. This may be due to the stacking of AAF residues with adjacent bases. The difference absorption spectra and the circular dichroism spectra indicate that the antibodies to Guo-AAF and DNA-AAF interact with the AAF residues. Thus, the AAF residues are accessible to the antibodies even in ds DNA-AAF. (20 refs)

79-4362 The Sensitivity and Heterogeneity of Histochemical Markers for Altered Foci Involved in Liver Carcinogenesis. (Eng) Hirota, N. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595); Williams, G. M. *Am J Pathol* 95(2): 317-328; 1979.

Resistance to iron accumulation during hepatocarcinogenesis was studied in male Fischer rats fed 0.02% N-2-fluorenylacetamide (FAA) for 13 wk. Iron loading was induced by the simultaneous feeding of 0.08% 8-hydroxyquinoline + 2.9% ferrous gluconate or by six sc injections of iron dextran (0.1 ml/g) administered 2 wk prior to sacrifice. Carcinogen-induced liver tumors were typical trabecular hepatocarcinomas and were resistant to iron accumulation. Iron-resistant foci were also identified in FAA-treated rats given parenteral or dietary iron. All iron-resistant foci exhibited a positive reaction for gamma-glutamyl transpeptidase or were deficient in ATPase or glucose-6-phosphatase. The enzyme abnormalities that were present in individual iron-resistant foci were generally uniform throughout each focus. However, more lesions were detected by resistance to iron accumulation than by

any one of the enzyme markers, since each of the enzyme abnormalities was not always present in a particular focus. It is concluded that resistance to iron accumulation is a more sensitive and reliable marker for carcinogen-induced altered foci than is any other histochemical property. (37 refs)

79-4363 Differential Effect of a Microsomal Deacetylase Inhibitor on the Mutagenicity of *Salmonella typhimurium* of 2-Acetylaminofluorene by Liver Homogenates of Guinea Pigs, Mice and Rats. (Eng) Okuno, S. (Div. Serology and Virology, Saitama Cancer Center Res. Inst., Ina-machi, Saitama 362, Japan); Takeishi, K.; Seno, T. *Cancer Lett* 6(1): 1-5; 1979.

The effect of paraoxon, a microsomal deacetylase inhibitor, on the microsome-mediated mutagenicity of 2-acetylaminofluorene (AAF) to *Salmonella typhimurium* TA98 was compared using liver homogenates from male Hartley guinea pigs, which are resistant to AAF carcinogenesis, and from male C57BL mice and male Sprague-Dawley rats, which are susceptible. Animals were induced by a combination of phenobarbital (PB) and 5,6-benzoflavone (BF). The mutagenic activity of AAF in the presence of liver homogenates from guinea pigs treated with PB + BF was inhibited almost completely by 10^{-4} M paraoxon. In contrast, approx 60% and 55% of the respective mutagenic activity activated by liver homogenates from treated mice and rats was resistant to 10^{-4} M paraoxon. Thus, a paraoxon-resistant enzyme activity was important in the mutagenic activation of AAF by liver homogenates of treated mice and rats, although deacetylase activity was also responsible for about half of the mutagenic activity. A probable candidate for the paraoxon-resistant enzyme is arylhydroxamic acid acyltransferase. In untreated guinea pigs, 90% of the mutagenic activity was inhibited by paraoxon, and in untreated mice and rats, 75% and 85% of the mutagenic activity was inhibited, respectively. These results suggest that microsomal deacetylase activity is significantly involved in the mutagenic activation of AAF by guinea pig liver homogenates but that an additional enzyme activity is also important in the activation of AAF by liver homogenates from induced mice or rats. (15 refs)

79-4364 Mutagenic Activity of Propoxur, Carbaryl, and Their Nitroso Derivatives: Reversion Induction in *Salmonella typhimurium*. (Pol) Jaszcuk, E. (Zaklad Toksykologii Sanitarnej, Panstwowy Zaklad Higieny, ul. Chocimska 24, 00-791 Warsaw, Poland); Syrowatka, T.; Cybulski, J. *Rocz Panstw Zakl Hig* 30(1): 81-88; 1979.

The mutagenic activity of propoxur, carbaryl, Nitrosopropoxur (NP), and nitrosocarbaryl (NC) (0.25-

1,000 µg/plate) was studied in *Salmonella typhimurium* strains TA1535, TA100, TA1537, TA1538, and TA98. Carbaryl and propoxur slightly decreased the revertant frequency in TA100 and TA98 and slightly increased this frequency in TA1537. These compounds did not display any bactericidal activity. NP and NC had a strong dose-dependent mutagenicity in strain TA1535—at 50-100 µg/plate, they increased the revertant frequency approx 100-fold compared with that in control cultures. Mutation induction was also observed in strains TA1537 and TA98, but the increase in revertant frequency was no greater than 10-fold. At NP and NC concentrations >100 µg/plate, the frequency of revertants was much lower than that in control cultures as a result of the bactericidal action of these compounds. Thus, the findings indicate that, unlike propoxur and carbaryl, NP and NC have pronounced mutagenic effects. (18 refs)

79-4365 Conformational Changes Induced in DNA by In Vitro Reaction with N-Hydroxy-N-2-aminofluorene. (Eng) Spodheim-Maurizot, M. (Centre de Biophysique Moléculaire, C.N.R.S., 1A, avenue de la Recherche Scientifique, 45045 Orleans Cedex, France); Saint-Ruf, G.; Leng, M. *Nucleic Acids Res* 6(4): 1683-1694; 1979.

The conformation of calf thymus DNA modified in vitro by the covalent binding of N-hydroxy-N-2-aminofluorene was investigated by determination of UV absorption, circular dichroism spectra and by radioimmunoassay (RIA). The results were in agreement with a model involving destabilized regions in the double helical DNA around the carcinogen molecule, although the aminofluorene (AF) residues were stacked next to the adjacent nucleotides. The RIA results showed that the AF residues were less accessible to antibodies in native DNA-AF than in denatured DNA-AF, which indicates that AF residues are partially buried in the interior of the DNA helix. This model was compared with a model for DNA modified by reaction with N-acetoxyacetylaminofluorene. (31 refs)

79-4366 Effects of Diamines on Ornithine Decarboxylase Activity in Control and Virally Transformed Mouse Fibroblasts. (Eng) Bethell, D. R. (Dept. Physiology and Specialized Cancer Res. Center, Milton S. Hershey Medical Center, 500 University Drive, Hershey, PA 17033); Pegg, A. E. *Biochem J* 180(1): 67-94; 1979.

The effects of spermidine and a series of $\alpha\omega$ -diamines on ornithine decarboxylase (OD) activity in 3T3 fibroblasts and simian virus 40 (SV40)-transformed 3T3 cells (SV-3T3) were studied. Putrescine, spermidine, a variety of other diamines, and aliphatic $\alpha\omega$ -diamines were able to prevent the increase in OD activity in serum-stimulated 3T3 cells

maintained in serum-free medium. Histamine had no inhibitory effect on the increased OD activity. The SV-3T3 cells required much higher concentrations of putrescine or spermidine for a decrease in OD activity similar to that seen in the 3T3 cells. The difference between 3T3 and SV-3T3 cells was not as evident when nonphysiological $\alpha\omega$ -diamines were added to serum-stimulated cells. The 3T3 cells were considerably more sensitive to putrescine than to propane-1,3-diamine, whereas the SV-3T3 cells responded almost equally to the two agents. The longer chain $\alpha\omega$ -diamines had similar effects at equivalent concentrations on both 3T3 and SV-3T3 cells. The decrease in OD activity in response to the various di- and polyamines was not due to metabolism of the added amines by the serum or to direct inhibition of enzyme activity by any amine present in the cell extracts. (46 refs)

79-4367 Synthesis of N-Nitrosamino Aldehydes, Metabolic Intermediates Possibly Involved in the Induction of Tumors in Rats by N-Butyl-N-(ω -hydroxyalkyl) nitrosamines. (Eng) Suzuki, E. (Tokyo Biochemical Res. Inst., Takada 3-41-8, Toshima-ku, Tokyo 171, Japan); Okada, M. *Chem Pharm Bull (Tokyo)* 27(2): 541-544; 1979.

Three N-nitrosamino aldehydes that are metabolic intermediates of the N-butyl-N-(ω -hydroxyalkyl) nitrosamines and may be involved in bladder tumor induction in rats were synthesized and tested for mutagenicity in an activated *Salmonella typhimurium* system. N-Butyl-N-(formylmethyl)nitrosamine (BFMN), N-butyl-N-(2-formylethyl)nitrosamine, and N-butyl-N-(3-formylpropyl)nitrosamine were synthesized and demonstrated to be relatively stable compounds. All three compounds were mutagenic to *S. typhimurium* strain TA1535 with metabolic activation. A spontaneous decomposition product(s) of BFMN, which has not yet been identified, showed very potent mutagenicity without metabolic activation. (13 refs)

79-4368 Neoplastic and Nonneoplastic Urinary Bladder Lesions Induced in Fischer 344 Rats and B6C3F1 Hybrid Mice by N-Nitrosodiphenylamine. (Eng) Cardy, R. H. (Chemical Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Lijinsky, W.; Hildebrandt, P. K. *Ecotoxicol Environ Saf* 3(1): 29-35; 1979.

The effects of N-nitrosodiphenylamine (NDPA, a retarder in the vulcanization of rubber and an important industrial N-nitroso compound) on the bladders of Fischer 344 rats and B6C3F₁ hybrid mice were studied in a 2-yr bioassay. The compound was incorporated into the diet at 2,000-4,000 ppm for the rats, 10,000-20,000 ppm for the male mice, and 5,000-10,000 ppm (later reduced to 1,000-4,000 ppm) for the female mice. NDPA significantly reduced the

survival of female mice, but it had no significant effects on the survival of rats or male mice. Male and female rats in the high dose groups showed a high incidence of bladder tumors ranging from transitional cell hyperplasia to transitional cell carcinomas, the latter developing in 90% of the females and 40% of the males. The lesions first appeared as foci of epithelial hyperplasia, with fibrous strands forming a connective tissue stroma as the hyperplasia increased. The degree of infiltration into deeper layers of the bladder was variable, and none of the tumors metastasized. In the mice, there was a high incidence of chronic submucosal inflammatory lesions of the bladder. The overall thickness of the submucosa was somewhat increased and two animals showed edema. Epithelial hyperplasias were usually focal. No tumors developed in the mice. (16 refs)

- 79-4369 Detection of Trace Amounts of Dimethylnitrosamine in a Modified Salmonella/Microsome Assay. (Eng) Guttenplan, J. B. (New York Univ. Dental Center, New York, NY 10010). *Mutat Res* 64(2): 91-94; 1979.

Mutagenesis induced in *Salmonella typhimurium* TA1530 by dimethylnitrosamine (DMN) at concentrations as low as 0.1 mM was readily detected in assays in which cells were grown exponentially, the pH was 6.0-6.3, mouse liver microsomes were added, and the bacteria were pretreated with low doses of DMN or N-methyl-N-nitrosourea. (23 refs)

- 79-4370 Factors Influencing Carcinogen Absorption In Vitro. (Eng) Capel, I. D. (Marie Curie Memorial Foundation, The Chart, Oxted, Surrey, RH8 0TL, England); Williams, D. C. *IRCS Med Sci (Cancer)* 7(4): 214; 1979.

Gastrointestinal absorption of the water-soluble carcinogen dimethylnitrosamine was significantly enhanced by alcohol, severe stress (maintenance at 4 C in a Boltzman cage for 3 hr), aspirin, 5-fluorouracil, and ether inhalation in male Sprague-Dawley rats. In contrast, absorption of the fat-soluble carcinogen benzo(a)pyrene from the gut was not affected significantly by any of the treatments. (4 refs)

- 79-4371 Methylation of DNA in Target and Non-target Organs of the Rat with Methylbenzyl nitrosamine and Dimethylnitrosamine. (Eng) Fong, L. Y. (Dept. Biochemistry, Faculty Medicine, Univ. Hong Kong, Kong Kong); Lin, H. J.; Lee, C. L. *Int J Cancer* 23(5): 679-682; 1979.

The sites of the labeling of DNA with ^{14}C -methyl groups from methylbenzyl nitrosamine (MBN) and dimethylnitro-

samine (DMN) were studied in esophageal epithelium and liver from male Sprague-Dawley rats. About 140-190 mg of tissue were incubated at 37 C with 13-15 μCi of test compound; the concentrations of MBN and DMN were 1.1 and 0.32 mM, respectively. The DNA was purified and hydrolyzed to free purines and apurinic acid. Methylation of liver DNA with MBN or DMN was nearly the same: about three methyl groups were incorporated for every 10^4 nucleotides. There were two methyl groups for every 10^3 nucleotides in MBN-labeled esophageal DNA, but with DMN, methylation was only 0.025 of this value. Apurinic acid and 7-methylguanine (7-MG) were extensively labeled in all four combinations of carcinogen and tissue. Esophageal DNA labeled with the organ-specific MBN contained higher proportions of O⁶-methylguanine (O-MG) than either DMN-labeled specimens or MBN-labeled liver DNA. Liver DNA labeled with DMN had a very similar proportion of O-MG, which was significantly higher than that found in DMN-labeled esophageal DNA or MBN-labeled liver DNA. Incubation of esophageal or liver tissue with their respective organ-specific carcinogens also resulted in higher proportions of 7-MG than that appearing in MBN-labeled liver DNA. Thus, O-MG and 7-MG showed a positive correlation with the carcinogenic potential of the alkylating agent in the particular organ. These results demonstrate the feasibility of studying methylation patterns in DNA from tissue slices incubated in vitro. (24 refs)

- 79-4372 Structural Defects in Rat Liver Deoxyribonucleic Acid: Endogenous Single-stranded Regions in Comparison with Damage Induced In Vivo by a Carcinogen. (Eng) Stewart, B. W. (Sch. Pathology, Univ. New South Wales, P.O. Box 1, Kensington, N.S.W. 2033, Australia); Huang, P. H.; Brian, M. *J. Biochem J* 179(2): 341-352; 1979.

Comparative analyses were made of rat liver DNA preparations obtained by stepwise caffeine or NaCl elution from benzoylated-2-(diethylamino) ethanol cellulose in an attempt to define structural change induced by methylation. Female Wistar rats were subjected to partial hepatectomy and 23 hr after surgery received (^3H)thymidine (50 μCi , ip). Dimethylnitrosamine (DMN) (10 mg/kg, ip) was given following a minimum recovery period of 2 wk. DNA was isolated 4 hr after carcinogen administration. The results of rat liver binding to nitrocellulose indicated that the caffeine-eluted fraction exhibited some degree of single-strandedness. Denaturation kinetics indicated structural defects in the NaCl-eluted DNA from DMN-treated animals. These defects did not cause binding to benzoylated-DEAE cellulose and were not substrates for *Neurospora crassa* endonuclease. These changes may be attributed to alkylated bases in the DNA. The short interval required for complete denaturation of caffeine-eluted DNA was indicative of extensive disruption of structure compared with the respective control preparation. Only a

minor fraction of caffeine-eluted DNA was digested with endonuclease, which makes it unlikely that long tracts of single-stranded DNA constitute the principal structural defect. The data suggest that regions of local denaturation may occur at critical intervals within each DNA fragment. It is concluded that the use of benzoylated-DEAE-cellulose permits definition of degrees of structural damage in rat liver DNA isolated immediately after DMN administration. The structural defects detected in these experiments are probably the direct consequences of alkylation rather than subsequent repair processes. (51 refs)

- 79-4373 Metabolism of N-Nitroso-2-oxopropylpropylamine by Rat Liver: Formation of Products Resulting from Both Oxidation and Reduction.** (Eng) Park, K. K. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139); Archer, M. C. *Cancer Biochem Biophys* 3(1): 37-40; 1978.

The metabolism of N-nitroso-2-oxopropylpropylamine (NOPPA) by isolated liver preparations from phenobarbital-pretreated (1 g/liter drinking water) male Sprague-Dawley rats was studied. Incubation of NOPPA (69 micromoles) with three different fractions of liver homogenate (9,000 x g supernatant, 105,000 x g pellet, and 105,000 x g supernatant) yielded N-nitroso-2-hydroxypropylpropylamine (NHPPA) as the major product (11%, 5%, and 7%, respectively). Reduction of NOPPA was carried out by both soluble and microsomal enzymes. Alpha oxidation of NOPPA to yield propanol or propionaldehyde (PA) was confined to the 105,000 x g pellet. NHPPA formation was not inhibited by incubation of the 105,000 x g pellet or the 105,000 x g supernatant with SKF-252A (7.14 nM), a potent inhibitor of a number of hepatic drug-metabolizing enzymes. Incubation of NOPPA with boiled 9,000 x g supernatant yielded no NHPPA. NOPPA and NHPPA appeared to be readily interconvertible. With both the 9,000 x g supernatant and the 105,000 x g pellet fractions, NOPPA yielded n-propanol and isopropanol; they were formed via decomposition of the nitrosamine in the presence of water as a nucleophile. Incubation of NOPPA with boiled microsomes or microsomes containing SKF-525A (7.14 nM) gave neither PA nor propanols. The yield of PA was more than double the yield of n-propanol and isopropanol combined, indicating that microsomal oxidation at the α -carbon atom remote from the oxo group was apparently favored. (6 refs)

- 79-4374 Effects of a Choline-devoid Diet on the Emergence of γ -Glutamyltranspeptidase-positive Foci in the Liver of Carcinogen-treated Rats.** (Eng) Shinozuka, H. (Dept. Pathology, Univ. Pittsburgh Sch.

Medicine, Pittsburgh, PA 15261); Sells, M. A.; Katyal, S. L.; Sell, S.; Lombardi, B. *Cancer Res* 39(7): 2515-2521; 1979.

The effect of a choline-deficient (CD) diet on the induction of hepatocytes with γ -glutamyl transpeptidase (GGT)-positive foci was studied in Sprague-Dawley rats initiated with diethylnitrosamine (DEN). After a single ip injection of DEN (30 or 150 mg/kg) into male rats, a CD diet containing 0.02% acetylaminofluorene (AAF) resulted in many more foci of GGT-positive hepatocytes than did a choline-supplemented (CS) diet containing AAF. On the other hand, an approx equal number of foci developed in rats given a single DEN injection while on a plain CD or a plain CS diet and then subjected to a partial hepatectomy while being fed a CS diet containing AAF. Immunofluorescence staining of liver sections showed that most α -fetoprotein (AFP)-positive cells were oval and/or intermediate cells scattered in the parenchyma and in the vicinity of the foci of GGT-positive hepatocytes. Occasional cells were seen in newly formed ductules. On the other hand, foci of GGT-positive hepatocytes were consistently AFP negative. Oval and/or intermediate cells, as well as hepatocytes in the foci, stained positively for albumin. Plasma GGT levels correlated positively with the number of GGT-positive foci in the liver, whereas serum AFP concentrations showed no correlation. The results indicate that a CD diet promotes the evolution of initiated cells to foci of altered GGT-positive hepatocytes but has no effect on initiation of liver cells by DEN. The lack of AFP in the cells of the foci suggests the possibility that more than one pathway exists in the development of hepatocellular carcinomas. (32 refs)

- 79-4375 Effect of Simultaneous Administration of DDT on the Toxicity of Dimethylnitrosamine in Rats in a Long-Term Experiment.** (Pol) Syrowatka, T. (Zaklad Toksykologii Sanitarnej, Panstwowy Zaklad Higieny, ul. Chocimska 24, 00-791 Warsaw, Poland); Tyrkiel, E.; Nazarewicz, T. *Rocz Panstw Zakl Hig* 30(1): 67-79; 1979.

The effect of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT: 50 mg/kg food for 35-50 days) on the general toxic and carcinogenic effects of dimethylnitrosamine (DMNA: 25-75 mg/liter drinking water for 30-35 days) was studied in male Wistar rats in 50- and 70-wk experiments. DDT increased the extent of liver damage, increased tumor frequency, reduced the tumor latent period somewhat, and lowered the minimum tumor inducing dose of DMNA, but it did not induce tumors when given alone. At 25 mg/liter, DMNA induced liver tumors (adenomas, carcinomas, and fibrosarcomas) only when administered with DDT. DDT had a similar enhancing effect on the histological and neoplastic changes induced by DMNA in the kidney. (22 refs)

79-4376 A Positive Correlation Between Declining Immune Competence and Early Mortality Associated with Diethylnitrosamine Carcinogenesis in Aging Mice. (Eng) Perkins, E. H. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Clapp, N. K.; Cacheiro, L. H.; Glover, P. L.; Klima, W. C. *Mech Ageing Dev* 10(3/4): 225-232; 1979.

The effect of age at time of treatment on diethylnitrosamine (DEN)-induced carcinogenesis and immune competence was assessed in female BALB/c mice. The mice (2.5, 9.5, and 17 mo old) received DEN in the drinking water for 11 wk. The cumulative doses, based on water consumption and body wt, for the three age groups were 408, 303, and 281 mg/kg, respectively. DEN induced squamous tumors of the forestomach, vascular tumors of the liver, and lung adenomas. Median times of death in the three age groups were 193, 168, and 125 days, respectively. Immune competence, as measured by cell-mediated and humoral immune parameters within 1 wk after completion of DEN treatment, was not significantly different among treated and age-matched untreated controls. However, a significant age-related decline in immune competence was detected in DEN-treated and untreated mice. These data demonstrate a direct and positive correlation between the natural age-related decrease in immune competence and cancer-induced advanced mortality. (14 refs)

79-4377 Rapid Determination of Methylated Purines in DNA Treated with N-Methyl-N-Nitrosourea Using High-Performance Liquid Chromatography. (Eng) Thielmann, H. W. (Deutsches Krebsforschungszentrum, Institut für Biochemie, Im Neuenheimer Feld 280, 6900 Heidelberg, W. Germany). *Cancer Lett* 6(6): 311-317; 1979.

A method was developed for determining methylated purine bases in DNA treated in vitro with (³H)N-methyl-N-nitrosourea (MNU). The method combines reversed-phase high-performance liquid chromatography (HPLC) of methylated DNA after hydrolysis in dilute acid with the determination of radioactivity in the fractionated eluates. The peaks of the respective methylated purines were identified by internal standards. Quantitative separation of 3-methyl-adenine, 7-methyl-adenine, 3-methyl-guanine, 7-methyl-guanine, and O⁶-methyl-guanine was achieved using this procedure. Preliminary experiments indicate that the method is applicable to the detection and analysis of DNA modifications in tissue culture cells after (³H)MNU treatment. (12 refs)

79-4378 Cellular Heterogeneity in an Ethylnitrosourea-induced Glioma: Malignancy, Karyology and Other Properties of Tumour Cell Types. (Eng) Claisse, P.

J. (Dept. Cell Pathology, Sch. Pathology, Middlesex Hosp. Medical Sch., Riding House St., London W1P 7LD, England); Roscoe, J. P.; Lantos, P. L. *Br J Exp Pathol* 60(2): 209-224; 1979.

Two sublines (A10 and A15) of a cell line obtained from a glioma transplacentally induced by ethylnitrosourea in a BD-IX rat were characterized, along with their recently derived clones. A10 and A15 were distinguished in culture by their different morphologies, responses to dibutyryl cyclic AMP, inducibility of glycerol phosphate dehydrogenase, and growth in soft agar. They had different karyotypes, with distinctive numbers and arrangements of chromosomes. One cell type had an apparently normal diploid set of 42, and the other had 43 chromosomes. An additional chromosome 4 was identified in the latter by Giemsa banding. Translocations and other abnormalities involving this chromosome were observed consistently. Both cell types produced malignant, astrocytic tumors when injected into newborn syngeneic rats, but the tumors had different latent periods and morphological features. (37 refs)

79-4379 Differences in the Removal of N-Methyl-N-nitrosourea-methylated Products in DNase I-sensitive and -resistant Regions of Rat Brain DNA. (Eng) Cox, R. (Cancer Res. Lab., Veterans Admin. Hosp., Memphis, TN 38104). *Cancer Res* 39(7): 2675-2678; 1979.

The reaction of N-methyl-N-nitrosourea (MNU) with rat brain DNA was studied to determine the distribution of alkylated products and the difference in the removal of these products from DNase I-sensitive and -resistant regions of the DNA. Brain nuclei were isolated from male Sprague-Dawley rats injected iv with ³H-MNU (10 mg/kg) and then incubated with DNase I (5 µg/ml). Digested DNA was further hydrolyzed in 0.1 N HCl, and the alkylated products were identified by chromatography on a cation-exchange column. Resistant DNA was isolated, hydrolyzed, and again the alkylated products were determined. At 4 hr, the specific activity of each alkylated product in the sensitive regions of DNA was several times higher than in the resistant fraction. The rate of loss of the products was greater in the sensitive fractions than in the resistant fractions. O⁶-Methylguanine was removed from the sensitive regions for 3 days after MNU administration, but it was more stable in the resistant regions. These results suggest that DNase I-sensitive regions of the DNA are preferentially alkylated by MNU and that the alkylated products, including O⁶-methylguanine, are selectively removed from the DNase I-sensitive regions of the DNA. (19 refs)

79-4380 Biochemical Evidence of Cocarcinogenesis: Tumor Promoting Agent Enhances Methyl-

nitrosoarea Activation of Rat Guanylate Cyclase Activity. (Eng) Veseley, D. L. (Dept. Medicine, Univ. Arkansas Medical Sciences, Little Rock, AR 72201). *Res Commun Chem Pathol Pharmacol* 24(2): 329-338; 1979.

The effects of 12-O-tetradecanoylphorbol-13-acetate (TPA) alone and in combination with maximal and sub-maximal doses of methylnitrosoarea (MNU) on guanylate cyclase (GC) activity was studied using tissues from male Sprague-Dawley rats. TPA (1 μ M) alone caused a marked enhancement of liver, lung, colon, stomach, kidney, and epidermal GC activity. Increases in cyclic guanosine 3',5'-monophosphate accumulation secondary to GC activation were highly significant in all tissues. Increasing the concentration of TPA to 5 or 10 μ M caused no further enhancement of GC activity. TPA stimulated in an additive fashion MNU activation of hepatic GC activity when MNU was given in a submaximal stimulatory dose (1 μ M). The addition of TPA to a maximal stimulatory dose of MNU (10 μ M) caused no further increase in GC activity. The data suggest that at the cellular level a promoter is not absolutely necessary for the changes observed in cancer cells. However, promoters appear to be potentially able to contribute to the development of a cancer cell. (18 refs)

79-4381 New Neural Cell Lines Derived from Experimentally Induced Rat Tumors and from Human Neuroblastomas. (Eng) Herschman, H. R. (Sch. Medicine, Univ. California, Los Angeles, CA 90024); Seeger, R.; West, G.; Stahn, R.; Uki, J. *Natl Cancer Inst Monogr* (48): 355-357; 1978.

The development of several new cell lines from CNS tumors of rats and humans is described. Pregnant rats were treated with ethylnitrosoarea and nerve growth factor, which resulted in a high incidence of CNS tumors in the offspring. Of 22 clones derived from these tumors, 2 showed both neuronal [as indicated by the neuronal Na^+ action potential ionophore (API)] and glial [as indicated by the presence of cortisol-inducible glycerol phosphate dehydrogenase (GPDH)] properties. Several others showed either neuronal or glial properties, but not both. The rat cell lines did not show elevated tyrosine hydroxylase activity or acetylcholine levels relative to normal brain. Five cell lines were also developed from human neuroblastomas. Of these, 2 were adrenergic lines showing the Na^+ API, 1 was a cholinergic line with the Na^+ API, 1 was an inactive line with the Na^+ API, and 1 was a line that had neither the enzyme nor the Na^+ API. The lines were indistinguishable morphologically, but each was unique in terms of its isozyme characteristics and karyotype. All five lines are capable of growth in nude mice, and it is hoped that this system will be useful as a model in the development of specific chemotherapeutic regimens based on residual phenotypic biochemical properties of the cells. (5 refs)

79-4382 Tests of the Potential Carcinogenic Properties of Isobutylidenediurea. (Rus) Didenko, G. G. (All-Union Res. Inst. Toxicology Pesticides, Polymers and Plastics, Kiev, USSR); Gupalovich, T. D.; Petrovskaia, O. G. *Vopr Pitan* (2): 72-73; 1979.

The potential carcinogenicity of the synthetic food additive isobutylidenediurea (IBDU) was tested in random-bred rats and mice. The drug was given intragastrically, 2 times/wk for 20 wk, at max permissible doses (1,600 mg/kg for mice and 800 mg/kg for rats). The animals were followed for 92 wk. The incidence of tumors in mice treated with IBDU did not differ from the incidence of spontaneous tumors in controls (22.4% and 23.4%, respectively). The av latent period of tumor development in both treated and control mice was 13-15 mo. Among the tumors observed in treated and control mice were pulmonary adenomas (9.5% and 11.3%, respectively), mammary gland tumors (6.2% and 4.7%), leukemias (2.4% and 4.7%), pulmonary adenocarcinomas (1.2% and 1.7%), mesotheliomas (1.6% and 1.1%), and fibromas (0.8% and 0%). IBDU administration to rats also did not increase the incidence of tumors (7.3%, vs 7.9% in controls). These findings indicate that IBDU is not carcinogenic for mice and rats. (6 refs)

79-4383 Metabolism of the Liver Carcinogen N-Nitrosopyrrolidine by Rat Liver Microsomes. (Eng) Hecker, L. I. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701); Farrelly, J. G.; Smith, J. H.; Saavedra, J. E.; Lyon, P. A. *Cancer Res* 39(7): 2679-2686; 1979.

The metabolism of the hepatocellular carcinogen N-nitrosopyrrolidine (NO-PYR) by rat liver microsomes and postmicrosomal supernatant was studied. [2,5- ^{14}C]NO-PYR, which is totally extractable from aqueous soln with methylene chloride, was converted to radioactive nonmethylene chloride-extractable products by these fractions. The initial rate of conversion to nonmethylene chloride-extractable products followed simple Michaelis-Menten kinetics with an apparent K_m of 3.6×10^{-4} M NO-PYR. The major products of NO-PYR metabolism by rat liver microsomes and postmicrosomal supernatant were isolated and identified. One NO-PYR metabolite was 2-hydroxytetrahydrofuran, formed by α -hydroxylation by the microsomes. In the presence of postmicrosomal supernatant enzymes, this compound existed only as a transient intermediate that was rapidly converted to 1,4-butanediol or γ -hydroxybutyrate. These compounds may be cycled into general cellular metabolism, resulting in the production of CO_2 . Two minor metabolites were also found, but their identities were not established. (23 refs)

79-4384 Isolation of N-Nitroso-2-methylthiazolidine from a Cysteamine-acetaldehyde-sodium

Nitrite Model System. (Eng) Sakaguchi, M. (Ogawa and Co., Ltd., 6-32-9 Akabanenishi, Kita-ku, Tokyo, Japan); Shibamoto, T. *Agric Biol Chem* 43(3): 667-669; 1979.

N-nitroso compounds were synthesized using a cysteamine-acetaldehyde-sodium nitrite browning model system. One main product was N-nitroso-2-methylthiazolidine, which was formed from the reaction of 2-methylthiazolidine with nitrite; another was N-nitroso-thiazolidine. The data suggest that thiazolidines might potentially react with nitrite to produce nitrosamines during digestion. (11 refs)

79-4385 Mutagenicity of Metronidazole (Letter to Editor). (Eng) Hartley-Asp, B. (Aktiebolaget Leo Res. Labs., S-251 00 Helsingborg, Sweden). *Lancet* 1(8123): 981; 1979.

Evidence supporting the conclusion that metronidazole may be regarded as a safe drug for short-term treatment is presented. The dominant lethal test in mice and rats was negative, there was no increase in unscheduled DNA repair in human lymphocytes or fibroblasts, and there was no increase in the level of sister-chromatid exchanges or chromosome aberrations after in vitro treatment with metronidazole and its metabolites. In addition, 10-4,000 mg/kg metronidazole had no clastogenic effect in mice. (4 refs)

79-4386 Mutagenicity of Some Trichomonacides for *Salmonella typhimurium* (Ames' Test). (Ita) Tamaro, M. (Istituto di Microbiologia, Università degli Studi di Trieste, Trieste, Italy); Baraggino, E.; Pilotto, A. *Minerva Ginecol* 31(4): 263-272; 1979.

The mutagenicity of different trichomonacides was studied in the Ames' test using *Salmonella typhimurium*. Metronidazole, nimorazole, several nitrofur derivatives (nitrofurazone, nitrofuraxime, furazolidone, nifuratel, and nitrofurantoin), and two 8-oxyquinoline derivatives proved to be mutagenic. Myconazole, clotrimazole, phenylarsinic acid derivatives, phenylmercuric acid, benzalkonium chloride, polyvinyl pyrrolidone iodide, nonylphenoxypolyethoxyethanol, and mepartricine showed no mutagenic activity. (31 refs)

79-4387 Mutagenicity of Cyclic Nitrosamines in *Salmonella typhimurium*: Effect of Ring Size. (Eng) Rao, T. K. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Ramey, D. W.; Lijinsky, W.; Epler, J. L. *Mutat Res* 67(1): 21-26; 1979.

The mutagenicity of seven cyclic nitrosamines with different carcinogenic potencies was assayed in the *Salmonella*

histidine-reversion system using tester strains TA1535, TA1537, TA1538, TA98, and TA100. Mutagenicity in the pour-plate assay was compared with that in the liquid preincubation assay. Each of the smaller ring compounds [nitrosoazetidine (NAZ), nitrosopyrrolidine (NPY), and nitrosopiperidine (NPI)] exhibited a similar effect in both assays. NAZ was only slightly mutagenic in both assays; NPY and NPI were moderately strong mutagens, and they were three times more mutagenic in the liquid preincubation assay than in the plate assay. The larger ring compounds nitrosohexamethyleneimine, nitrosoheptamethyleneimine, and nitrosooctamethyleneimine, which are potent carcinogens, were strong mutagens in the plate assay, and they exhibited a 10-fold increase in mutagenicity when they were preincubated for 60 min before being added to the minimal medium plates. Nitrosodecamethyleneimine, another large ring compound, was a weaker mutagen than these three compounds, although it was stronger than NPI; it is a rather weak carcinogen. Except for NPI, a relatively potent carcinogen, the cyclic nitrosamines tested showed a fair correlation between their mutagenicity and carcinogenicity. Mutagenicity tended to increase with the number of ring carbon atoms. (19 refs)

79-4388 Effects of N-Nitrosopiperidine Substitutions on Mutagenicity in *Drosophila melanogaster*. (Eng) Nix, C. E. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Brewen, B.; Wilkerson, R.; Lijinsky, W.; Epler, J. L. *Mutat Res* 67(1): 27-38; 1979.

N-Nitrosopiperidine (NP) and several of its derivatives were fed to *Drosophila melanogaster* males over a wide concentration range, and the ability of these compounds to induce X-linked recessive lethals and chromosome loss was assessed. NP was effective in inducing lethals, as were its halogenated and methylated derivatives, with the exception of 2,6-dimethyl-NP. (Methyl substitutions at the alpha carbon atoms reduce or eliminate mutagenic activity.) Substitution of halogen groups on the piperidine ring enhanced mutagenicity. The 3,4-dichloro, 3-chloro, and 4-chloro derivatives were more effective mutagens for *Drosophila* than NP, with the 3-chloro compound being the most mutagenic. In contrast, substitutions with a hydroxyl, carboxyl, or keto group resulted in a loss of mutagenicity. None of the compounds tested increased the frequency of chromosome loss or breakage in mature sperm. (19 refs)

79-4389 Sister Chromatid Exchange Induction Resulting from Systemic, Topical, and Systemic-Topical Presentations of Carcinogens. (Eng) Shuler, C. F. (Clinical Genetics Div., Mental Retardation Program, Children's Hosp. Medical Center, Boston, MA 02115); Latt, S. A. *Cancer Res* 39(7): 2510-2514; 1979.

Chinese hamster cheek pouch mucosal cells were examined for in vivo sister chromatid exchange (SCE) formation resulting from exposure of the animals to carcinogens presented in three manners: systemic, topical, and combined systemic-topical. Systemic cyclophosphamide (5 or 10 mg/kg ip) increased SCE frequency from 4.8 to 9.9/cell. Topical application of 7,12-dimethylbenz(a)anthracene (0.5% in mineral oil, 0.1 ml) resulted in an SCE frequency of 11.5/cell, compared with 5.0/cell in animals treated only with mineral oil. Systemic administration of 8-methoxypsoralen (8-MOP) (0.5, 1, 2.5, or 5 mg/kg ip), followed by activation with topical near-UV light (3.75×10^4 ergs/sq mm at 365 nanometers) resulted in an increase in SCE that reached 15.4/cell at 5 mg/kg 8-MOP. Exposure of animals to 8-MOP or near-UV light alone did not increase SCE. SCE frequencies in cheek pouch cells were compared with the frequencies in marrow cells of identically treated animals, but the differences were not sufficient to justify a conclusion about the importance of exposure mode and tissue specificity in SCE formation. (32 refs)

79-4390 Carcinogenic Action of Low-Dose Cyclophosphamide Given Orally to Sprague-Dawley Rats in a Lifetime Experiment. (Eng) Schmahl, D. (Institut für Toxikologie und Chemotherapie, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, 69 Heidelberg, W. Germany); Habs, M. *Int J Cancer* 23(5): 706-712; 1979.

Sprague-Dawley rats were fed cyclophosphamide (CP) in their drinking water over their lifetime in an experiment designed to simulate long-term maintenance therapy with this drug. Daily doses of 2.5 (1), 1.25 (2), 0.63 (3), and 0.31 (4) mg/kg were given to groups of 40 male and 40 female rats 5x/wk. All rats were observed for life. The percentage of control animals with malignant tumors was 11% for males and 15% for females. The corresponding figures for CP-treated animals was 42% and 33% (Group 1), 43% and 33% (Group 2), 39% and 35% (Group 3), and 32% and 30% (Group 4). The total 97 tumor-bearing CP-treated rats had 115 malignant tumors in various organs. There was a distinctly increased risk for tumors of the urinary bladder (transitional-cell carcinomas), lymphoid and hematopoietic tissue, and nervous system. Significantly more urinary bladder carcinomas were seen in males than in females. Acute leukemias were seen most frequently among lymphoid and hematopoietic tissue tumors. The nervous system tumors were neurogenic sarcomas arising mostly from peripheral nerves. These results support evidence emerging from case reports showing an increased risk of urinary bladder cancer and leukemia in patients treated with CP. The results demand the classification of CP as a strong carcinogen and indicate that the iatrogenic risk of inducing cancer with alkylating agents like CP is a serious problem. (38 refs)

79-4391 Risk of Acute Leukosis after Treatment of Patients with Chronic Inflammatory Diseases with Immunosuppressive Cytotoxic Agents. Results of a Retrospective Study of 2,006 Patients. (Fre) Kahn, M. F. (Service de Rhumatologie, Hopital Bichat, 170 boulevard Ney, F 75877 Paris Cedex 18, France); Arlet, J.; Bloch-Michel, H.; Caroit, M.; Chaouat, Y.; Renier, J. C. *Rev Rhum Mal Osteoartic* 46(3): 163-167; 1979.

The incidence of acute leukemia (AL) among 2,006 patients suffering from chronic inflammatory diseases (1,853 with rheumatoid arthritis, 80 with lupus erythematosus disseminatus, 35 with psoriatic rheumatism, and 38 with scleroderma, periarteritis nodosa, dermatomyositis, Behcet's syndrome, or spondylarthritis) and treated with immunosuppressive cytotoxic agents was determined by a retrospective study. The cytotoxic agents were chlorambucil (CA), cyclophosphamide (CP), and azathioprine (AZ). The follow-up period varied between 1 and 13 yr. Nineteen of the patients developed AL 2.8-5.7 yr after the start of treatment. This number is probably lower than the actual number, as some patients were lost to follow-up or were observed for too short a period. Myeloblastic leukemia was the predominant type of AL, occurring in 12/19 patients. AL occurred in 16/1,711 patients treated with CA, 3/229 patients treated with CP, and 0/66 patients treated with AZ. AL did not occur in patients treated for < 6 mo or with < 1 g CA or < 50 g CP. The risk of AL was similar for CA and CP. Four of the 35 psoriatic rheumatism patients developed AL, a relatively high percentage. The incidence of AL in the entire series was comparable to that seen in large-scale investigations of patients with various malignant diseases who were treated with cytotoxic agents. (15 refs)

79-4392 Conformation of Exocyclic Amino Groups in Purines and Pyrimidines: Crystal Structure and Conformation of 1-Methyl-N⁴-hydroxycytosine Hydrochloride. (Eng) Birnbaum, G. I. (Div. Biological Sciences, Natl. Res. Council Canada, Ottawa, Ontario K1A 0R6, Canada); Kulikowski, T.; Shugar, D. *Can J Biochem* 57(4): 308-313; 1979.

The crystal structure and conformation of the hydrochloride salt of 1-methyl-N⁴-hydroxycytosine were examined by x-rays. The structure was determined by direct methods, and atomic parameters were refined by block-diagonal least squares. No intramolecular hydrogen bond was demonstrated in the solid state. However, the N⁴-hydroxy substituent was found to be syn to the ring N(3). In soln, when the molecules are far apart, an intramolecular hydrogen bond could play a role in stabilizing the syn conformation. The existence of the 1-methyl-N⁴-hydroxycytosine cation in the syn conformation may be relevant to the mechanism of attack of 1-

methyleytosine by hydroxylamine, which proceeds predominantly with the cationic form of cytosine. The conformation of substituted exocyclic amino groups in pyrimidines and purines is also of considerable importance in other biological phenomena. One of these involves N⁴-acetylcytidine, which occurs in several transfer RNA's (tRNA's) and the small ribosomal unit of eukaryotes and has the role of preventing errors during protein synthesis. The conformation of the exocyclic N⁶-methylamino group in N⁶-methylaminoadenosine, which, at the monomer level, exhibits a 20:1 preference for the form in which the methyl group is syn to the ring N(1), interferes with Watson-Crick base pairing. The presence of such residues in double-helical structures may affect their recognition by DNA modification-restriction enzymes. (25 refs)

79-4393 Synthesis and Coding Properties of Dinucleoside Diphosphates Containing Alkyl Pyrimidines Which Are Formed by the Action of Carcinogens on Nucleic Acids. (Eng) Singer, B. (Dept. Molecular Biology and Virus Lab., Univ. California, Berkeley, CA 94720); Pergolizzi, R. G.; Grunberger, D. *Nucleic Acids Res* 6(4): 1709-1719; 1979.

Dinucleoside diphosphates of the general type pGpN were prepared and used to test the ability of alkylated uridines (U's) and cytidines (C's), products of carcinogens acting on nucleic acids, to stimulate the binding of alanine- or valine-transfer RNA (tRNA) to ribosomes. Alkylation of U at the O² or O⁴ position caused a marked decrease in binding, whereas substitution at the N-3 position totally abolished the coding properties of the doublet. The highest binding occurred with pGpO²-ethyl-C and pGp3-methyl-C. 3-Ethyl-U acted like C and stimulated the binding of alanine-tRNA to approx the same extent that pGpC did. Doublets containing O²- and O⁴-ethyl-U's were poor templates. There was no stimulation of binding when the doublets contained O²- or N-3-alkyl-C. Thus, modifications of the hydrogen-bonding sites of U or C cause miscoding and could be considered to represent mutagenic reactions. (23 refs)

79-4394 Spontaneous and Mitomycin-C Induced Sister-Chromatid Exchanges. Comparison of In Vivo and In Vitro Systems. (Eng) Kram, D. (Section on Cellular Aging and Genetics, Lab. Cellular and Molecular Biology, Gerontology Res. Center, Natl. Inst. Aging, NIH, Baltimore, MD 21224); Schneider, E. L.; Senula, G. C.; Nakanishi, Y. *Mutat Res* 60(3): 339-347; 1979.

Baseline and mitomycin C (MMC)-induced levels of sister chromatid exchanges (SCE's) were determined in vivo, in C57BL/6J mouse bone marrow cells, and in vitro, in mouse fibroblasts with the use of bromodeoxyuridine (BrdU) differential labeling techniques. In addition the in-

teraction of BrdU with MMC in the induction of SCE's in vivo was analyzed. Although BrdU induced SCE in both systems, baseline SCE levels were estimated to be two- to threefold higher in vitro than in vivo. SCE induction was found to be a linear function of MMC concentration in vivo and in vitro; however, the slope of the in vivo curve was five-fold higher. The interaction of BrdU-substituted DNA and MMC was examined by giving the mice a fixed dose of MMC (2.5 mg/kg) and increasing concentrations of BrdU (25-125 mg/kg/hr). The induced SCE frequencies appeared to be additive. In addition to allowing the measurement of drug-induced SCE's, the BrdU differential staining technique allows concomitant measurement of the inhibition of cellular replication by test drugs. (33 refs)

79-4395 Synthesis of 2-Amino-9H-pyrido[2,3-b]indole Isolated as a Mutagenic Principle from Pyrolytic Products of Protein. (Eng) Matsumoto, T. (Central Res. Inst., Japan Tobacco and Salt Public Corp., 6-2 Umegaoka, Midori-ku, Yokohama, Kanagawa 227, Japan); Yoshida, D.; Tomita, H.; Matsushita, H. *Agric Biol Chem* 43(3): 675-677; 1979.

The synthesis of 2-amino-9H-pyrido(2,3-b)indole (API) identical to the mutagenic compound obtained from pyrolysis products of soybean globulin is described. The synthetic API was mutagenic for *Salmonella typhimurium* strain TA98 when tested with a liver microsome fraction from polychlorinated biphenyl-pretreated rats. Its mutagenic potency was comparable to that of aminobenzoquinoline. (10 refs)

79-4396 The Enhancing Effect of Cysteine and Its Derivatives on the Mutagenic Activities of the Tryptophan-Pyrolysis Products, TRP-P-1 and TRP-P-2. (Eng) Negishi, T. (Faculty Pharmaceutical Sciences, Okayama Univ., Tsushima, Okayama 700, Japan); Hayatsu, H. *Biochem Biophys Res Commun* 88(1): 97-102; 1979.

The effect of cysteine and its derivatives on the mutagenicity of the tryptophan-pyrolysis products Trp-P-1 and Trp-P-2 and of a beef-extract mutagen was determined in the *Salmonella*-microsome assay. Cysteine and its derivatives enhanced the mutagenic activity of Trp-P-1 and Trp-P-2. A severalfold increase in the number of revertant colonies was caused by the addition of 10 mM cysteine, cysteine ethyl ester, or cysteamine to the reaction mixture. Both the thiol and amino groups were found to be necessary for the enhancing effect. The cysteine derivatives did not affect the mutagenic activity of benzo(a)pyrene or the beef-extract mutagen. (19 refs)

- 79-4397 Teratogenic Effects of the Plant Hormone Indole-3-acetic Acid in Mice and Rats. (Eng) John, J. A. (Toxicology Res. Lab., Health and Environmental Res., Dow Chemical U.S.A., Midland, MI 48640); Blogg, C. D.; Murray, F. J.; Schwetz, B. A.; Gehring, P. J. *Teratology* 19(3): 321-326; 1979.

The teratogenic potential of indole-3-acetic acid (IAA), a naturally occurring plant hormone, was evaluated in CF-1 mice and Sprague-Dawley rats given max tolerated doses during the period of major organogenesis. Mice were given 5, 50, 200, or 500 mg/kg/day by gavage on days 7 through 15 of gestation. Rats were given 50, 200, or 500 mg/kg/day by gavage on days 7 through 15 of gestation. IAA was teratogenic in mice and rats at 500 mg/kg/day; cleft palate was induced in both species at this dose level. In mice, other malformations, including exencephaly, ablepharia, dilated cerebral ventricles, and crooked tail, were also observed. Mice given 500 mg/kg of IAA gained less wt than control mice during gestation; no evidence of maternal toxicity was observed in the rats. IAA did not cause fetal resorptions in either species, and it was not teratogenic at dose levels < 500 mg/kg. (22 refs)

- 79-4398 Microsomal Mediated Metabolism of Dialkylaryltriazenes. I. Demethylation of Ring Halogenated 3,3-Dimethyl-1-phenyltriazenes. (Eng) Pool, B. L. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany). *J Cancer Res Clin Oncol* 93(3): 215-220; 1979.

The degree of oxidative N-demethylation of several ring-substituted (halogenated) 3,3-dimethyl-1-phenyltriazenes was investigated. The results indicate that substitution of the ring with deactivating atoms correlates well with the extent of demethylation. The percentages of demethylation were 45% for 3,3-dimethyl-1-phenyltriene, 92% for 3,3-dimethyl-1-(4-chlorophenyl) triene, 89% for 3,3-dimethyl-1-(4-bromophenyl) triene, 122% for 3,3-dimethyl-1-(2,4,6-trichlorophenyl) triene, and 85% for 3,3-dimethyl-1-(2,4,6-tribromophenyl) triene. (22 refs)

- 79-4399 Microsomal Mediated Metabolism of Dialkylaryltriazenes. II. Isolation and Identification of Metabolites of 3,3-Dimethyl-1-phenyltriene. (Eng) Pool, B. L. (Inst. Toxicology and Chemotherapy, German Cancer Res. Center, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany). *J Cancer Res Clin Oncol* 93(3): 221-231; 1979.

An attempt was made to isolate and identify microsome-mediated metabolites of 3,3-dimethyl-1-phenyltriene. After 3,3-dimethyl-1-phenyltriene was incubated with rat liver microsomes, acetanilide, derivatives of aniline, and,

possibly, derivatives of 3-methyl-1-phenyltriene were found as metabolites and identified by mass spectrometry. This is the first time that directly formed metabolites (other than formaldehyde) of a dialkyltriene were identified. The isolation of a derivative of 3-methyl-1-phenyltriene as 3-acetyl-3-methyl-1-phenyltriene supports other evidence that the trienes are enzymically demethylated. Evidence of the formation of phenylhydrazine was also obtained. In addition, hydrolysates of the polar fractions of the incubation mixture contained aniline and 4-hydroxyaniline as aglycones. (25 refs)

- 79-4400 Heterotransplantation of Rat Small Intestinal Adenocarcinoma Induced by Panfuran-S Containing 3-Di(hydroxymethyl)amino-6-(5-nitro-2-furyl-ethenyl)-1,2,4-triazine into Nude Mice. (Eng) Konishi, Y. (Dept. Oncological Pathology, Cancer Center, Nara Medical Univ., Nara, Japan); Sunagawa, M.; Yoshimura, H.; Denda, A.; Kojima, K.; Takahashi, S.; Miyagi, N.; Shiratori, T. *J Nara Med Assoc* 29(4/5): 663-668; 1978.

An experimental small intestinal carcinoma line was established in nude mice to obtain information on the growth of these tumors. Tumors transplanted sc were obtained from jejunal tumors induced by feeding rats 3,500 ppm Panfuran-S containing 3-di(hydroxymethyl)amino-6-(5-nitro-2-furyl-ethenyl)-1,2,4-triazine (DHNT). Tumors transplanted ip were obtained at passage 3 from nude mice that had been given serial sc transplants of the rat jejunal adenocarcinoma. The serial transfer of sc tumors was successful through passage 12, with a 100% tumor take. Following ip transplantation (1×10^7 cells/0.5 ml), carcinomatous peritonitis was consistently found by 6 wk with a single transfer and by 3-4 wk with the serial passages. Neither invasion nor metastases were seen in the transplanted sc tumors. Tumor cells transplanted ip grew with bloody ascites, local invasion, and, occasionally, metastases. They invaded the peritoneum, diaphragm, omentum, mesenteries, perirenal fatty tissues, and pancreas. Histologically, the rat jejunal tumors were tubular or papillary adenocarcinomas. The tumors that grew in the sc space showed a more medullary pattern, but they were basically similar to the original rat jejunal adenocarcinoma. It is concluded that this tumor system will provide a useful experimental model for studying the growth of small intestinal carcinoma. (21 refs)

- 79-4401 Assessment of the Use of the *Salmonella* Mutagenesis Assay to Determine the Influence of Antioxidants on Carcinogen-induced Mutagenesis. (Eng) Rosin, M. P. (Environmental Carcinogenesis Unit, British Columbia Cancer Res. Centre, 601 W. 10th Ave., Vancouver, British Columbia V5Z 1L3, Canada); Stich, H. F. *Int J Cancer* 23(5): 722-727; 1979.

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The *Salmonella typhimurium* mutagenesis assay was used to study the effect of antioxidants on the mutagenicity of N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) and N-acetoxy-2-acetylaminofluorene (N-acetoxy-AAF). None of the antioxidants had any effects on the spontaneous mutation frequency of treated bacteria. Cysteine, glutathione, cysteamine, and sodium bisulfite were very effective inhibitors of MNNG mutagenicity. Sodium selenite, sodium ascorbate, and propyl gallate were only slightly less effective. α -Tocopherol and butylated hydroxyanisole had no effect. The mutagenicity of N-acetoxy-AAF was significantly reduced only with cysteamine, propyl gallate, and selenite. The antioxidant concentration necessary to inhibit 50% of carcinogen-induced mutagenesis (IN_{50}) was estimated. Cysteamine and bisulfite, which have a low IN_{50} ($1.5-2.5 \times 10^{-5} M$) with MNNG treatments, would be very efficient inhibitors of the activity of this carcinogen. Selenite, propyl gallate, and ascorbate would be less effective (IN_{50} from 30 to $300 \times 10^{-5} M$). There was little change in the effectiveness of a particular antioxidant in inhibiting activity at high or low carcinogen doses. Dimethyl sulfoxide, when used as a solvent, significantly affected the action of an antioxidant. The results indicate that no general pattern exists in the ability of an antioxidant to inhibit the mutagenicities of carcinogens. (27 refs)

- 79-4402 **Animal Models in Cancer Research Which Could Be Useful in Studies of the Effect of Alcohol on Cellular Immunity.** (Eng) Chirigos, M. A. (Virus and Disease Modification Section, Lab. Chemical Pharmacology, NIH, Developmental Therapeutics Program, Div. Cancer Treatment, Bethesda, MD 20205); Schultz, R. M. *Cancer Res* 39(7, part 2): 2894-2898; 1979.

Animal models for studying the relationship between alcoholism (which could be a stress to the host immune system) and cancer are described. When 1,3-bis(2-chloroethyl)-1-nitrosourea (30 mg/kg, sc) was administered to BALB/c x DBA/2F, mice up to 4 days prior to inoculation of MBL-2 leukemic cells, all animals developed progressively growing tumors. However, treatment 6 days prior to tumor inoculation resulted in only a 40% death with tumor and a 60% regression. Macrophages from unstressed mice treated with interferon (100 units/ml) inhibited MBL-2 tumor cell proliferation by 77%. Immobilization stress 1 day prior to interferon treatment resulted in a 75% reduction of interferon activity, and stress on the same day as interferon treatment resulted in a 54% reduction in interferon activity. Hydrocortisone, prednisone, and dexamethasone, when administered ip at 1-100 mg/kg simultaneously with interferon, suppressed macrophage tumor killing in a dose-dependent fashion. The interferon induction of tumoricidal macrophages was markedly reduced when prostaglandin E_1 or E_2 was added to macrophage cultures simultaneously with interferon. Indomethacin had no effect on resting macrophages. Since immune deficiency is a trait of alcoholism and cancer,

animal models with defined, measurable, immunological parameters would be useful in studying the effect of alcohol on cellular immunity. (29 refs)

- 79-4403 **Promoting Action of Croton Oil on Gastrocarcinogenesis by N-Methyl-N'-nitro-N-nitrosoguanidine in Rats.** (Eng) Matsukura, N. (Biochemistry Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan); Kawachi, T.; Sano, T.; Sasajima, K.; Sugimura, T. *J Cancer Res Clin Oncol* 93(3): 323-327; 1979.

The promoting effect of croton oil on gastrocarcinogenesis induced by N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) was examined in male Wistar rats. Gastric carcinomas were found in 5/10 rats given 83 $\mu g/ml$ MNNG in the drinking water for 3 mo and then 0.02% croton oil with 0.5% Tween 60 as solvent for 9 mo. Histologically, all the gastric carcinomas were well-differentiated adenocarcinomas that showed proliferation of well-differentiated atypical glandular epithelium. No gastric carcinomas were found in rats given MNNG for 3 mo and then Tween 60 only for 9 mo. The incidence of gastric carcinomas in these two groups was significantly different ($p < 0.05$). No tumors were found in rats given only croton oil with Tween 60. (9 refs)

- 79-4404 **Vitamin C Is Positive in the DNA Synthesis Inhibition and Sister-Chromatid Exchange Tests.** (Eng) Galloway, S. M. (Lab. Radiobiology, Univ. California, San Francisco, CA 94143); Painter, R. B. *Mutat Res* 60(3): 321-327; 1979.

The sister-chromatid exchange (SCE) and DNA synthesis inhibition tests were used to investigate the effects of sodium ascorbate and/or N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) on Chinese hamster ovary (CHO) cells and in human lymphocytes and HeLa cells. The ascorbate (0.1-10 mM) caused a dose-dependent increase in SCE's in the CHO cells and in the human lymphocytes. Moreover, in the DNA synthesis inhibition test with HeLa cells, ascorbate gave results typical of DNA-damaging chemicals. At 2-20 mM, it caused a dose-dependent inhibition of DNA synthesis in the HeLa cells. Catalase reduced SCE induction by ascorbate, prevented its cytotoxicity in CHO cells, and prevented its effect on HeLa DNA synthesis. Ascorbate reduced the induction of SCE's in CHO cells by MNNG, probably by direct chemical reduction of MNNG. The results indicate that ascorbate damages DNA and, therefore, that it is a mutagen and potential carcinogen. (25 refs)

- 79-4405 **The Relevance of Caffeine Post-treatment to SCE Incidence Induced in Chinese Hamster**

Cells. (Eng) Popescu, N. C. (Lab. Biology, NCI, Bethesda, MD 20014); Amsbaugh, S. C.; Dipaolo, J. A. *Mutat Res* 60(3): 313-320; 1979.

The effect of caffeine posttreatment on sister-chromatid exchange (SCE) and chromosome aberration frequencies in Chinese hamster V79-4 cells exposed to a variety of chemical and physical agents and then stained with bromodeoxyuridine (BrdUrd) was determined. After 2 hr treatment, N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) and cis-platinum(II) diammine dichloride [cis-Pt(II)] induced a seven- and sixfold increase in SCE's, respectively, but 4-nitroquinoline 1-oxide (4NQO), methyl methanesulfonate (MMS), proflavine, and N-hydroxyfluorenylacetylacetamide (OH-AAF) caused a two- to threefold increase in SCE's compared with controls treated with BrdUrd alone. UV light doubled the number of SCE's. The lowest increase of SCE's was obtained with bleomycin and x-radiation. Caffeine posttreatment (0.5 mM) caused a statistically significant increase in the frequency of SCE's induced by UV and x-radiation as well as by 4NQO and MMS, but it did not alter the number of SCE's induced by MNNG, cis-Pt(II), proflavine, OH-AAF, and bleomycin. Caffeine posttreatment increased the number of cells with chromosome aberrations induced by MNNG, cis-Pt(II), UV light, 4NQO, MMS, and proflavine. With the exception of proflavine, these agents are dependent on DNA and chromosome replication for the expression of chromosome aberrations. Caffeine enhancement of cis-Pt(II)-induced chromosome aberrations occurred independently of the time interval between treatment and chromosome preparation. Chromosome damage produced by bleomycin and x-irradiation, agents known to induce chromosome aberrations independent of the S phase of the cell cycle, as well as damage produced by OH-AAF was not influenced by caffeine posttreatment. The enhancement by caffeine, an inhibitor of the gap-filling process in postreplication repair, of chromosome aberrations induced by S-dependent agents is consistent with the involvement of this type of repair in chromosome aberration formation. The lack of inhibition of SCE frequency by caffeine indicates that postreplication repair is probably not important in SCE formation. (17 refs)

79-4406 Cardiovascular Lesions and Various Tumors Found in Rats Given T-2 Toxin, a Trichothecene Metabolite of *Fusarium*. (Eng) Schoental, R. (Dept. Pathology, Royal Veterinary Coll., Univ. London, London, NW1 0TU, England); Joffe, A. Z.; Yagen, B. *Cancer Res* 39(6, part 1): 2179-2189; 1979.

Following the intragastric administration of 3 α -hydroxy-4 β ,15(diacetoxy-8 α -(3-methylbutyryloxy)-12,13-epoxy-trichothec-9-en (T-2 Toxin), a trichothecene metabolite of several *Fusarium* species, white Wistar-Kortrat rats developed various topical and systemic lesions, both acute and chronic. The rats that survived 12-27 mo

after the first of 3-8 doses of T-2 toxin (0.2-4 mg/kg), alone or in conjunction with nicotinamide (200-250 mg/kg, ip), developed cardiovascular lesions of various degrees of severity and/or benign and malignant tumors of the digestive tract and brain. T-2 toxin has been known occasionally to contaminate cereals and other agricultural products, harvested or stored under damp and cold conditions. In the USSR, it was responsible for an often fatal human disease, known as alimentary toxin aleukia. The toxin has also been implicated in outbreaks of hemorrhagic mycotoxicoses in livestock in various countries. T-2 toxin and other *Fusarium* mycotoxins may be involved in the etiology of cardiovascular lesions and of certain animal and human tumors considered to be spontaneous. (43 refs)

79-4407 Studies of Nicotine-metabolizing Enzyme Activities in Various Animal Strains and Species. (Jpn) Kuroguchi, Y. (Dept. Pharmacology, Nara Medical Univ., Kashiwara 634, Japan); Nakashima, T.; Nakanishi, Y.; Inoki, M.; Morita, N. *J Nara Med Assoc* 29(4/5): 677-682; 1978.

Species and strain differences in the metabolism of nicotine (N), acetylaminofluorene (AAF), and benzo(a)pyrene (BP) were studied by analyzing the hepatic drug-metabolizing enzyme activities of several strains of male mice and rats, Hartley guinea pigs, Japanese white rabbits, and mongrel dogs. The animals were given water and commercial food ad lib for 1 wk, fasted for 18 hr, and then sacrificed. Their livers were homogenized and centrifuged, and the supernatant was analyzed for enzyme activity. There were significant differences in the metabolism of N, AAF, and BP among 11 Wistar, 14 Sprague-Dawley, 16 Donryu, and 8 Fischer rats, all aged 7 wk. Similarly, significant differences were seen among various strains of 7-wk-old mice: 4 ddY, 10 C57BL/6J, 15 ICR, 10 DBA/2J, 10 C3H/He, 9 BALB/c, and 10 BDF1. Differences across species lines were observed by comparing the enzyme activities of 9 dogs, 9 rabbits, 9 guinea pigs, 11 Wistar rats, and 4 ddY mice. The results show that there was no particular similarity in metabolic activity among the various strains and species tested. (17 refs)

79-4408 Investigation of Nicotine Metabolism in Mongrel Dogs. (Jpn) Kuroguchi, Y. (Dept. Pharmacology, Nara Medical Univ., Kashiwara 634, Japan); Nakashima, T.; Morita, N. *J Nara Med Assoc* 29(4/5): 669-676; 1978.

Gas chromatography was used to study the metabolism of tritium-labeled nicotine in 10-kg mongrel dogs. In the first experiment, a dog was given a single dose of 100 μ g/kg nicotine iv. The decrease in the serum nicotine level that occurred 30-90 sec after the injection corresponded to a peak level of cotinine at 30 sec. A peak level of nornicotine was

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seen from 5 to 20 min. The butanol extract of serum collected after iv injection of 2 $\mu\text{g}/\text{kg}$ nicotine every minute for 1 hr showed multiple peaks of nicotine, cotinine, nor-nicotine, and other compounds, some of which were presumed to be free fatty acids. Peak retention times were 5.9 min for cotinine, 6.4 min for nicotine, and 14.7 min for nornicotine. The retention curve for the 2- $\mu\text{g}/\text{ml}$ injections was the same as that for the single 100- $\mu\text{g}/\text{kg}$ injection in the early stage of the elution half-time and was slightly more prolonged, although similar, thereafter. When a single dose of 200 $\mu\text{g}/\text{kg}$ nicotine was injected sc, the peak retention times for cotinine, nicotine, and nornicotine were 5.9, 6.4, and 14.8 min, respectively. In a 24-hr urine specimen, nicotine, cotinine, and nornicotine plus three other compounds were identified by gas chromatography-mass spectrometry. The results indicate that the in vivo metabolism of nicotine in the dog starts immediately after administration. (20 refs)

(Medical Res. Council, Clinical and Population Cytogenetics Unit, Western General Hosp., Edinburgh, Scotland); Evans, H. J. *Nature* 279(5710): 241-242; 1979.

The formation of DNA lesions resulting in sister chromatid exchanges (SCE) following exposure of human lymphocytes (from two healthy, nonsmoking males) to tobacco smoke condensate was studied. In lymphocyte cultures from both subjects, 2.5 mg of condensate was cytotoxic, whereas small amounts caused dose-dependent increases in SCEs. The SCE frequency was detectably increased by 0.1 mg of condensate and more than doubled by 0.5 mg, these doses representing 1/400th and 1/80th of a single high-tar cigarette, respectively. The ability of the tobacco smoke condensate to produce DNA lesions resulting in SCE cannot be ascribed to its benzo(a)pyrene content alone. The results indicate that the levels of agents that can react with cellular DNA may be particularly high in the respiratory airways of inhaling cigarette smokers. (27 refs)

79-4409 Assessment of Tobacco-specific N-Nitrosamines in Tobacco Products. (Eng) Hoffmann, D. (Div. Environmental Carcinogenesis, Naylor Dana Inst. Disease Prevention, Dana Road, Valhalla, NY 10595); Adams, J. D.; Brunnemann, K. D.; Hecht, S. S. *Cancer Res* 39(7, part 1): 2505-2509; 1979.

Tobacco-specific nonvolatile N-nitrosamines in tobacco and in fresh mainstream and sidestream smoke of cigarettes and cigars were quantitatively determined with a thermal energy analyzer (TEA). The smoke was trapped in ascorbic acid solution buffered at pH 4.5 and extracted with dichloromethane, and the organic phase was chromatographed and analyzed by high-performance liquid chromatography with TEA. The nonvolatile nitrosamines were further enriched by repeated chromatography and positively identified by gas-liquid chromatography-mass spectrometry. [2'- ^{14}C]N'-Nitrosonornicotine served as internal standard for the quantitative analysis. The tobacco of five different cigarettes contained 0.22-7.0 ppm of the carcinogen N'-nitrosonornicotine, 0.13-0.74 ppm of the carcinogen 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone, and 0.44-3.2 ppm of the newly identified N'-nitrosoanatabine. In unaged mainstream and sidestream smoke of the same cigarettes, the respective values were 0.24-0.37 and 0.15-6.1 $\mu\text{g}/\text{cigarette}$ for N'-nitrosonornicotine, 0.11-0.42 and 0.19-0.66 $\mu\text{g}/\text{cigarette}$ for 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone, and 0.33-4.6 and 0.15-1.5 $\mu\text{g}/\text{cigarette}$ for N'-nitrosoanatabine. These relatively high concentrations in sidestream smoke should make it possible to detect tobacco-specific nitrosamines in polluted indoor environments. (26 refs)

79-4411 Low Natural Killer-Cell Activity and Immunoglobulin Levels Associated with Smoking in Human Subjects. (Eng) Ferson, M. (Kanematsu Memorial Inst., Sydney Hosp., Sydney, N. S. W., Australia); Edwards, A.; Lind, A.; Milton, G. W.; Hersey, P. *Int J Cancer* 23(5): 603-609; 1979.

A study was conducted to determine whether an association between smoking and the increased incidence of cancer may be due to an effect on natural killer (NK) activity. Age- and sex-matched normal smokers (60) and nonsmokers (59) and male smoking (13) and nonsmoking (27) melanoma patients were studied with respect to the NK activity of their WBC against cultured melanoma and Chang cells. In addition, serum IgG, IgM, and IgA levels and erythrocyte (E) rosette levels were assessed in the normal subjects. The WBC NK activity of the normal subjects and melanoma patients who were smokers against cultured melanoma target cells was much lower than that of the nonsmokers. The same trend was seen against the Chang target cells, but it was not as pronounced. Smokers had lower IgG and IgA, but not IgM, levels in their sera than nonsmokers. There was no significant difference in the percentage of T cells, measured as E rosettes, between the nonsmoking and smoking groups. The changes in NK activity and Ig levels may explain the link between smoking and the increased incidence of malignancy. These results suggest that a closer examination of the effects of smoking on the immune system in relation to malignancy is warranted. (33 refs)

79-4410 Cigarette Smoke Condensates Damage DNA in Human Lymphocytes. (Eng) Hopkin, J. M.

79-4412 Multicentric Metachronous Primary Carcinoma of the Upper Alimentary Tract in 'Biri' Smokers. (Eng) Gill, R. S. (Dept. ENT, S. N. Medical Coll., Agra, India); Gill, J. K.; Rhotgi, V. K.;

Mishra, U. C.; Lal, M. *J Laryngol Otol* 93(5): 527-531; 1979.

The occurrence of multiple metachronous primary squamous cell carcinomas (SCC) of the upper alimentary tract in three Biri smokers (a form of cheap cigarette made by rolling sun-dried, uncured tobacco in dried leaf of Tam-burni) is reported. A 45-yr-old man with a long history of heavy (40-50/day) Biri smoking presented with a SCC of the left pyriform fossa. After radiotherapy, he reduced the number of Biris smoked/day by half, but he presented 13 yr later with a SCC of the left tonsil. A 50-yr-old Biri smoker (30-50/day) presented with a SCC of the left cheek. He was treated with surgery and radiotherapy, and he reduced his smoking to approx 16/day. After 4 yr and 10 mo he presented with a SCC of the soft and hard palates. A 40-yr-old man who smoked 20-30 Biris/day presented with a SCC of the left pyriform fossa. He was treated with radiotherapy, but returned 2.5 yr later with a SCC of the esophagus. In all three cases, the second tumors responded to radiotherapy. (10 refs)

79-4413 Mutagenicity of 8-Ethoxycaffeine In Vitro. Induction of Point Mutations in the *Salmonella*/Microsome Test and of Sister-Chromatid Exchanges as Well as Chromosomal Aberrations in Chinese Hamster Ovary Cells (CHO Line). (Eng) Strobel, R. (Dept. Human Genetics and Anthropology, Univ. Dusseldorf, 4000 Dusseldorf 1, W. Germany); Roszinsky-Kocher, G.; Rohrborn, G. *Mutat Res* 60(3): 349-355; 1979.

The mutagenicity of the caffeine derivative 8-ethoxycaffeine (EOC) was determined in three in vitro test systems. Each experiment was carried out in the presence and absence of an S9 mix. Incubation temperatures were 20 and 37 C. In the *Salmonella*/microsome test, EOC behaved as a promutagen in *Salmonella typhimurium* strain TA1535. No mutagenic activity was found in experiments without an S9 mix. The influence of temperature was negligible. The mutagenicity of EOC depended mainly on the mammals used to prepare the S9 fraction and on the agents given to them to induce liver enzymes. S9 fractions from mice were more effective than those from rats, and Aroclor-1254 was a more effective inducing agent than phenobarbital. EOC did not induce sister-chromatid exchanges in Chinese hamster ovary cell cultures at 20 or 37 C. However, EOC induced chromosome aberrations in Chinese hamster ovary cells when they were incubated at 37 C without the S9 mix. (12 refs)

79-4414 Mutagenicity of p-Nitrophenyl-p'-guanidinobenzoate on *Salmonella typhimurium* Strain TA98. (Eng) Bracha, M. (Dept. Field and Vegetable Crops, Hebrew Univ., P. O. Box 12, Rehovot, Israel). *Mutat Res* 67(1): 81-83; 1979.

The mutagenicity of p-nitrophenyl-p'-guanidinobenzoate (NPGB: an active site inhibitor of serine proteases) for *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538 was studied. NPGB increased the number of revertant colonies when incubated with strain TA98, but it had no effect on the reversion frequencies in the other strains. Thus, NPGB appears to be a specific frameshift mutagen. Addition of a rat liver homogenate reduced the mutagenicity of NPGB two- to threefold, and the reversion frequency was not increased by incubation of TA98 with p-nitrophenol or guanidinobenzoate (the products of NPGB hydrolysis). In nutrient broth culture, NPGB (125 µg/ml) reduced the viability of TA98 cells to 27%. At higher concentrations, the effect was much greater. (9 refs)

79-4415 Chromosome Changes Induced by Industrial Chemicals. (Jpn) Koizumi, A. (Dept. Public Health, Faculty Medicine, Univ. Tokyo, Tokyo, Japan); Dobashi, Y.; Tachibana, Y. *Jpn J Ind Health* 21(1): 3-10; 1979.

Radiation-induced chromosome damage has been widely recognized and intensively studied. Recently, attention has focused on chromosome changes induced by various industrial chemicals. In the case of occupational exposure to ionizing radiation, chromosome breaks are one of the most sensitive biological effects. Chromosome breaks have also been reported in workers exposed to benzene, vinyl chloride monomer, or styrene. Ionizing radiation, benzene, and vinyl chloride monomer are known carcinogens, and attention is now being given to carcinogenicity of clastogens or chromosome-breaking agents. Studies on in vitro chromosome breakage induced by benzene and its metabolites, as well as by cadmium, lead, and chromium compounds, are reviewed. The inhibition of repair of radiation-induced chromosome breaks by clastogens and the significance of cytogenetic studies in industrial medicine are also discussed. (40 refs)

79-4416 Chromosome Analysis in Two Unusual Malignant Blood Disorders Presumably Induced by Benzene. (Eng) Van den Berghe, H. (Div. Human Genetics, Minderbroedersstraat 12, B-3000 Leuven, Belgium); Louwagie, A.; Broeckaert-Van Orshoven, A.; David, G.; Verwilghen, R. *Blood* 53(4): 558-566; 1979.

Two patients with malignant blood disorders presumably induced by benzene were monitored cytogenetically through the courses of their disease. A 25-yr-old draftsman who had used a solvent containing 2.2% benzene for 4 yr developed acute myelomonocytic leukemia. He presented with preleukemia characterized by an increased number of WBC with immature elements in the blood and an elevated number of blasts in the marrow. He experienced a seeming-

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ly complete remission without treatment, but a fatal relapse occurred 3 mo later. Cytogenetic investigation showed a familial translocation characterized by exchange of the long arm of chromosome No. 3 and the short arm of No. 16. An additional acquired translocation was demonstrated during the first phase of his disease: part of the short arm of No. 10 was translocated on the short arm of No. 9. This anomaly persisted, and an additional one was demonstrated after the patient became frankly leukemic: the long arm of No. 15 was almost completely exchanged with the short arm of No. 4. Also, two cells with 47 chromosomes showed an extra minute marker. A 67-yr-old retired man who had used glues containing benzene for many years also presented with preleukemia. He experienced an apparently complete remission without treatment, but he subsequently died from gastrointestinal bleeding and infectious complications. The patient initially showed chromosome No. 7 monosomy, which was later found in 100% of his cells. After spontaneous remission, when the cellularity of the marrow became normal, all metaphases showed a normal karyotype. (18 refs)

79-4417 Enzymatic Changes in Peripheral Blood Leukocytes in Rats in Subacute Benzene Vapours Poisoning. II. The Activity of Neutrophil Enzymes. (Eng) Starek, A. (Chair Medicine Work and Occupational Diseases, Medical Acad., 30-969 Krakow, Poland); Moszczynski, P.; Czarnobilski, Z. *Pol J Pharmacol Pharm* 30(4): 483-487; 1978.

The activity of enzymes from the peripheral blood neutrophils of male Wistar rats exposed to subacute concentrations of benzene vapor (27,000 mg/m³, 6 hr/day, for 10 consecutive days) was studied. Relative to preexposure levels, exposure to benzene resulted in a 40% reduction in the number of peripheral granulocytes ($p < 0.05$), a 20% reduction in acid phosphatase activity ($p < 0.001$), a 77% reduction in β -glucuronidase activity ($p < 0.001$), a 68% reduction in N-acetyl- β -glucosaminidase activity ($p < 0.001$), and a 36% reduction in alkaline phosphatase activity ($p < 0.001$). The acid phosphatase activity in benzene-exposed rats did not differ significantly from that in control rats. The results indicate a destructive action of benzene on the lysosomes of peripheral blood neutrophils. (27 refs)

79-4418 Nitrosamines in Agricultural and Home-Use Pesticides. (Eng) Bontoyan, W. R. (Office Pesticide Programs, Benefits and Field Studies Div., Chemical and Biological Investigations Branch, U.S. Environmental Protection Agency, Beltsville, MD 20705); Law, M. W.; Wright, D. P. *J Agric Food Chem* 27(3): 631-635; 1979.

Ninety-one technical and commercial pesticides used in

agriculture and in homes were analyzed for the presence of N-nitroso compounds (at the 1-ppm level) by three methods: gas-liquid chromatography (GLC) with a thermal energy analyzer (TEA) detector, GLC with a Hall electrolytic conductivity detector, and high-pressure liquid chromatography (HPLC) with a UV and a TEA detector. For the nonvolatiles, HPLC-UV and HPLC-TEA were used. Twenty-five test samples contained ≥ 1 ppm nitrosamines (NA's): 14 were dinitroaniline formulations, 7 were amine salts, 3 were amines used in the manufacturing process, and 1 was a sample containing a large quantity of N-nitrosodiethanolamine. With the exception of atrazine, none of the triazine herbicides were analyzed for nonvolatile NA's. However, the analyses did not indicate the presence of any volatile NA's in these products. The N-nitroso compounds found in dinitroaniline products probably result from a reaction of residual HNO₂ left from the nitration of chlorobenzene and the excess secondary amine used in the amination step. N-Nitroso compounds in formulations of amine salts of phenoxy herbicides probably result from the reaction of nitrite and the corresponding secondary amine. The results indicate that the higher NA levels are primarily found in substituted amine, dinitroaniline, and amine salt formulations, whereas the triazine compounds are free of NA contamination. (4 refs)

79-4419 Intrahepatic Bile Duct Proliferation Induced by 4,4'-Diaminodiphenylmethane in Rats. (Eng) Fukushima, S. (Dept. Pathology, Nagoya City Univ. Medical Sch., Kawasumi-cho, Mizuho-ku, Nagoya 467, Japan); Shibata, M.; Hibino, T.; Yoshimura, T.; Hirose, M.; Ito, N. *Toxicol Appl Pharmacol* 48(1): 145-155; 1979.

Sequential changes in the livers of male Wistar rats treated with 4,4'-diaminodiphenylmethane (DDPM: 1,000 ppm in the basal diet for 8-40 wk) were studied. Macroscopically, the liver remained essentially normal during the first 16 wk of treatment. After 24 wk, the surface was rough and granular, and the organ showed fibrotic changes and marked resistance to cutting. These alterations were more pronounced after longer periods of DDPM treatment. No tumors, enlargement of the common bile duct, or gallstones were observed, and no morphologic abnormalities were detected in other organs. The predominant histologic findings were bile duct proliferation and oval cell infiltration in portal areas throughout the liver. Bile duct proliferation began as early as 8 wk after treatment, and it was irregularly associated with oval cell proliferation. These changes gradually increased with longer periods of DDPM treatment and were marked after 24 wk. Although focal necrosis accompanied by an ingrowth of granulation tissue occurred occasionally in the periportal parenchyma, most hepatocytes appeared normal. Fatty changes were found in the livers of most rats given DDPM, and they were located at the periphery of the lobules. The proliferating bile duct cells showed increased γ -glutamyl transpeptidase (GGT) activity after 32 wk of treatment,

and GGT levels were also elevated in the serum. Alkaline phosphatase and ATPase were elevated in the periductular connective tissue. SGOT and SGPT levels initially increased, but they returned to normal values toward the last stage of DDPM intoxication. (30 refs)

- 79-4420 Macromolecular Binding and Metabolism of the Carcinogen 4-Chloro-2-methylaniline.** (Eng) Hill, D. L. (Kettering-Meyer Lab., Southern Res. Inst., Birmingham, AL 35205); Shih, T. W.; Struck, R. F. *Cancer Res* 39(7): 2528-2531; 1979.

The mechanisms of action and activation of the carcinogen 4-chloro-2-methylaniline were investigated. Radioactivity from 4-chloro-2-[methyl-¹⁴C]methylaniline became extensively bound to protein, DNA, and RNA of Osborne-Mendel rat liver but macromolecules of some of the other tissues examined contained little radioactivity. Enzymatic activity dependent on NADH and leading to irreversible binding of radioactivity from labeled 4-chloro-2-methylaniline to macromolecules in the reaction system was present in microsomes from rat liver. The activity was inducible by phenobarbital. Two soluble products of microsomal enzymes were identified by mass spectral analysis and chemical synthesis as 5-chloro-2-hydroxylaminotoluene and 4,4'-1-dichloro-2,2'-dimethylazobenzene. The hydroxylamino compound appears to be a more activated form of 4-chloro-2-methylaniline. (18 refs)

- 79-4421 Subchronic Effects of Piperonyl Butoxide on Carcinogen Metabolism in Hamster Liver.** (Eng) Friedman, M. A. (Dept. Pharmacology, Medical Coll. Virginia, Health Sciences Div., Virginia Commonwealth Univ., Richmond, VA 23298). *Bull Environ Contam Toxicol* 21(6): 815-821; 1979.

The effects of acute and subchronic exposure to piperonyl butoxide (PiB) on the activities of several enzymes involved in carcinogen metabolism were investigated in the Syrian golden hamster liver. Groups of hamsters were fed a diet containing 1% PiB for 1-91 days. The hamsters were killed, and their livers were removed and processed for determination of enzyme activity. There was a 34% inhibition of hepatic aminopyrine demethylase activity at 7 days after treatment, but a 1.49-fold induction at 91 days. After 1 day of feeding, there was a decrease in aryl hydrocarbon hydroxylase (AHH) activity to 36% of control values that further decreased to 64% on day 2. The AHH activity of the test animals was consistently above control levels throughout the rest of the first month. From days 2 through 91, there was a biphasic stimulation of dimethylnitrosamine demethylase activity, with an early component in the first week and a subchronic component at 2 and 3 mo. There was no consistent effect of PiB on

acetylaminofluorene hydroxylation. These results indicate that hamsters can adapt to PiB exposure and adequately regulate their liver enzyme systems. Since complex regulatory systems are involved, it is not possible to conclude from these results that PiB would have no effects on carcinogenesis in chronic experiments. (14 refs)

- 79-4422 Mutagenicity Test of Dyes Used in Cosmetics with the Salmonella/Mammalian-Microsome Test.** (Eng) Muzzall, J. M. (Dept. Biology, Georgia State Univ., Atlanta, GA 30303); Cook, W. L. *Mutat Res* 67(1): 1-8; 1979.

Thirty-seven dyes, including 3 anthraquinones, 22 azo compounds, 5 xanthenes, 5 fluorandiols, and 2 thioindigo dyes, were tested for mutagenicity in the Salmonella/mammalian-microsome test. Two frameshift histidine mutants (TA1537 and TA98) and two base-pair-substituted histidine mutants (TA1535 and TA100) of *Salmonella typhimurium* were employed. Of the 37 dyes screened, only the azo dye D&C Orange No. 17 was mutagenic. Both the spot test and the plate-incorporation assay indicated that D&C Orange No. 17 was mutagenic to three of the bacterial test strains. The mutagenic response of this dye was depressed by the addition of a rat liver microsomal fraction. Of the chemicals used to synthesize D&C Orange No. 17, β -naphthol was not mutagenic, but 2,4-dinitroaniline was mutagenic to the same *Salmonella* strains as D&C Orange No. 17. Dimethyl sulfoxide extracts of lipsticks of similar formula but with and without D&C Orange No. 17 were tested in the plate incorporation assay. Only those containing D&C Orange No. 17 were mutagenic, and the dye was mutagenic at concentrations consumed in normal daily use. (11 refs)

- 79-4423 Suppression of Naphthylamine Mutagenicity by Amaranth.** (Eng) Stoltz, D. R. (Health Protection Branch, Health and Welfare Canada, Tunney's Pasture, Ottawa, Ontario K1A 0L2, Canada); Stavric, B.; Iverson, F.; Bendall, R.; Klassen, R. *Mutat Res* 60(3): 391-393; 1979.

The mutagenicity of α - and β -naphthylamine (NA, a contaminant of FD & C Red No. 2 (Amaranth)) for *Salmonella typhimurium* strains TA1535 and TA100 was studied. β -NA was a potent mutagen, particularly for TA1534, in the presence of liver homogenate from Aroclor-induced rats. Although weakly positive with Aroclor-induced rat liver, α -NA was considerably more mutagenic for TA100 with uninduced hamster-liver activation. The mutagenic activity of 10 μ g β -NA was lost in the presence of 9.99 mg Amaranth (0.1% β -NA in Amaranth). Similar results were obtained with α -NA in Amaranth. The results indicate that the Ames Salmonella test cannot be used to screen for NA in Amaranth. (12 refs)

- 79-4424 Algal Oxidation of Aromatic Hydrocarbons: Formation of 1-Naphthol from Naphthalene by *Agmenellum quadruplicatum*, Strain PR-6. (Eng) Cerniglia, C. E. (Dept. Microbiology, Univ. Texas at Austin, Austin, TX 78712); Gibson, D. T.; Van Baalen, C. *Biochem Biophys Res Commun* 88(1): 50-58; 1979.

Agmenellum quadruplicatum strain PR-6, *Coccochloris elabens* strain 17A, and *Oscillatoria* species strain JCM, grown photoautotrophically in the presence of [¹⁴C]-naphthalene, produced six radioautographically detected metabolites. A large-scale biotransformation experiment with *A. quadruplicatum* led to the isolation and identification of 1-naphthol as the major reaction product. Preliminary evidence for the formation of cis-1,2-dihydroxy-1,2-dihydronaphthalene was also obtained. (12 refs)

- 79-4425 A Simplified Method of Separating and Determining Residues of Organochlorine Pesticides and Polychlorinated Biphenyls in Human Fatty Tissue. (Pol) Gorski, T. (Zaklad Toksykologii Sanitarnej, Panstwowy Zaklad Higieny, ul. Chocimska 24, 00-791 Warsaw, Poland); Syrowatka, T. *Rocz Panstw Zakl Hig* 30(1): 57-65; 1979.

A method for separating polychlorinated biphenyls (PCB's) from interfering organochlorine pesticides for the gas-chromatographic determination of PCB's in human fatty tissue is described. The organochlorine pesticides are separated from PCB's by dechlorination and oxidation. (11 refs)

- 79-4426 DNA Single Strand Breaks Caused by 2,2',5,5'-Tetrachlorobiphenyl and Its Metabolites. (Eng) Stadnicki, S. S. (Dept. Pathology and Experimental Pathology Unit, Regional Primate Res. Center, Univ. Wisconsin, Madison, WI 53706); Lin, F. S.; Allen, J. R. *Res Commun Chem Pathol Pharmacol* 24(2): 313-327; 1979.

The ability of tetrachlorobiphenyl (TCB) and its phenolic and arene oxide metabolites to cause single-strand breaks in the DNA of L-929 cells was studied. TCB, TCB-phenols, and TCB arene oxide were all able to induce single-strand breaks in DNA. However, the TCB arene oxide induced complete breakage of DNA at concentrations as low as 20 µg/ml, whereas TCB and TCB-phenols did not induce complete DNA breakage in concentrations less than 100 µg/ml. The data support the idea that polychlorinated biphenyl (PCB) epoxide may be involved in PCB carcinogenesis. (28 refs)

- 79-4427 Carcinogenic Azo Dyes. XI. Analysis of Biliary and Urinary Metabolites of

3'-Methyl-4-(methylamino) azobenzene in Rat. (Eng) Mori, Y. (Gifu Coll. Pharmacy, Mitahora-higashi 5-6-1, Gifu 502, Japan); Yamamoto, T.; Toyoshi, K. *Chem Pharm Bull (Tokyo)* 27(2): 379-385; 1979.

Metabolites of 3'-methyl-4-(methylamino) azobenzene (3'-Me-MAB) in rat bile and urine were analyzed quantitatively by reverse isotope dilution using [³H]3'-Me-MAB. [³H]3'-Me-MAB (46 mg/kg) in cottonseed oil was given po by stomach tube to male Wistar rats. The 24-hr urine and bile samples were hydrolyzed with β-glucuronidase/arylsulfatase. The hydrolyzed metabolites were extracted with chloroform or separated by chromatography. The N-demethylated metabolite, 3'-Me-MAB, and their azo-reduced metabolites (3-aminotoluene and 3-acetaminotoluene) were the major products detected in the bile. Products oxidized at the ring methyl group, and 3-aminobenzoic acid and 3-acetaminobenzoic acid were also detected in the bile. Metabolites retaining the azo linkage were excreted in very small quantities in the urine. Products oxidized at the ring methyl group formed 50% of the urinary metabolites (3-aminobenzoic acid, 3-acetaminobenzoic acid, 3-aminobenzyl alcohol, and 3-acetaminobenzyl alcohol) and the aryl hydroxylated ones, 30% (3-amino-6-hydroxytoluene and 3-acetamino-6-hydroxytoluene). These results indicate that 3'-Me-MAB metabolism in the rat involves oxidation of the ring methyl group. It is suggested that hydroxylation of the ring methyl group is one of the metabolic activation reactions for carcinogenic aminoazo dyes. (11 refs)

- 79-4428 Effect of Citric Acid on 3'-Methyl-4-dimethylaminoazobenzene Induced Decrease in Rat Liver pH. (Eng) Kitagawa, Y. (Dept. Biochemical Toxicology, Sch. Pharmaceutical Sciences, Showa Univ., 5-8, Hatanodai 1 chome, Sinagawa-ku, Tokyo 142, Japan); Kuroiwa, Y. *Chem Pharm Bull (Tokyo)* 27(2): 295-300; 1979.

The effect of citric acid on 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB)-induced decreases in blood and liver pH was studied in 7- and 10-wk old male Donryu rats. The livers of rats fed 0.06% 3'-Me-DAB in the diet with or without simultaneous citrate administration in drinking water (0.1%) showed small white nodules after 5 wk of treatment and these increased in number and size with prolonged feeding. The blood and liver pH remained within the normal range in rats fed a normal diet and citrate-treated water. Rats given 3'-Me-DAB plus citrate maintained normal pH values until the ninth week; after the 11th wk, pH values tended to decrease. N-Demethylase and azoreductase activities decreased in all groups that received 3'-Me-DAB, including those given only a single po dose (40 mg). Time-course examination of protein-bound dyes in the liver indicated that the amount of bound dyes peaked

earlier in rats receiving 3'-Me-DAB in the diet and citrate than in rats receiving 3'-Me-DAB without simultaneous citrate. The amount of 3'-Me-DAB bound to each liver protein fraction was similar in rats receiving 3'-Me-DAB plus citrate and rats receiving 3'-Me-DAB alone. It is concluded that citrate may not play a major role in the reduction of 3'-Me-DAB-induced hepatic injury. (11 refs)

- 79-4429 Isolation and Structure Determination of a Mutagenic Substance in L-Lysine Pyrolysate.** (Eng) Wakabayashi, K. (Shizuoka Coll. Pharmacy, 2-2-1, Oshika, Shizuoka-shi, Shizuoka 422, Japan); Tsuji, K.; Kosuge, T.; Takeda, K.; Yamaguchi, K.; Shudo, K.; Iitaka, Y.; Okamoto, T.; Yahagi, T.; Nagao, M.; Sugimura, T. *Proc Jpn Acad* 54(9): 569-571; 1978.

The isolation and identification of a mutagenic substance produced by pyrolysis of L-lysine is reported. A basic fraction of this pyrolysate showed mutagenic activity in *Salmonella typhimurium* strains TA100 and TA98 in the presence of S-9 mix. This fraction was separated into several active components, one of which was identified by crystallographic techniques as 3,4-cyclopentenopyrido(3,2- α)carbazole and was designated Lys-P-1. The lysine pyrolysate contained other unidentified mutagens. The mutagenicity of Lys-P-1 was as strong as that of benzo(a)pyrene and other carcinogenic polycyclic aromatic hydrocarbons under similar test conditions. (9 refs)

- 79-4430 High Pressure Liquid Chromatographic Determination of Aflatoxins in Corn.** (Eng) Pons, W. A. (U.S. Dept. Agriculture, Science and Education Admin., Southern Regional Res. Center, New Orleans, LA 70179). *J Assoc Off Anal Chem* 62(3): 586-594; 1979.

A high-pressure liquid chromatographic (HPLC) method for determining aflatoxins (AF's) in corn involves extraction with methanol-10% NaCl (4 + 1), pigment precipitation with zinc acetate, clean-up on a small silica gel (SG) column, AF resolution by normal-phase HPLC on a microparticulate SG column with water-saturated chloroform-cyclohexane-acetonitrile solvent, and AF detection by fluorescence on a SG-packed flow cell. In 5/6 samples containing aflatoxin B₁, B₂, G₁, and G₂, methanol-10% NaCl extracted more AF than chloroform-water, but in samples containing only B₁ and B₂, both solvents were equally effective. Agreement was good between HPLC and thin-layer chromatography for each solvent. (21 refs)

- 79-4431 Simultaneous Extraction and Fractionation and Thin Layer Chromatographic Determination of 14 Mycotoxins in Grains.** (Eng) Takeda, Y. (Div. Food, Natl. Inst. Hygienic Sciences, 1-18-1 Kamiyoga

Setagaya, Tokyo 158, Japan); Isohata, E.; Amano, R.; Uchiyama, M. *J Assoc Off Anal Chem* 62(3): 573-578; 1979.

An analytical method for the detection of 14 mycotoxins, including aflatoxins B₁, B₂, G₁, and G₂, is described. The mycotoxins are extracted with 20% sulfuric acid-4% potassium chloride-acetonitrile, isoctane, and then chloroform. The chloroform extract is cleaned up by silica gel column chromatography. The mycotoxins are eluted with chloroform-methanol or benzene-acetone-acetic acid, and the fractions are analyzed by thin-layer chromatography. This method has been applied to polished rice, rough rice, corn, wheat, and peanuts, and the detection limits have ranged from 10.0 to 800.0 $\mu\text{g/kg}$. (8 refs)

- 79-4432 Laser Fluorometric Determination of Aflatoxin B₁ in Corn.** (Eng) Diebold, G. J. (Dept. Chemistry, Brown Univ., Providence, RI 02912); Karny, N.; Zare, R. N.; Seitz, L. M. *J Assoc Off Anal Chem* 62(3): 564-569; 1979.

A two-step chromatographic separation, using both thin layer chromatography (TLC) and high-pressure liquid chromatography (HPLC), in conjunction with laser fluorometry permits extension of the detection limits of aflatoxin contamination in corn to 0.1 ppb ($\mu\text{g/kg}$) with a 26% root mean square variation. Aflatoxin recovery from TLC plates was linear from 10-1,000 picograms. Aflatoxin B₁ is converted to the more highly fluorescent B₂A derivative by treatment with 1N HCl. (13 refs)

- 79-4433 Thin Layer Chromatographic Determination of Aflatoxins, Ochratoxins, Sterigmatocystin, Zearalenone, Citrinin, T-2 Toxin, Diacetoxyscirpenol, Penicillic Acid, Patulin, and Penitrem A.** (Eng) Gimeno, A. (Piensos Compuestos Rosell S.A.-Picrosa, Laboratorio Control de Calidad, Francisco Moragas, 12, Manresa, Barcelona, Spain). *J Assoc Off Anal Chem* 62(3): 579-585; 1979.

A general method for determining 16 mycotoxins, including aflatoxin B₁ and aflatoxin G₁, in food products involves extraction and clean-up with the use of solvents of different pH, separation by thin-layer chromatography, and quantitation by the limit detection method. The minimum detectable aflatoxin concentration was 4-5 $\mu\text{g/kg}$. (24 refs)

- 79-4434 Degradation of Aflatoxin B₁ by Microorganisms Isolated from Rat Feces. Studies of the Degradation of Aflatoxins by Intestinal Microorganisms Part II.** (Jpn) Itoh, Y. (Dept. Biomedical Res.

Foods, Natl. Inst. Health, Shinagawa-ku, Tokyo 141 Japan); Morishita, Y.; Aibara, K. *Nippon Nogeikagaku Kaishi* 53(3): 97-102; 1979.

Bacteria isolated from a mixed culture originated from rat feces having aflatoxin B₁-degrading potency were examined in vitro for degrading aflatoxin B₁ after three-day incubation. They were composed of four strains of *Clostridium* species, three strains of *Bacteroides* species and three strains of *Catenabacterium* species, which were anaerobic, and a strain of *Pseudomonas aeruginosa*, a strain of *Proteus mirabilis*, three strains of *Streptococcus* species, and a strain of *Escherichia coli*, which were aerobic. *P. mirabilis* Y2 degraded 50.9% of the initial concentration of aflatoxin B₁ in 72 hr; three strains of *Catenabacterium* species, *P. aeruginosa* Y1, *Streptococcus* species Y5 degraded 25.0-35.5%, and the anaerobes other than *Catenabacterium* species, *Streptococcus* species Y4 and Y6, and *E. coli* Y8 degraded 12.4-22.0%. Combinations of *P. mirabilis* Y2 plus each strain of the *Streptococcus* species degraded 85.6-100% of aflatoxin B₁, and a combination of all strains of the aerobes degraded 78.8%. All other combinations degraded less than 54.0%. *P. aeruginosa* Y1 alone and the combinations of *P. aeruginosa* plus *P. mirabilis* Y2, *P. aeruginosa* Y1 plus *Streptococcus* species Y4, and a mix of all six strains formed a very small amount of a new substance from aflatoxin B₁. This substance has a bright-blue fluorescence and an R_f value of 0.39 on thin-layer chromatography plate, compared with 0.42 for aflatoxin B₁ and 0.34 for B₂. Formation of the new fluorescent substance seems to be concerned with *P. aeruginosa* Y1. (4 refs)

- 79-4435 The Formation of 2,3-Dihydro-2,3-dihydroxy Aflatoxin B₁ by the Metabolism of Aflatoxin B₁ In Vitro by Rat Liver Microsomes. (Eng) Neal, G. E. (Toxicology Unit, MRC Labs., Carshalton, Surrey SM5 4EF, England); Colley, P. J. *FEBS Lett* 101(2): 382-386; 1979.

The production of 2,3-dihydro-2,3-dihydroxy aflatoxin B₁ (AFB-dhd) from aflatoxin B₁ (AFB) in the presence of rat (adult male F344/TIF Lac) liver microsomes was studied. The metabolism of AFB by liver microsomes in phosphate or Tris buffer produced similar metabolites, except that a degradation product of AFB (Tris-diol) was present in the Tris incubations and absent from the phosphate incubations. Paralleling the absence of this metabolite from the phosphate incubations, there was an increased binding of AFB to the microsomes. Authentic AFB-dhd reacted with certain compounds having an amino group, both this reaction and the subsequent binding of AFB-dhd to microsomal protein being pH dependent. The results obtained were probably due to the pH-dependent formation of a dialdehydic phenolate ion from AFB-dhd which then underwent Schiff's base reaction with amino compounds. The reaction of AFB-dhd with microsomal protein did not

appear to inhibit the mixed function oxidase system. (7 refs)

- 79-4436 Induction of Sister-Chromatid Exchanges in Human Lymphocytes and Chinese Hamster Cells Exposed to Aflatoxin B₁ and N-Methyl-N-nitrosourea. (Eng) Thomson, V. E. (Medical Res. Council, Clinical and Population Cytogenetics Unit, Western General Hosp., Crewe Road, Edinburgh EH4 2XU, England); Evans, H. J. *Mutat Res* 67(1): 47-53; 1979.

The ability of aflatoxin B₁ (AFB₁: 10⁻³ to 10⁻⁶ M for 1 hr) to induce sister-chromatid exchanges (SCE's) in Chinese hamster ovary (CHO) cells and human lymphocytes was determined in the presence and absence of mixed function oxidases (S9 mix). CHO cells were also exposed to a graded series of doses (10⁻³ to 10⁻⁷ M) of N-methyl-N-nitrosourea, a powerful inducer of SCE's whose action was independent of the presence or absence of an S9 mix. The responses of the CHO and human cells to SCE induction by AFB₁ were closely correlated. SCE incidence increased with AFB₁ dose. In both cell systems, the addition of mixed function oxidases in the S9 mix markedly enhanced the action of AFB₁. (17 refs)

- 79-4437 Short-Term Effects of Aflatoxin B₁ on Serum Lipids in Subhuman Primates. (Eng) Bassir, O. (Dept. Biochemistry, Univ. Ibadan, Ibadan, Nigeria); Alozie, T. C. *Lab Anim* 13(2): 67-68; 1979.

The short-term response of serum lipids to aflatoxin B₁ (AFB, 60 or 100 µg/kg. ip) was studied in African green monkeys, patas monkeys, mona monkeys, and anubis baboons. Both doses of toxin significantly depressed serum lipid levels. In each species, the serum cholesterol, total phospholipids, and total lipids were lowered to different extents by the same dose of AFB. (11 refs)

- 79-4438 Note on Inactivation of Aflatoxin in Ammonia-treated Shelled Corn at Low Temperatures. (Eng) Nofsinger, G. W. (Northern Regional Res. Center, Agricultural Res., U.S. Dept. Agriculture, Peoria, IL 61604); Anderson, R. A. *Cereal Chem* 56(2): 120-121; 1979.

The inactivation of aflatoxin B₁ (AFB) in shelled corn by ammonia (NH₃) was studied. Corn adjusted to 17% moisture content and held at 30 C for 24 hr had a deep mahogany color after treatment with 1-1.5% NH₃. Addition of 1% NH₃ reduced the initial AFB content (266-896 ppb) by one-half or more in 60 days and to 100 ppb or lower after 179 days. Addition of 1.5% NH₃ reduced the

AFB content to less than one-third the initial level in 60 days and to less than 50 ppb after 179 days. During the first 90 days, these levels of inactivation were attained at temperatures seldom exceeding 0 C. The cause of a general increase in AFB content from days 8 to 59 is unknown. (5 refs)

- 79-4439 Chemical and Toxicological Studies in Bracken Fern, *Pteridium aquilinum* Var. *latiusculum*. IV. Surveys on Bracken Constituents by Mutagen Test. (Eng) Fukuoka, M. (Natl. Inst. Hygienic Sciences, Kamiyoga-1-chome, Setagaya-ku, Tokyo 158, Japan); Kuroyanagi, M.; Yoshihira, K.; Natori, S.; Nagao, M.; Takahashi, Y.; Sugimura, T. *J Pharmacobio Dyn* 1(5): 324-331; 1978.

The mutagenicities of >20 compounds isolated from bracken fern (*Pteridium aquilinum* var. *latiusculum*) were tested using *Salmonella typhimurium* strains TA100 and TA98. All 15 pterosins isolated from the bracken were non-mutagenic up to 1 mg/plate, but kaempferol, one of the phenolic constituents of bracken, was mutagenic to both strains in the presence of a fortified rat liver microsomal preparation (S-9 mix). A boiling water extract was non-mutagenic, but mutagenic activity was noted in one fraction after hesperidinase treatment. This activity was due primarily to kaempferol, the hydrolysis product of astragalin, and partly to another more water-soluble mutagen. Astragalin was not mutagenic without hesperidinase treatment. The acetone and ether extracts of bracken were also mutagenic, but only after hesperidinase treatment; the mutagenic extracts contained flavonol glucosides and astragalin and/or isoquercitrin. The toxic fraction of bracken that causes hemorrhage in calves was nonmutagenic before and after hesperidinase treatment. Food processing (treatment with 0.3% NaHCO₃, 1% NaHCO₃, or salt) removed or destroyed to some extent the bracken mutagens. Consequently, all the mutagenic potency and the pattern of distribution of the mutagenicity of these extracts cannot be attributable to free flavonols, kaempferol, and quercetin in the extracts. (38 refs)

- 79-4440 Long-Term Toxicity Study of Quillaia Extract in Mice. (Eng) Phillips, J. C. (British Industrial Biological Res. Assoc., Woodmansterne Road, Carshalton, Surrey, SM5 4DS, England); Butterworth, K. R.; Gaunt, I. F.; Evans, J. G.; Grasso, P. *Food Cosmet Toxicol* 17(1): 23-27; 1979.

The results of a long-term study of the effects of an extract of the bark of the quillaia (soapbark) tree in mice are presented. Quillaia extract is used as an emulsifier and foaming agent, particularly in soft drinks. A spray-dried aqueous extract of quillaia bark was prepared in such a manner that 100 parts by wt of bark yielded approx 15

parts of extract before drying. Groups of 48 male and 48 female TO mice were fed quillaia extract in mice. Groups of 48 male and 48 female TO mice were fed quillaia extract in the diet at levels of 0% (control), 0.1%, 0.5% or 1.5%, for 84 wk. The material had no adverse effect on the death rate or the incidence of histopathological findings, including tumors. However, there was a lower rate of body wt gain at the 1.5% dietary level, and there were isolated statistically significant differences between the treated and control animals, mainly at the 1.5% dietary level, in the hematological examinations and in some absolute and relative organ wts of both sexes. There were lower RBC counts in mice given 0.5% or 1.5% and a higher packed volume (PCV) in males given 1.5%, a lower PCV in females given 0.5%. It is concluded that, in mice, quillaia extract fed at levels up to 1.5% in the diet (approx 2.2 g/kg/day) did not exert a carcinogenic effect. The non-untoward-effect level from this study is considered to be 0.5% in the diet, giving an intake of approx 0.7 g quillaia extract/kg/day. (14 refs)

- 79-4441 Diazepam and Experimental Tumour Growth (Letter to Editor). (Eng) Guaitani, A. (Istituto di Ricerche Farmacologiche "Mario Negri", 20157 Milan, Italy); Carli, M.; Rocchetti, M.; Garattini, S. *Lancet* 1(8126): 1147-1148; 1979.

It was recently reported that diazepam (1 mg/kg ip) increases the growth of two experimental tumors in rats. Another benzodiazepine, oxazepam (2-32 mg/kg/day by stomach tube), did not influence the growth of transplanted Walker carcinomas in CD-COBS male rats or affect the survival time of the tumor-bearing animals. (2 refs)

- 79-4442 Determination of Environmental Polycyclic Aromatic Hydrocarbons by Liquid Chromatography. (Pol) Dutkiewicz, T. (Inst. Environmental Development, Center Environmental Protection, ul. Janka Krasickiego 2, 40-832 Katowice, Poland); Masny, N.; Ryborz, S.; Maslowski, J.; Grabka, A. *Chemia Analityczna* 24(1): 191-193; 1979.

A method for separating and isolating polycyclic aromatic hydrocarbons (PAH) in surface water, soil, and airborne particulates is described. The PAH are extracted with cyclohexane, separated by liquid chromatography, and analyzed spectrophotometrically at 210-470 nanometers. The detection limits for PAH in various environmental samples are 0.1-1.0 µg/cm³. The accuracy of the method for benzo(a)pyrene is ± 4% over the concentration range 0.125-5.0 µg/cm³. (no refs)

- 79-4443 Entropy Dominated High Performance Liquid Chromatographic Separations of Polynuclear

Aromatic Hydrocarbons. Temperature as a Separation Parameter. (Eng) Chmielowiec, J. (Canada Centre Mineral and Energy Technology, 555 Booth St., Ottawa, Canada K1A 0G1); Sawatzky, H. *J Chromatogr Sci* 17(5): 245-252; 1979.

The effect of temperature on the thermodynamic distribution behavior of a variety of polynuclear aromatic hydrocarbons (PAH) in a silica C₁₈/aqueous acetonitrile high pressure chromatographic system was studied. A temperature gradient of 2.6 C/min up to 50 C resulted in narrowing of the peaks and shortening of the separation time. As the temperature increased, the values of the capacity factors of the PAH decreased. Changes in temperature had different effects on the separation factors of different pairs of compounds: in some cases, there was a reversal of elution sequence; in other cases, the separation factors increased or decreased with an increase in temperature. The changes in elution sequences with temperature could be ascribed to domination by entropy effects. In most cases, the enthalpy term was larger than the entropy term (both were always negative), and temperature increases led to poorer separation. However, when the enthalpy term was smaller than the entropy term, temperature increases were beneficial for separation. (11 refs)

79-4444 Use of High-Performance Liquid Chromatography as a Clean-up Procedure in Analysis of Polycyclic Aromatic Hydrocarbons in Alcoholic Beverages. (Eng) Toussaint, G. (Unit Environmental Carcinogens, International Agency Res. Cancer, 150 cours Albert Thomas, 69372 Lyon Cedex 2, France); Walker, E. A. *J Chromatogr* 171: 448-452; 1979.

A technique in which high-performance liquid chromatography (HPLC) is used as a clean-up procedure in the analysis of polycyclic aromatic hydrocarbons in alcoholic drinks is described. Clean-up involves the use of 5- μ m particulates and a single HPLC stage. Extracts are obtained from alcoholic beverages by continuous liquid-liquid extraction, and the determination step involves gas chromatography on a packed column. The efficiency of the HPLC clean-up also permits the direct use of fluorimetric measurement, with HPLC separation on a reversed-phase column as a confirmatory test. (18 refs)

79-4445 Characteristics of DNA During In Vitro Binding of Polycyclic Aromatic Hydrocarbons. (Rus) Karamysheva, A. F. (Dept. Res. Carcinogenic Agents, Cancer Res. Center, Moscow, USSR); Kobliakov, V. A.; Mironov, N. M. *Biull Eksp Biol Med* 87(1): 19-21; 1979.

The effect of various activation systems on the binding of

polycyclic aromatic hydrocarbons (PAH) to DNA was studied. Incubation of DNA with labeled PAH in medium containing rat liver microsomes, I₂, or ascorbic acid + EDTA + FeSO₄ resulted in the formation of DNA breaks. With the microsome system, the plateau in the PAH binding curve was not due to reduction of the PAH metabolism rate to zero or to reduction in the number of binding sites on the DNA molecule. The plateau was due to an equilibrium between two opposite processes: the binding of PAH metabolites and their removal due to degradation of the DNA. (6 refs)

79-4446 Gas Chromatographic Analysis of Polycyclic Aromatic Hydrocarbons. (Eng) Beernaert, H. (Departement van Farmaco-toxicologie, Instituut voor Hygiene en Epidemiologie, Afdeling Voedingswaren, Juliette Wytsmanstraat 14, 1050 Brussels, Belgium). *J Chromatogr* 173(1): 109-118; 1979.

The use of high-resolution capillary columns for the separation of polynuclear aromatic hydrocarbons (PAH's) by gas chromatography resulted in at least 50% resolution of all PAH's with two to seven rings except 1,2-benz(a)anthracene and chrysene. The gas chromatographic detection of 5 nanograms of 10 PAH's indicated that the glass capillary column (16 m, 2.5% SE-52) allowed a detection limit of 0.5 nanogram. (25 refs)

79-4447 Survey of Some Market Basket Commodities for Polynuclear Aromatic Hydrocarbon Content. (Eng) Joe, F. L. (Div. Chemistry, Food and Drug Admin., Washington, DC 20204); Roseboro, E. L.; Fazio, T. *J Assoc Off Anal Chem* 62(3): 615-620; 1979.

The Food and Drug Administration multicomponent regulatory procedure was used to determine polynuclear aromatic hydrocarbon (PAH) levels in 24 foodstuffs. The procedure has a reliable quantitation limit of 2 ppb. Benzo(a)pyrene was detected in only one of the products, at a level of 3 ppb. Pyrene and/or fluoranthene occurred in 19 samples, at levels of < 1 ppb to about 75 ppb. Comparison with data obtained 10 yr ago revealed that the types and levels of PAH found are essentially unchanged. (19 refs)

79-4448 Effect of N₂ on the Mutagenic and Lethal Activities of ICR-170 in *Neurospora crassa*. (Eng) Whong, W. Z. (Cancer Res. Group, Thermo Electron Corp., Waltham, MA). *Mutat Res* 60(3): 301-312; 1979.

The nature of the N₂ effect on 2-methoxy-6-chloro-9-[3-(ethyl-2-chloroethyl)aminopropylamino]acridine.2HCl (ICR-170), ie, the greater mutagenic and lethal activities of

this agent in the presence of N_2 than O_2 , was studied at the adenosine 3 (*ad-3*) region of *Neurospora crassa*. The characteristics of the N_2 effect for ICR-170 were that (1) the N_2 effect with ICR-170 was displayed in conidia when N_2 was administered during, but not before or after, ICR-170 treatment; (2) the highly increased mutagenic and lethal activities of ICR-170 in the presence of N_2 were due to an anoxic condition rather than to the presence of N_2 per se; (3) the high lethal activity of ICR-170 under N_2 was largely due to increased cytoplasmic inactivation, (4) the N_2 effect was a general phenomenon at the *ad-3* region of *N. crassa*; and (5) the greater mutagenicity of ICR-170 in the conidia under N_2 was attributable to enhancement of the mutagenic activity of ICR-170 rather than to selective killing. The results indicate that the N_2 effect was not due to more extracellular oxidative degradation of ICR-170 molecules in the presence of O_2 or to a greater uptake of ICR-170 by conidia under N_2 , but to the inhibition of conidial respiration under an anoxic environment. (22 refs)

- 79-4449 Mutagenicity Studies of R-Amino Salt, a Metabolite of Amaranth (FD & C Red No. 2), in Mouse Lymphoma Cells Heterozygous at the Thymidine Kinase Locus and in the Rat Dominant Lethal Test. (Eng) Palmer, K. A. (Genetic Toxicology Branch, Div. Toxicology, Food and Drug Admin., Washington, DC 20204); Sheu, C. W.; Green, S. *Food Cosmet Toxicol* 17(1): 5-9; 1979.

The results of the thymidine kinase heterozygous mouse lymphoma assay demonstrated that the R-amino salt of amaranth (FD & C Red No. 2) induced a dose-related increase in mutation frequency compared with control values. Giant cells were present in the cultures after treatment, and a distinct color change was observed when the salt was dissolved in the tissue culture medium. In the rat dominant lethal test, the effect of the R-amino salt was not statistically significant. (19 refs)

- 79-4450 Mammary Cancer in Mice Receiving Weekly Subcutaneous Injections of Sesame Oil (Meeting Abstract). (Eng) Szepeswol, J. (Dept. Biological Sciences, Florida International Univ., Miami, FL); Fletcher, J.; Toro-Goyco, E. *Proc Am Assoc Cancer Res* 20: 56; 1979 (no refs)

- 79-4451 The Induction of Sister-Chromatid Exchanges by 9-Aminoacridine Derivatives. I. The Relation Between the Yield of SCE Induction and Cell Kinetics in Cultured Human Lymphocytes. (Eng) Gibas, Z. (Genetics Lab., Inst. Medical Biology, Medical Sch., Debinki 1, 80-211 Gdansk, Poland); Limon, J. *Mutat Res* 67(1): 93-96; 1979.

In cultured lymphocytes, the yield of sister chromatid exchanges (SCE's) induced by two 9-aminoacridine derivatives [1-nitro-9(2-dimethylamino-1-methylethylamino)acridine dihydrochloride and 1-nitro-9(3-isopropylaminopropylamino)acridine dihydrochloride] and mitomycin C (MC) was correlated with cell cycle duration. Cells dividing twice during 72 hr of culture showed a greater number of SCE's per cell cycle after treatment than cells dividing three times in the same period. This effect was not related to the ability of an SCE inducer to affect cell division. MC, which does not affect cell kinetics, gave results similar to those of the two derivatives. (13 refs)

- 79-4452 Effect of Methyl Substituents on the Triplet State Properties of Benz[a]anthracene. (Eng) Co, T. T. (Dept. Chemistry, Univ. Washington, Seattle, WA 98195); Kwiram, A. L. *Photochem Photobiol* 29(5): 1021-1023; 1979.

The zero-field splitting parameters for benz(a)anthracene (BA) and five of its methyl derivatives were calculated for molecular orbital theory and determined by EPR (electron paramagnetic resonance) spectroscopy. The phosphorescence spectra of the six compounds were similar. Each compound was well characterized by its optical detection of magnetic resonance (ODMR) signals. It is suggested that ODMR provides a means for identifying the reaction sites and different metabolites of this series of compounds. (24 refs)

- 79-4453 Cell-mediated Mutagenesis in Cultured Chinese Hamster Cells by Polycyclic Hydrocarbons: Mutagenicity and DNA Reaction Related to Carcinogenicity in a Series of Compounds. (Eng) Wigley, C. B. (Dept. Cellular Pathology, Imperial Cancer Res. Fund, Lincoln's Inn Fields, London W.C. 2, England); Newbold, R. F.; Amos, J.; Brookes, P. *Int J Cancer* 23(5): 691-696; 1979.

The mutagenicity and toxicity of benz(a)anthracene (BA), 3-methylcholanthrene (MC), and 7,12-dimethylbenz(a)anthracene (DMBA) were studied in a cell-mediated system using baby hamster kidney (BHK-21) cells to metabolize the hydrocarbons and V79/4 cells as the targets. Lethally x-irradiated BHK cells were plated at 2×10^7 cells/flask. Eighteen hours later, 3×10^6 V79/4 cells were plated onto the confluent BHK cells. Two hours later, the mixed cultures were treated with 3H -labeled hydrocarbon. The V79/4 cells were harvested after 48 hr and their DNA was analyzed by column chromatography. The chromatographic profiles of MC- and DMBA-exposed DNA were qualitatively similar to those published for mouse skin or mouse embryo cell DNA after in vitro treatment with the same compounds. When binding indices were calculated from the degree of

product binding and total metabolism over 48 hr, the values correlated well with the carcinogenicity of the compounds. This was not true when the total ^3H content of the DNA was used to calculate the binding index. The V79/4 cells were also assayed for cytotoxicity (percentage plating efficiency) and mutation to azaguanine resistance after 48 hr incubation with ^3H -labeled BA, MC, or DMBA. BA was nontoxic and nonmutagenic, while MC and DMBA were cytotoxic and induced high mutation frequencies. The carcinogenic compounds were equally mutagenic for a given extent of DNA reaction. It is concluded that differences in biological activity between carcinogenic polycyclic hydrocarbons may be explained in terms of the direction and rate of cellular metabolism and the effective doses of particular reactive metabolites that reach the target macromolecules. (23 refs)

79-4454 The Oncogenic Potential of Three Different 7,12-Dimethylbenz(a)anthracene Transformed C3H/10T1/2 Cell Clones at Various Passages and the Importance of the Mode of Immunosuppression. (Eng) Saxholm, H. J. (Inst. Pathology, Univ. Oslo, Rikshospitalet, Oslo 1, Norway). *Eur J Cancer* 15(4): 515-526; 1979.

The ability of 7,12-dimethylbenz(a)anthracene (DMBA)-transformed C3H/10T1/2 cells to induce malignant tumors in syngeneic immunosuppressed mice was investigated. In vitro three types of DMBA-transformed foci were found after 6-8 wk and were labeled I, II, and III. These foci constituted, respectively, 10%-15%, 70%, and 15% of the total number of transformed foci. Cells of types I, II, and III were assayed at varying cell culture passages and at three dose levels, ie, 10^4 , 10^5 or 10^6 cells per inoculum, with or without immunosuppression by antithymocyte serum globulin fraction. The studies were performed in male and female syngeneic C3H NCI mice, C3H Charles River mice, and nude, athymic female mice. Type I cells could not be established as tumors, type II and type III cells developed oncogenic potential only after several passages in culture. The oncogenic potential was especially pronounced in the type III cells. Also tested were different methods of immunosuppression of the animal against the expression of the oncogenic potential of DMBA transformed C3H/10T1/2 cells from type II and type III clones. Immunosuppression by antithymocyte serum globulin fraction was an effective method of preparing the syngeneic host so that cells with a low oncogenic potential would grow as tumors, whereas total body irradiation was not effective. For cells with a high oncogenic potential both methods of immunosuppression were sufficient. Admixing lethally irradiated cells in the cell inoculum slightly enhanced the tumorigenicity of cells with low oncogenic potential, and such addition was clearly effective for cells with a higher oncogenic potential, both for the antibody-treated and for the irradiated series. The findings, which were reproducible, emphasize the importance of immunosup-

pression by antithymocyte globulins for detecting in vitro transformed, weakly oncogenic cells. (32 refs)

79-4455 Carcinogenesis Testing of Saccharin. No Transformation or Increased Sister Chromatid Exchange Observed in Two Mammalian Cell Systems. (Eng) Saxholm, H. J. (Inst. of Pathology, Univ. Oslo, Rikshospitalet, Oslo 1, Norway); Iversen, O. H.; Reith, A.; Brogger, A. *Eur J Cancer* 15(4): 509-513; 1979.

Saccharin was assayed in two mammalian cell systems for possible transforming or mutagenic effects. In mouse embryo fibroblast C3H/10T1/2 cells, saccharin was without transforming effects over a large concentration range. In 7,12-dimethylbenz(a)anthracene-transformed, but nononcogenic C3H/10T1/2 type I cells, saccharin had a slight effect on transition to the growth pattern morphology of types II and III cells, but this was not accompanied by characteristic changes in plasma membrane structure, as studied by scanning electron microscopy. Saccharin did not induce any sister chromatid exchange in human lymphocytes. Addition of a metabolic activation system (S-9 mix) did not change the sister chromatid exchange pattern, as usually seen after 3,4-benzopyrene treatment. Based on these studies, saccharin is neither a carcinogen nor a mutagen. (21 refs)

79-4456 Effect of 7,8-Benzoflavone on the Frequency of Skin Tumors Induced by Polycyclic Aromatic Hydrocarbons. (Rus) Linnik, A. B. (Lab. Chemical Carcinogenesis, Cancer Res. Center, Moscow, USSR); Kobliakov, V. A. *Biull Eksp Biol Med* 87(5): 454-455; 1979.

The effect of 7,8-benzoflavone (BF) on polycyclic aromatic hydrocarbon (PAH)-induced carcinogenesis was investigated in CBA and C57BL \times CBA mice that were divided into nine groups. Animals in Groups 1, 3, 5, and 7 received 20 weekly skin applications of a benzene soln of 7,12-dimethylbenz(a)anthracene (DMBA), benzo(a)pyrene (BP), 6-methyl-BP, and 6-formyl-BP, respectively. Animals in Groups 2, 4, 6, and 8 received each PAH in combination with BF (Total BF dose was 100 μg in Groups 1 and 2 and 800 μg in Groups 3-8). Mice in Group 9 received skin applications of BF alone. The mice were followed for 12-15 mo. BF inhibited the carcinogenic effect of methyl-substituted PAH: the incidence of carcinomas in Groups 2 and 6 was 13.3% and 10.0%, compared with 75.0% and 63.3% in Groups 1 and 5, respectively. BF had hardly any effect on the carcinogenic properties of BP and 6-formyl-BP: the incidence of mice with carcinomas in Groups 4 and 8 was 73.3% and 3.3%, compared with 80.0% and 6.6% in Groups 3 and 7. (3 refs)

79-4457 Melanoma Induced in Albino Guinea Pigs. A Neoplastic Disease with a Pathogenetic Mechanism Similar to that of Humans (Meeting Abstract). (Eng) Pawlowski, A. (Dept. Medicine, Univ. Toronto, Toronto, Ontario, Canada); Haberman, H. F.; Aravindakshan Menon, I. *J Invest Dermatol* 72(5): 273; 1979 (no refs)

79-4458 The Tumor Promoter 12-O-Tetradecanoylphorbol-13-acetate Stimulates Lactate Production in BALB/c 3T3 Preadipose Cells. (Eng) O'Brien, T. G. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA 19104); Saladik, D.; Diamond, L. *Biochem Biophys Res Commun* 88(1): 103-110; 1979.

The possibility that the ability of the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) to stimulate lactate production in BALB/c 3T3 cells might be involved in its inhibitory effect on triglyceride accumulation in this system was explored. At 1.6×10^{-7} M, TPA reversibly inhibited the adipose conversion of BALB/c 3T3 preadipose cells and reversibly increased lactate production by these cells. The stimulation of lactate production required 4-7 days of treatment for an optimal effect. Once TPA was removed from the cultures, the rate of lactate production fell to control levels. The concentration dependence for the TPA-mediated stimulation of lactate production was similar to that for its inhibitory effect on adipose conversion. Exogenous lactate in the absence of TPA also inhibited adipose conversion. These results suggest that the ability of TPA to interfere with the normal pattern of glucose metabolism may be important in its inhibitory effect on triglyceride accumulation in these cells. (18 refs)

79-4459 Stimulation of Choline Incorporation in Cell Cultures by Phorbol Derivatives and Its Correlation with Their Irritant and Tumor-promoting Activity. (Eng) Kinzel, V. (Inst. Experimental Pathology, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany); Kreibich, G.; Hecker, E.; Suss, R. *Cancer Res* 39(7): 2743-2750; 1979.

A rapid increase in choline incorporation into phosphatidylcholine was observed when HeLa and other cell lines (monolayer or suspension cultures) were treated with low concentrations (10^{-9} to 10^{-8} M) of the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA). Higher TPA concentrations resulted in still earlier max incorporation rates. This effect of TPA was also observed when cells were preincubated with radioactive choline, thereby excluding the possibility that TPA enhanced only the permeability of the radioactive precursor. Metabolic inhibitors of RNA and protein synthesis had little effect on choline incorporation, suggesting a direct activation of the

phospholipid metabolism by TPA. Tosylphenylalanine-chloromethyl ketone, which has been shown to inhibit the inflammatory and tumor-promoting effect of TPA on the mouse ear, did not influence the choline response to TPA in HeLa cells. Colchicine sensitized HeLa cells to the effect of TPA, leading to a further increase in the choline incorporation rate. Cultivation of HeLa cells in the prolonged presence of TPA did not change their response to the phorbol ester. Using choline incorporation in HeLa cells as a standard assay, a large number of phorbol derivatives were tested at concentrations differing by several orders of magnitude. The lowest concentration of a particular phorbol ester required to elicit after a twofold increase in choline incorporation after 5 hr correlated with its irritant activity in mouse ear skin. Structurally different tumor promoters, such as mezerein, cantharidin, and anthralin, did not elicit a significant response in the standard HeLa cell assay. (45 refs)

79-4460 Induction of Differentiation in Human Promyelocytic Leukemia Cells by Tumor Promoters. (Eng) Rovera, G. (Wistar Inst. Anatomy and Biology, Philadelphia, PA 19104); O'Brien, T. G.; Diamond, L. *Science* 204(4395): 868-870; 1979.

The ability of some tumor promoters to induce differentiation was studied in HL-60 cells derived from the peripheral blood WBC of a woman with acute promyelocytic leukemia. Phorbol diester tumor promoters and the promoter mezerein converted cultured HL-60 cells into adherent, nonproliferating cells with many of the characteristics of macrophages; the conversion was very rapid and affected 100% of the HL-60 population. Other types of promoters, including anthralin, phenobarbital, and saccharin, did not have this effect. A major determinant of the cell response to the promoters appeared to be the target cell, ie, whether it was a unipotent or multipotent stem cell. The chemical structure of the promoter was also important in that only diterpene esters that are promoters in vivo induced adherence and differentiation in HL-60 cells. The results suggest that mezerein and similar compounds merit further investigation as antitumor drugs that may be able to force leukemia cells to differentiate into nonproliferating macrophagelike cells. (23 refs)

79-4461 Effects of Structural Changes on the Tumor-promoting Activity of Phorbol Myristate Acetate on Mouse Skin. (Eng) Van Duuren, B. L. (Lab. Organic Chemistry and Carcinogenesis, Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016); Tseng, S. S.; Segal, A.; Smith, A. C.; Melchionne, S.; Seidman, I. *Cancer Res* 39(7): 2644-2646; 1979.

4 α -Phorbol-9,9a-didecanoate (4 α -PPD), 4 α -phorbol-9-

myristate-9a-acetate (4a α -PMA) phorbol-9-myristate-9a-acetate-3-aldehyde (PAMA) were tested for skin-tumor-promoting activity in female ICR/Ha Swiss mice given an initiating dose of 7,12-dimethylbenz(a)anthracene (20 μ g). Each phorbol ester (10 μ g in 0.1 ml acetone) was applied 3x/wk. Both 4a α -PMA and -PPD were completely inactive, but PAMA produced 25 papillomas in 10/30 mice and squamous carcinomas in 2/30 mice. None of the three compounds produced any notable inflammatory response. In positive controls promoted with phorbol-9-myristate-9a-acetate, multiple papillomas developed in 30 mice and squamous carcinomas in 15. The effects of structural and stereochemical changes on tumor-promoting activity suggest that a primary interaction of the phorbol ester series is binding at specific sites on the plasma membrane. (20 refs)

79-4462 Phorbol Ester-induced Morphological Changes in Transformed Chick Fibroblasts: Evidence for Direct Catalytic Involvement of Plasminogen Activator. (Eng) Quigley, J. P. (D.pt. Microbiology and Immunology, State Univ. New York, Downstate Medical Center, Brooklyn, NY 11203). *Cell* 17(1): 131-141; 1979.

The involvement of the serine protease plasminogen activator (PA) in phorbol myristate acetate (PMA)-induced morphologic changes in normal chick embryo fibroblasts (CEF) and Rous sarcoma virus-transformed CEF (RSVCEF) was studied. PMA induced PA in both CEF and RSVCEF, and incubation of RSVCEF with PMA for as little as 18-36 hr resulted in the formation of dense cellular aggregates. This pattern of morphologic alterations was not seen in RSVCEF incubated without PMA or in CEF incubated with PMA. The morphologic alterations in RSVCEF were prevented by several protease inhibitors, including leupeptin, p-nitrophenylguanidobenzoate, soybean trypsin inhibitor, benzamidine, and diisopropylfluorophosphate. Other protease inhibitors, including trypsin, chymotrypsin, elastase, thrombin, and plasmin, did not prevent PMA-induced morphological alterations. The pattern of inhibition of PA activity correlated with the inhibition of morphologic changes in RSVCEF. Characterization of the serine enzymes in the culture fluid from PMA-treated cells indicated that PA was the serine protease responsible for the morphologic changes. Thus, PA itself can catalytically alter cellular behavior in culture independently of plasminogen, until now its only known natural substrate. (44 refs)

79-4463 Carcinogenic Polycyclic Aromatic Hydrocarbons in Digested Sludge from Wastewater Treatment Plants. (Cze) Hotar, Z. (II. ustav lekárske chemie a biochemie, Fakulta všeobecného lékařství, University Karlovy, U nemocnice 5, 128 53 Prague 2,

Czechoslovakia); Sula, J.; Kremen, J.; Brizova, E.; Voznakova, Z.; Vencl, J. *Cas Lek Cesk* 118(4): 110-114; 1979.

Sewage sludge from municipal wastewater treatment plants was analyzed by gas chromatography for polycyclic aromatic hydrocarbons. Chrysene (0.11-1.31 ppm), benzo(b)fluoranthene (1-5.78 ppm), benzo(e)pyrene (0.017-0.083 ppm), benzo(a)pyrene (0.012-0.27 ppm), perylene (0.05-0.045 ppm), dibenz(a,j)anthracene (0.025-0.154 ppm), indeno(1,2,3,c,d)pyrene (0.38-1.87 ppm), and benzo(g,h,i)perylene (0.015-0.098 ppm) were found. (22 refs)

79-4464 Analysis of Chromosome Aberrations and Sister-Chromatid Exchanges in Human Lymphocytes Exposed In Vitro to Hydergine. (Eng) Tsuchimoto, T. (Biological and Medical Res. Div., Sandoz Ltd., CH-4002 Basel, Switzerland); Matter, B. E.; Deyssenroth, H. *Mutat Res* 67(1): 39-45; 1979.

The ability of Hydergine (dihydroergotoxine mesylate, Sandoz) to induce chromosome damage and sister-chromatid exchanges (SCE's) in human lymphocytes in vitro was determined. For the chromosome aberration study, 10 lymphocyte cultures were established for each of six donors. These cultures were divided into five groups (2 cultures/group): negative control, positive control (caffeine, 0.5 mg/ml), and Hydergine (0.1, 0.25, or 0.5 μ g/ml). For the SCE examination, four cultures were established for each of eight donors: negative control, positive control (mitomycin C, 0.1 μ g/ml), and Hydergine (0.1 and 0.5 μ g/ml). The lymphocytes were cultivated for 72 hr, and they were exposed to the respective treatments during the final 24 hr. The results showed that Hydergine induced no chromosome damage in human lymphocytes in vitro. (8 refs)

79-4465 Adenylate Cyclase Activity of Normal and Transformed Fibroblasts in Culture. (Eng) Anderson, W. B. (Lab. Molecular Biology, NCI, NIH, Public Health Service, US Dept. Health, Education and Welfare, Bethesda, MD 20014); Jaworski, C. J. *Natl Cancer Inst Monogr* (48): 365-374; 1978.

Regulation of the adenylate cyclase (AC) activity of fibroblasts by intra- and extracellular agents was studied. A defective AC system was a common feature of several virally transformed fibroblast lines, as well as those transformed spontaneously or by 3-methylcholanthrene. However, the means by which AC activity was altered differed with the cell system and type of transformation. The AC activity of normal rat kidney (NRK) cells was activated by magnesium, manganese, fluoride, guanosine triphosphate (GTP), and cholera toxin and was inhibited by calcium, adenosine, unsaturated fatty acids, lysolecithin, and, possibly, insulin. The AC activity from NRK cells was also

markedly stimulated by both L-epinephrine and prostaglandin E₁, this hormone stimulation being dependent on the presence of GTP. Of substantial interest was the isolation from serum of a 60,000-mol-wt factor that selectively inhibited GTP- and GTP-hormone-stimulated NRK AC activity. The modulation of hormone responsiveness may play a critical role in the regulation of AC and, consequently, in the regulation of intracellular cyclic AMP levels. (61 refs)

- 79-4466** Effects of Carrageenan, PVP and Tumour-Bearer Serum on Immunity Induced by Excision or Mitomycin C-treated Tumour Cells in Mice. (Eng) Kearney, R. (Dept. Bacteriology, Univ. Sydney, Sydney, N.S.W. 2006, Australia); Wu, R. L.; Orr, F. *Br J Cancer* 39(6): 648-658; 1979.

Two methylcholanthrene-induced syngeneic murine fibrosarcomas (H1 and H2) were studied to determine whether these tumors produce specific immunity after either the administration of mitomycin-C-treated tumor cells or the surgical removal of tumor isografts, and to determine whether the administration of carrageenan (Cg) altered the immunity induced by these methods. These tumors were found not to share major tumor-specific transplantation antigens. H2 appeared more immunogenic than H1. In contrast to H1, immunity induced by H2 was not affected by Cg, nor was its growth in Cg-treated normal mice augmented. Postoperative ip injections of Cg abolished the weak anti-H1 immunity produced by H1 tumor excision. Furthermore, the subsequent growth of the H1 tumor challenge in the Cg-treated immune mice was significantly greater than the augmented growth in Cg-treated normal mice. The prior administration of the macrophage-stabilizing agent polyvinylpyrrolidone (PVP) to immune mice significantly reduced the augmenting effect of Cg. The growth-promoting effect of Cg on a secondary H1 tumor challenge in mice immunized by tumor excision was abolished by 10⁶ MCT-H1 cells injected sc before Cg. In contrast to the immunity induced by tumor excision, Cg did not abolish the immunity induced by the injection of MCT-H1 cells. Passive administration of H1 tumor-bearer serum (TBS) did not enhance the growth of H1 cells in normal mice, nor did TBS abrogate the specific cell-mediated immunity (CMI) induced in vivo by MCT-H1 cells. However, TBS administered to Cg-treated, MCT-H1-immune mice abolished tumor immunity. We propose that TBS does not inhibit CMI in vivo provided that macrophages remain functional, but may do so when macrophages are rendered defective by antimacrophage agents or by products of neoplastic cells. Increasing the levels of specific effector cells can override the inhibiting effects of TBS, even when defective macrophages are present. (32 refs)

- 79-4467** Persistent Binding of 3-Methylcholanthrene to Mouse Lung DNA and Its Correlation with

Susceptibility to Pulmonary Neoplasia. (Eng) Eastman, A. (Vermont Regional Cancer Center, Univ. Vermont Coll. Medicine, Burlington, VT 05405); Bresnick, E. *Cancer Res* 39(7, part 1): 2400-2405; 1979.

The persistent binding of (³H)-3-methylcholanthrene (MCA, 250 μ Ci, iv) to lung and liver DNA was studied using four strains of male mice (A/J, C3H/HeJ, DBA/2J, and C57BL/6J). Radioactivity derived from MCA was associated with DNA from both tissues in all strains, removal of the bound radioactivity varying with tissue and strain and as a function of time, with appreciable levels of MCA still detectable by 28 days. The lung levels of nucleoside-bound adducts were higher than the liver levels at all times and in all strains. In all strains, some radioactivity was still bound to the lung DNA, but not the liver DNA, at 28 days. The levels bound to lung DNA were highest at all times in the A/J mice, the A/J strain being more susceptible than the other three strains to pulmonary neoplasia. Thus, the persistence of DNA-bound radioactivity appeared to correlate with the susceptibility of the lung to polycyclic aromatic hydrocarbon-induced carcinogenesis. In all preparations, the radioactivity eluted from Sephadex LH-20 with the column void volume or with underivatized nucleosides. Tissue, but not strain differences, were observed in these chromatographic profiles. The predominance of these early-eluting peaks in liver, rather than lung, suggests that they may represent non-carcinogenic lesions. This radiolabeled material remains uncharacterized. (39 refs)

- 79-4468** Inhibition of Chemical Transformation in C3H/10T1/2 Cells by Protease Inhibitors. (Eng) Kuroki, T. (Dept. Cancer Cell Res., Inst. Medical Sciences, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Drevon, C. *Cancer Res* 39(7): 2755-2761; 1979.

Five protease inhibitors, antipain, chymostatin, elastatinal, leupeptin and pepstatin, were studied for their effects on chemical transformation in C3H/10T1/2 cells and mutagenesis in V79 Chinese hamster cells. These inhibitors blocked 3-methylcholanthrene (MCA)-induced transformation in C3H/10T1/2 cells when present at a nontoxic dose (50 μ g/ml) during the 6-wk transformation course or when added 1 wk after MCA. The simultaneous addition of these inhibitors during the first 48 hr of carcinogen treatment did not inhibit transformation. With the exception of elastatinal, the protease inhibitors reduced the saturation density of the transformed cells and suppressed their growth. The inhibitors were not lethal to the cells. The growth suppression was reversible. The transformed cells exhibited a marked heterogeneity in their sensitivity toward protease inhibitors, suggesting that the proteases involved in transformation may not be a single species. Mutations, as determined by 8-azaguanine resistance and/or ouabain resistance, were induced in V79 Chinese hamster-cells by

N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) and UV irradiation in the direct assay and by MCA in the cell-mediated assay. The protease inhibitors were added at a concentration of 50 $\mu\text{g/ml}$ during the treatment and the expression period. Little or no effects on mutation frequency were observed at a comparative dose, with the exception of pepstatin, which reduced 8-azaguanine resistance but not ouabain resistance induced by MNNG. (36 refs)

79-4469 Enhanced Mutagenesis of the Flame Retardant 2,3-Dibromopropyl Phosphate (TRIS) by Skin Microsomal Enzymes (Meeting Abstract). (Eng) Bickers, D. R. (Div. Dermatology, Case Western Reserve Univ., Cleveland, OH); Rosenkranz, H. S. *J Invest Dermatol* 72(5): 271-272; 1979 (no refs)

79-4470 Increased Inducibility of Aryl Hydrocarbon Hydroxylase (AHH) by Methylcholanthrene (MC) in the Active Phases of the Hair Cycle in Mice (Meeting Abstract). (Eng) Manil, L. (Lab. Chimie Medicale-Lab. Dermatologie, Univ. de Liege, Institut de Pathologie, Liege, Belgium); Van Cantfort, J.; Gielen, J. E.; Lapiere, C. M. *J Invest Dermatol* 72(5): 276; 1979 (no refs)

79-4471 Autoradiographic Studies of [^3H]-Prostaglandin $\text{F}_{2\alpha}$ into the Nuclei of Epidermal Neoplastic Cells (Meeting Abstract). (Eng) Lupulescu, A. P. (Dept. Dermatology, Wayne State Univ., Detroit, MI). *J Invest Dermatol* 72(5): 279-280; 1979 (no refs)

79-4472 The Ontogeny of Nuclear Aryl Hydrocarbon Hydroxylase (Meeting Abstract). (Eng) Nunink, J. (Dept. Biochemistry, Univ. Vermont, Burlington, VT); Bresnick, E. *Clin Res* 26(4): 640A; 1978 (1 ref)

79-4473 No Neoplastic Alteration of Metabolically Competent Rat Liver Cells In Vitro by Chemical Carcinogens: 3-Methylcholanthrene, Dimethylnitrosamine and Natulan. (Eng) Thust, R. (Inst. Pathology, Medical Acad., Erfurt, E. Germany); Neupert, G.; Kleeberg, U. *Cancer Biochem Biophys* 3(2): 1-6; 1978.

A rat liver cell line (RL-19) obtained by trypsinization of pooled livers from three 10-day-old Wistar rats was analyzed for aryl hydrocarbon hydroxylase (AHH) and dimethylnitrosamine demethylase activity. AHH activity was produced at a rate of about 14.5 picomoles of 3-

hydroxybenzo(a)pyrene/min/mg protein. This activity was not inducible by 3-methylcholanthrene or by phenobarbital, and it was independent of passage number. From passages 45 to 59, the mean demethylase activity produced was about 1.08 nanomoles (nmol) of HCHO/min/mg protein, but it decreased to 0.64 nmol HCHO/min/mg protein at the 131st passage. RL-19 cells were treated with 3-methylcholanthrene (0.5-1.0 $\mu\text{g/ml}$), dimethylnitrosamine (100-400 $\mu\text{g/ml}$), or Natulan (50 $\mu\text{g/ml}$), for 7-10 days. No neoplastic changes were observed in the cultures over the next 6 mo, as revealed by morphological investigations, soft agar assays, and transplantation experiments. It is suggested that metabolic competence for carcinogen activation is only one prerequisite for neoplastic alteration in vitro and that RL-19 cells are resistant to the action of carcinogens in spite of their metabolic competence. (34 refs)

79-4474 Effect of Phenobarbital and 3-Methylcholanthrene on Decrease in pH in Liver Induced by 3'-Methyl-4-dimethylaminoazobenzene, of Partial Hepatectomy and Hepatotoxic Agents. (Eng) Kitagawa, Y. (Dept. Biochemical Toxicology, Sch. Pharmaceutical Sciences, Showa Univ., Showa, Japan); Kuroiwa, Y. *Chem Pharm Bull (Tokyo)* 27(3): 586-591; 1979.

The effects of 3-methylcholanthrene (3-MC, 20 mg/kg, ip) and phenobarbital (PB, 80 mg/kg/day for 1 or 3 days, ip) on the decreases in intra- and extracellular hepatic pH (pHi and pHe, respectively) induced by 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB, 0.06% of the diet) were studied using male Donryu rats. In rats given 3-MC or PB alone, pHe and pHi remained within normal limits. In rats on the 3'-Me-DAB diet, pretreatment with 3-MC or PB prevented the decrease in pH seen in the first and fifth wk of 3'-Me-DAB feeding. There was no decrease in pHe or pHi in the regenerating liver of partially hepatectomized rats on the control diet. However, in partially hepatectomized rats on the 3'-Me-DAB diet, pHi in the regenerating liver was significantly decreased. In rats given 50 ppm diethylnitrosamine in the drinking water, liver nodules developed but pHe and pHi decreased only slightly. The administration of bromobenzene effected no significant decrease in pHe or pHi, but both pHe and pHi decreased with increased dosage of carbon tetrachloride (16 refs)

79-4475 Radiation-induced Oxidation of Benzo(a)pyrene. (Eng) Gibson, T. L. (Div. Biochemistry, Dept. Human Biological Chemistry and Genetics, Univ. Texas Medical Branch, Galveston, TX 77550); Smith, L. L. *J Org Chem* 44(11): 1842-1846; 1979.

The isolation and identification of oxidation products recovered from mutagenic preparations of oxidized ben-

zo(a)pyrene (BP) are described. The oxidation of BP induced by ^{60}Co γ radiation gave over two dozen products, half of which were identified. The products included three isomeric 7,8,9,10-tetrahydro-BP-7,8,9,10-tetraols, 9,10-dihydro-BP-trans-1,9,10-diol, a 7,8-dihydro-BP-7,8-diol, a 4,5-dihydro-BP-4,5-diol, BP-1,6-dione, BP-3,6-dione, BP-6,12-dione, 9-(2'-formylphenyl)phenalen-1-one, and, tentatively, benzo(a)pyren-3-ol, -6-ol, and -9-ol. These results establish that air oxidation of BP yields products similar to those found as mammalian metabolites, with oxidative attack at the K region, bay region, and the 6 position. Some of the air products were weakly mutagenic toward *Salmonella typhimurium* test strains. (22 refs)

- 79-4476 Chemical Mutagenesis by Benzo[a]Pyrene in *Escherichia coli* in the Absence of Any Activating Agents. (Eng) Hass, B. S. (Div. Biological and Medical Res., Argonne Natl. Lab., 9700 South Cass Ave., Argonne, IL 60439); Webb, R. B.; Gambill, T. B. *Mutat Res* 60(3): 395-399; 1979.

Continuous culture techniques were used to study the mutagenicity of benzo(a)pyrene (BP) for *Escherichia coli* B(s-1) in the absence of exogenous activation. Bacterial colonies that appeared after phage T5 challenge were considered T5-resistant mutants. A linear relationship was observed between BP concentration ($1.5 \times 10^{-6}\text{M}$) and the mutation rate, expressed as mutant frequency/day or as mutant frequency/generation. The BP-induced mutation rate was 30 mutants/ 10^8 cells/day/ μM BP, or 5.2 mutants/ 10^8M cells/generation/ μM BP. Wild-type *E. coli* strain B and the *recA* mutant WP10 showed no mutational response to BP under the conditions employed. (13 refs)

- 79-4477 Inactivation of SV40 Replication by Derivatives of Benzo(a)pyrene. (Eng) Chang, G. T. (Franklin McLean Memorial Res. Inst., Univ. Chicago, Chicago, IL 60637); Harvey, R. G.; Hsu, W. T.; Weiss, S. B. *Biochem Biophys Res Commun* 88(2): 688-695; 1979.

The effect of benzo(a)pyrene (BP) derivatives on the replication of simian virus 40 (SV40) was examined. When SV40-infected African green monkey kidney cell lines were exposed to benzo(a)pyrene-7,8-dihydrodiol or anti-benzo(a)pyrene-7,8-dihydrodiol-9,10-epoxide, progeny virus formation was inhibited. Alkylation of SV40 DNA with anti-BPDE inhibited the infectivity of this viral DNA; however, the inactivation did not follow a single-hit mechanism. Studies of [^3H]thymidine incorporation indicated that SV40 DNA synthesis is markedly impaired for the first 12 hr following BPDE treatment; 24-36 hr later, however, SV40 DNA synthesis is almost normal. These data suggest that the inhibition of SV40 DNA synthesis by

BP derivatives is reversible and that the observed reduction in viral titer requires some other explanation. (14 refs)

- 79-4478 Comparative Reactivities of Diolepoxide Metabolites of Carcinogenic Hydrocarbons with ΦX174 Viral DNA. (Eng) Hsu, W. T. (Dept. Biochemistry, Franklin McLean Memorial Res. Inst., Chicago, IL 60637); Lin, E. J.; Fu, P. P.; Harvey, R. G.; Weiss, S. B. *Biochem Biophys Res Commun* 88(1): 251-257; 1979.

The ΦX174 DNA assay system was used to determine the comparative reactivities with nucleic acid of the diol epoxides of a series of polycyclic aromatic hydrocarbons varying in carcinogenic potency. The infectious ΦX174 viral DNA was exposed to the hydrocarbon derivative for 10 min, and the infectivity of the treated DNA was assayed by incubation with *Escherichia coli* spheroplasts and plating on agar plates, with *E. coli* HF4714 being used as an indicator of plaque formation. The bay region diol epoxides of benzo(a)pyrene, chrysene, and dibenz(a,h)anthracene, implicated as the ultimately active carcinogenic metabolites of the parent hydrocarbons, exhibited potent viral inhibitory activity. On the other hand, no correlation was evident between viral inhibitory activity and the location of the diol epoxide function in a bay region or the theoretically calculated β -delocalization energies of the carbonium ion arising from opening the epoxide ring. Benzo(a)pyrene and triphenylene compounds that did not inactivate viral DNA to a significant degree had higher β delocalization values than a chrysene derivative that was one of the most active compounds tested. The significance of these findings are discussed in relation to the bay region theory of carcinogenesis. (28 refs)

- 79-4479 The Effects of Cytochrome P-450-448 Inhibitors on the Binding of Benzo(a)pyrene and Derivatives to DNA upon Microsomal Activation. (Eng) Liu, W. I. (Dept. Biochemistry, Univ. Tennessee, Center Health Sciences, Memphis, TN 38163); Sloane, N. H. *Xenobiotica* 9(3): 165-171; 1979.

The effects of α -naphthoflavone and 1-benzylimidazole, two different types of cytochrome P-450-448 inhibitors, on the binding of benzo(a)pyrene (BP) and several 6-substituted derivatives of BP to DNA were investigated. [^3H]BP and its 6-substituted derivatives were covalently bound to calf thymus DNA upon reaction with microsomal preparations from female Holtzman rats pretreated with 3-methylcholanthrene in the presence of NADPH. α -Naphthoflavone and 1-benzylimidazole inhibited the binding of BP to DNA by >80%. In the presence of these inhibitors, 6-hydroxymethylbenzo(a)pyrene, 6-methylbenzo(a)pyrene, and 6-formylbenzo(a)pyrene showed varying degrees of inhibition of binding to DNA depending upon

the inhibitor employed. Polyguanylic acid was the most effective substrate for the binding of each activated polynuclear aromatic hydrocarbon; polyadenylic acid and DNA demonstrated essentially equivalent binding. (31 refs)

- 79-4480 Benzo(a)pyrene-7,8-dihydrodiol: Selective Binding to Single Stranded DNA and Inactivation of Φ X174 DNA Infectivity.** (Eng) Hsu, W. T. (Dept. Biochemistry, Franklin McLean Memorial Res. Inst., Chicago, IL 60637); Sagher, D.; Lin, E. J.; Harvey, R. G.; Fu, P. P.; Weiss, S. B. *Biochem Biophys Res Commun* 87(2): 416-423; 1979.

The direct binding of benzo(a)pyrene-7,8-dihydrodiol (7,8-BPD) to single- and double-stranded Φ X174 DNA was studied. Treatment of the DNA with 7,8-BPD resulted in a max inactivation of viral DNA infectivity of 60%-70%, whereas trans-7,8-dihydroxy-anti-9, 10-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (anti-BPDE) caused a max inhibition of 99.8%. Although anti-BPDE reacted with single- and double-stranded DNA's and with RNA, 7,8-BPD bound extensively only to single-stranded DNA. Among the synthetic polymers studied, poly dI and denatured poly dG were most reactive with 7,8-BPD. Anti-BPDE reacted extensively only with poly dC and those polymers containing guanine. The cis stereoisomers of 7,8-BPD and the benz(a)anthracene (BA) 1,2- and 3,4-dihydrodiols were also effective inhibitors of viral replication, and weak activity was shown by 9,10-H₄BPD and 10-HO-H₄BP. No significant activity was exhibited by the BP 4,5- and 9,10-dihydrodiols, BA-10,11-dihydrodiol, benzo(e)pyrene-9,10-dihydrodiol, or 9-HO-BP. It is suggested that the active dihydrodiols may favor a relatively facile dissociation, with loss of a hydroxyl group and formation of a particularly stable benzylic or allylic carbonium ion. The data suggest that the diol and diol epoxide derivatives recognize different binding sites in nucleic acids and that the diol derivative may play an important role in the mutagenesis and carcinogenesis induced by polycyclic aromatic hydrocarbons. (24 refs)

- 79-4481 Modulation of the Cell Cycle of Cultured Mouse Liver Cells by Benzo(a)pyrene and Its Derivatives.** (Eng) Bartholomew, J. C. (Lab. Chemical Biodynamics, Lawrence Berkeley Lab., Univ. California, Berkeley, CA 94720); Pearlman, A. L.; Landolph, J. R.; Straub, K. *Cancer Res* 39(7): 2538-2543; 1979.

Alterations in cell cycle distributions caused by chemical carcinogens were monitored by flow cytometry. Two closely derived mouse liver cell strains growing in culture were studied with regard to the effect of benzo(a)pyrene (BP) and three BP derivatives on DNA synthesis. The derivatives were (\pm)-trans-7 α ,8 β -dihydroxy-7,8-dihydro-

benzo(a)pyrene, (\pm)-7 α ,8 β -dihydroxy-9 β ,10 β -epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (diol-epoxide), and 7,8,9,10-tetrahydroxy-7,8,9,10-tetrahydrobenzo(a)pyrene. One of the two cell strains, NMuLi clone 7, was not highly inducible for aryl hydrocarbon monooxygenase, which converts the parent compound into the derivatives. The second strain, NMuLi clone 8, was highly inducible for the enzyme. The high level of metabolic activity was correlated with an increased sensitivity to the cytotoxicity of the parent compound. However, both strains were equally sensitive to the diol-epoxide. Flow cytometry analysis and measurements of [³H]thymidine incorporation into DNA showed that the diol-epoxide increased the number of cells involved in DNA synthesis but that the DNA synthesis rate was greatly reduced. BP and \pm (trans)-7 α ,8 β -dihydroxy-7,8-dihydrobenzo(a)pyrene had this same effect on NMuLi clone 8 but not on NMuLi clone 7. The perturbations caused by the diol-epoxide took place within one cell cycle, and the cell did not traverse a second cycle. Kinetic modeling studies indicated that the cell cycle perturbations caused by the diol-epoxide are consistent, with the rate of traverse of S being slower than other phases of the cell cycle, and that the DNA synthesis rate decreases as the cells move through S. (46 refs)

- 79-4482 A Comparison of Benzo(a)pyrene-4,5-epoxide Hydrase Activity in Hamster Embryo Cells, Hepatocytes and Livers Using High-Pressure Liquid Chromatography.** (Eng) Raineri, R. (Chemical Carcinogenesis Program, NCI, Frederick Cancer Res. Center, Frederick, MD 21701); Pooley, J. A.; Hillesund, T.; Pienta, R. J. *J Liquid Chromatogr* 2(4): 577-587; 1979.

High-pressure liquid chromatography (HPLC) was used to compare the benzo(a)pyrene-4,5-epoxide (BP-4,5-epoxide) hydrase activities of intact Syrian golden hamster hepatocytes and embryo cells and homogenates of these cells, as well as those of adult liver. The product of the epoxide hydrase (EH) reaction, trans-4,5-dihydro-4,5-dihydroxybenzo(a)pyrene (BP-4,5-diol), was isolated by HPLC with a Waters Bondapak C₁₈/Corasil column and acetonitrile-water as the mobile phase. It was found that 266 nanomoles of BP-4,5-diol/10⁶ cells were produced by the hepatocytes, but no diol was formed by the embryo cells. EH activity in the intact hepatocytes was eightfold greater than that in the hepatocyte homogenates. (16 refs)

- 79-4483 The Rabbit Pulmonary Monooxygenase System. Catalytic Differences Between Two Purified Forms of Cytochrome P-450 in the Metabolism of Benzo(a)pyrene.** (Eng) Wolf, C. R. (Lab. Pharmacology, National Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); Smith, B. R.; Ball, L. M.; Serabjit-Singh, C.; Bend, J. R.; Philpot, R. M. *J Biol Chem* 254(9): 3658-3663; 1979.

The metabolism and activation of the pulmonary carcinogen benzo(a)pyrene (BP) were studied in reconstituted rabbit pulmonary monooxygenase (RRPM) systems. Two forms of purified pulmonary cytochrome P-450 (forms I and II) were compared. Both forms catalyzed the metabolism of BP at similar rates in the RRPM systems. Seventy percent of the metabolites produced by form I chromatographed with 3-hydroxy-BP and only 1.5% chromatographed with 9-hydroxy-BP. Of the metabolites produced by form II, 40%-50% chromatographed with BP 9,10-diol or 9-hydroxy-BP and 30%-40% chromatographed with 3-hydroxy-BP. Formation of the 4,5- or 7,8-dihydrodiol metabolites accounted for <5% of the total produced by either cytochrome. The metabolism of BP by cytochrome II was inhibited by α -naphthoflavone ($K_i = 8.2 \times 10^{-9}$ M) in a manner that did not affect the profile of the metabolites produced. Metabolism by cytochrome I was not significantly inhibited by α -naphthoflavone at a concentration of 10^{-4} M. Cytochrome II, unlike cytochrome I, catalyzed the metabolism of BP to products that bound covalently to calf thymus DNA. (60 refs)

- 79-4484** Carcinogen Activation by Human Uterine Enzymes. (Eng) Tsibris, J. C. (Dept. Obstetrics and Gynecology, Univ. Florida Coll. Medicine, Gainesville, FL 32610). *Ann Clin Lab Sci* 9(3): 236-242; 1979.

Adult human ovaries, uterus, and placenta were assayed for benzo(a)pyrene (BP)-metabolizing enzymes by high-pressure liquid chromatography. Microsomal proteins from a human placenta, an inflamed ovary (oophoritis), and a noncancerous myometrium metabolized BP. All three tissues contained uridine diphosphate (UDP)-glucuronosyl transferase, and the ovarian and myometrial tissues contained UDP-glucuronate transferase. It is hypothesized that environmental carcinogens (eg, cigarette smoke, polycyclic and polyhalogenated hydrocarbons) induce special forms of cytochrome P-450 monooxygenases and related enzyme systems that activate endogenous or prescribed estrogens and nonsteroid antiestrogens to tumor initiators and/or promoters in estrogen-dependent gynecologic tissues. The role of estrogen receptors is perceived as that of a homing device that transports the activated estrogen to a specific site(s) on chromatin. (31 refs)

- 79-4485** Carcinogenicity of 2-Hydroxybenzo(a)pyrene and 6-Hydroxybenzo(a)pyrene in Newborn Mice. (Eng) Chang, R. L. (Dept. Biochemistry and Drug Metabolism, Hoffman-La Roche Inc., Nutley, NJ 07110); Wislocki, P. G.; Kapitulnik, J.; Wood, A. W.; Levin, W.; Yagi, H.; Mah, H. D.; Jerina, D. M.; Conney, A. H. *Cancer Res* 39(7): 2660-2664; 1979.

Benzo(a)pyrene (BP), 2-hydroxybenzo(a)pyrene (2-

HOBP), and 6-hydroxybenzo(a)pyrene (6-HOBP) were tested for tumorigenicity by ip injection into newborn Swiss Webster mice. The mice were treated sequentially with 200, 400, and 800 nanomoles (nmol) of compound at age 1, 8, and 15 days and were killed at age 24 wk. 2-HOBP caused about fourfold more pulmonary tumors than BP; 6-HOBP had little or no tumorigenic activity. Newborn mice treated with 2-HOBP, BP, and 6-HOBP had a 98%, 81%, and 11% incidence, respectively, of pulmonary adenomas, with an av of 24, 6.4, and 0.11 adenomas per mouse. In the control group, 7.5% of the animals had pulmonary adenomas, with an av of 0.08 adenoma per mouse. When 25, 50, or 100 nmol of BP or 2-HOBP was applied to mouse skin once every 2 wk for 60 wk, both compounds had about the same carcinogenic activity. These results demonstrate the importance of evaluating the carcinogenic potential of chemicals in more than one tumor system. BP and 2-HOBP were tested for mutagenicity in two *Salmonella typhimurium* strains and in Chinese hamster V79 cells in the presence of hepatic microsomes from rats pretreated with Aroclor 1254. The products formed during the metabolism of 2-HOBP or BP by liver microsomes had significant mutagenic activity. (22 refs)

- 79-4486** Dose Response for Benzo(a)pyrene Adducts in Mouse Epidermal DNA. (Eng) Pereira, M. A. (U.S. Environmental Protection Agency, 26 West St. Clair St., Cincinnati, OH 45268); Burns, F. J.; Albert, R. E. *Cancer Res* 39(7): 2556-2559; 1979.

The dose dependency of the binding of benzo(a)pyrene (BP) to the DNA of Sprague-Dawley mouse epidermis was investigated. BP-conjugated epidermal DNA was isolated and enzymatically degraded to deoxyribonucleosides. The BP-DNA adducts were separated by Sephadex LH-20 column or high-performance liquid chromatography. Two major BP-DNA adducts were found. One was in the region of the elution profile that contained polycyclic aromatic hydrocarbons adducted to deoxyribonucleosides. The other adduct was eluted from Sephadex LH-20 and high-performance liquid chromatography columns before the deoxyribonucleosides and after deoxyribonucleotides. Both BP adducts in epidermal DNA reached a max 7 hr after a single skin application and remained constant for the next 49 hr. Both adducts varied as a linear function of topical dose in the range 0.01 to 300 μ g/mouse. Formation of the BP-DNA adducts occurred at doses several orders of magnitude below those that are feasible in tests for carcinogenicity. (28 refs)

- 79-4487** Cellular Progression of Neoplasia in the Subcutis of Mice after Implantation of 3,4-Benzpyrene. (Eng) Westwood, F. R. (Central Toxicology Lab., Imperial Chemical Industries Limited, Alderley Park, Macclesfield, Cheshire, England);

Longstaff, E.; Butler, W. H. *Br J Cancer* 39(6): 761-772; 1979.

An implantation model was used to locate and characterize the cell types involved in the progression of sc neoplasms induced by 3,4-benzpyrene (BP, 5 mg) in female Alderley Park strain Swiss albino mice. Pyrene (4 mg) and the gelatin control induced an initial exudation and inflammation around the implants, with subsequent encapsulation by fibroblasts, macrophages, and collagen. No sc tumors induced by either pyrene or the gelatin control were discovered by palpation in 14 mice over an 18 mo period. BP produced an initial inflammation similar to that in the controls, and this was followed by a prolonged exudation and infiltration by macrophages and lymphocytes. There was a temporal progression from aberrant filter- or muscle-associated cells through proliferative foci to large invasive sarcomas. Presarcomatous cell foci consisted of one of two different cell types: spindle cells with ultrastructural characteristics similar to those of foreign-body-induced sarcoma; and cells with the ultrastructural features of rhabdomyosarcoma. The subsequent appearance of two types of sarcoma that ultrastructurally resembled the cells of the early proliferative foci indicated that both elements may progress to form tumors. However, the constituent cells of both types of tumors displayed a broad histologic and ultrastructural spectrum and the marked similarity between undifferentiated cells of each suggested that both may have arisen from diverse differentiation of a common pluripotential cell such as the pericyte. (29 refs)

79-4488 A High-Capacity Condensation Aerosol Generation System. (Eng) Tu, K. W. (Environmental Measurement Lab., U. S. Dept. Energy, 376 Hudson St., New York, NY 10014); Kanapilly, G. M. *Environ Sci Technol* 13(6): 698-701; 1979.

A convenient, efficient, and compact condensation aerosol generation system capable of producing aerosols with high mass concentration and large volume from various materials is described. With laminar flow and warmed sheathing air in the system, the loss of aerosol due to condensation on the walls was minimized. This system was successfully used to generate aerosols of pyrene and benzo(a)pyrene with a mass concentration of 800 $\mu\text{g/liter}$ at a flow rate of 20 liters/min. (10 refs)

79-4489 Vitamin A and the Susceptibility of Respiratory Tract Tissues to Carcinogenic Insult. (Eng) Nettesheim, P. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Snyder, C.; Kim, J. C. *Environ Health Perspect* 29: 89-93; 1979.

The effect of vitamin A on the development of chemically induced lung carcinomas in specific pathogen-free Fischer

344 rats was investigated. Rats were maintained on low (total, 17.4 $\mu\text{g/wk}$), "normal" (174 $\mu\text{g/wk}$), and excess (1,740 $\mu\text{g/wk}$) levels of retinyl acetate (RA); the RA supplements were given twice weekly, by gavage. Squamous cell carcinomas of the respiratory tract were induced by intratracheal injections of 3-methylcholanthrene (3-MC: total doses of 10, 5, 2.5, or 1.25 mg for each RA dose level) 5 wk after the start of RA administration. Serial sacrifices conducted during the first 20 wk following carcinogen exposure showed that metaplastic lung nodules, presumed to be precursors of later-appearing carcinomas, occurred earlier and at a higher incidence in rats maintained on low levels of RA than in rats maintained on moderate or high levels of RA. The development of invasive pulmonary carcinomas was enhanced at all four carcinogen doses in rats receiving low levels of RA, compared with rats receiving moderate or high levels of RA. No consistent difference in lung cancer incidence existed between the groups receiving normal and high levels of RA. The data clearly show an increased susceptibility of vitamin A-deficient rats to the development of chemically induced lung cancers. (21 refs)

79-4490 Cryptofluorescent Analogs of Cobalamin Coenzymes: Synthesis and Characterization. (Eng) Jacobsen, D. W. (Dept. Biochemistry, Scripps Clinic and Res. Foundation, La Jolla, CA); Holland, R. J.; Montejano, Y.; Huennekens, F. M. *J Inorg Biochem* 10(1): 53-65; 1979.

The synthesis and properties of cryptofluorescent analogs of cobalamin coenzymes are described, along with their possible use in examining enzymatic mechanisms involving scission of the C-Co bond. Upper-axial (β -position) ligand analogs of the B_{12} coenzymes 5'-deoxy-5'-adenosylcob(III) alamin and methylcob(III) alamin were synthesized by reaction of the 5'-chloro-5'-deoxy derivatives of fluorescent nucleosides (1,N⁶-ethenoadenosine, formycin, 2-aminonebularine, and 2,6-diaminonebularine) and a fluorescent alkyl halide (dansylamidopropyl chloride) with cob(I) alamin. These analogs were nonfluorescent, but fluorescent products could be generated by photolysis or cyanolysis of the C-Co bonds. Under anaerobic and aerobic conditions, the major fluorescent photolysis products of 1,N⁶-ethenoadenosylcob(III) alamin were 1,N⁶-etheno-5',8-cyclic-5'-deoxyadenosine and the 5'-aldehyde of 1,N⁶-ethenoadenosine, respectively. The cryptofluorescent property of these analogs was utilized to follow the kinetics of aerobic photolysis. First-order rate constants determined by this method were comparable to those obtained spectrophotometrically [via appearance of aquacob(III) alamin]. Pseudo-first-order rate constants determined fluorometrically for the cyanolysis (at 25°C) of 1,N⁶-ethenoadenosylcob(III) alamin, 2,6-diaminonebularinylob(III) alamin, 2-aminonebularinylob(III) alamin, and formycinylcob(III) alamin were 5.8×10^{-2} , 2×10^{-2} , 1.8×10^{-2} , and $3 \times 10^{-5} \text{ min}^{-1}$, respectively; values in good agreement were obtained spectrophotometrically (via ap-

pearance of dicyanocobalamin). Dansylamidopropylcob-(III)alamin was stable in the presence of cyanide. The nucleoside α -ribazole was fluorescent in the free state but nonfluorescent when present as the lower axial (α -position) ligand in cobalamin coenzymes. Thus, fluorescence of ligands in both the α - and β -positions of cobalamins is quenched, probably as a result of intramolecular energy transfer between the ligands and the nonfluorescent corrinoid. (36 refs)

- 79-4491 Steroid-induced Acanthosis Nigricans in Dermatomyositis.** (Eng) Randle, H. W. (Mayo Clinic, 200 First St. SW, Rochester, MN 55901); Winkelmann, R. K. *Arch Dermatol* 115(5): 587-588; 1979.

A case of steroid-induced acanthosis nigricans is presented, and two additional cases and the literature are reviewed. The patient was first seen at age 4, when she was started on a regimen of triamcinolone (2-4 mg/day po) for dermatomyositis. The steroid was given for the next 9 yr. After 8 yr of steroid therapy, hyperpigmentation and verrucous epithelial hyperplasia of the axillae, groin, and neck were observed. Three years after the steroid treatment was discontinued, the patient had a slight improvement in the acanthosis. Ten years after steroid therapy was stopped, a pronounced resolution of the acanthosis nigricans was observed. Two other patients with childhood dermatomyositis developed acanthosis nigricans while on long-term cortisone therapy. The association of steroids with the development of acanthosis nigricans indicates that the endocrine system may be involved in this condition. The hypothesis that malignant acanthosis nigricans is due to a humoral mediator is supported by the finding of enterochromaffin-like cells of the APUD (ability for amine precursor uptake and decarboxylation) series of endocrine cells in a gastric carcinoma from a patient with acanthosis nigricans. (10 refs)

- 79-4492 Evidence of Epoxide Hydrase Activity in Human Intestinal Microflora.** (Eng) Hwang, K. K. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701); Kelsey, M. I. *Cancer Biochem Biophys* 3(1): 31-35; 1978.

Cholesterol-5 α ,6 α -epoxide (α -CE), an oxidation product of cholesterol, is carcinogenic in rats and mice in aqueous soln, and it has been implicated as an etiologic agent in human colon cancer. The epoxide was metabolized by human intestinal microflora to a polar product that was characterized by thin-layer and gas-liquid chromatography as well as combined gas-liquid chromatography-mass spectrometry as being cholestan-3 β ,5 α ,6 β -triol. These results suggest that microbial epoxide hydrase activity is present in the human colon. α -CE is currently being used as a

biological marker in studies of normal and high-risk colon cancer populations in order to identify susceptible individuals. (17 refs)

- 79-4493 Nuclear Retention of All Steroid Hormone Receptor Classes in the Hamster Renal Carcinoma.** (Eng) Li, J. J. (Res. Service, Building 49, Veterans Admin. Hosp., 54th St. and 48th Ave. South, Minneapolis, MN 55417); Li, S. A.; Cuthbertson, T. L. *Cancer Res* 39(7): 2647-2651; 1979.

Estrogen, androgen, progesterone, glucocorticoid, and mineralocorticoid receptors were studied in an estrogen (diethylstilbestrol)-induced and estrogen-dependent hamster renal carcinoma. No cross-competition was apparent for either the estrogen or progesterone receptors, but progesterone competed for both androgen and andro corticoid binding. The relative concentrations of the different receptors in the renal tumor decreased in the order of progesterone > estrogen > androgen > glucocorticoid > mineralocorticoid. The levels of all but the androgen receptor were remarkably uniform in different primary renal tumor specimens and in the metastases. The five cytosolic steroid receptors sedimented as either 7S or 8S binding components following sucrose gradient centrifugation in a low-salt medium. In purified tumor nuclei and tumor minces, the hormone-receptor complexes underwent nuclear translocation at elevated temperatures. Salt-extractable nuclear receptors for estrogen (5S), androgen (3.2S), progesterone (2.7S), and corticosteroid (3.5S) were identified. (37 refs)

- 79-4494 Cellular Proliferation and Secretion in the Adenohypophysis of Diethylstilbestrol-treated Rats.** (Eng) Delides, G. S. (International Center Environmental Safety, Albany Medical Coll., P.O. Box 1027, Holloman Air Force Base, NM 88330); Iatropoulos, M. J. *Environ Res* 18(2): 444-453; 1979.

Cellular proliferation and secretion in the anterior pituitary was studied during the course of diethylstilbestrol (DES-induced, 10 mg implanted sc in the form of a pellet every 30 days) tumorigenicity in female inbred and Fischer rats. Anterior pituitary tumors appeared earlier (140 days) and in a higher incidence (100%) in the DES-treated Fischer rats than in the DES-treated inbred rats. All of the tumors were of mammatropic cell type and none was malignant. The secretory activity of the anterior pituitary was initially enhanced in DES-treated rats with and without tumors. However, after Day 50, secretory activity decreased substantially in the animals with tumors while remaining high in the group without tumors. Tumorous pituitaries showed increased patterns of proliferation and decreased secretion, these patterns being accentuated in the larger tumors. (11 refs)

- 79-4495 Increased Progesterone Receptor Concentrations in Bladder Lesions of Estrogen-treated Syrian Hamsters. (Eng) Lin, Y. C. (Dept. Biological Chemistry, Harvard Medical Sch., Boston, MA 02115); Talley, D. J.; Vilee, C. A. *Cancer Res* 39(7): 2614-2617; 1979.

The concentration of progesterone-binding sites in the bladder and bladder lesions of diethylstilbestrol-treated castrated male Syrian hamsters were determined. After 3-4 mo of estrogen treatment, the concentration of binding sites in the bladder tissue cytosols was near the limit detectable by the assay (0.17×10^{-10} M). The concentration of binding sites in bladders of normal size (0.19 g) was similar after 7-10 mo of treatment, but the concentration of binding sites in enlarged bladders (0.89 g) at 7-10 mo was significantly greater ($8.67-61.56 \times 10^{-10}$ M) ($p < 0.01$). The affinity constant for progesterone in the cytosols of progesterone-binding sites was 1.66×10^{-10} M. The binding of progesterone to the cytosol binding sites was not decreased by prior incubation with saturating concentrations of 17β -estradiol, estrone, testosterone, 5α -dihydrotestosterone, or aldosterone. The bladder lesions ranged from predominantly inflammatory reactions to squamous metaplasia and hyperplasia of the transitional epithelium. These changes were more severe and extensive in the larger bladders. (17 refs)

- 79-4496 The Effects of Prenatal Diethylstilbestrol (DES) Exposure on the Genitalia of Pubertal *Macaca mulatta*. I. Female Offspring. (Eng) Hendrickx, A. G. (California Primate Res. Center, Univ. California, Davis, CA 95616); Benirschke, K.; Thompson, R. S.; Ahern, J. K.; Lucas, W. E.; Oi, R. H. *J Reprod Med* 22(5): 233-240; 1979.

The transplacental teratogenic and potential carcinogenic activities of diethylstilbestrol (DES) were studied using female rhesus monkeys (*Macaca mulatta*). The animals were given 1 mg/day DES po from day 21, 100, or 130 of gestation until delivery. The 19 animals delivered 20 offspring, 8 of which died before 5 yr of age; none of the deaths appeared related to DES. No DES-related irregularities in the reproductive cycle were observed. Vaginal ridging and/or cervical hooding were observed in 7/8 of the DES-exposed female offspring after 3 yr of age and 3 animals had histologically demonstrable vaginal adenosis. No evidence of lesions similar to those in the DES-exposed animals was observed in age-matched controls. There was no observed relationship between any of the abnormalities noted and the treatment period. None of the animals showed evidence of adenocarcinoma. The results indicate that the rhesus monkey may be suitable for evaluation of such lesions as adenosis and vaginocervical abnormalities associated with prenatal exposure to estrogen-like substances. However, this species may not be a suitable

model for study of the carcinogenic effects of DES. (35 refs)

- 79-4497 Lack of Evidence for a Direct Permanent Effect of Neonatal Sex Steroid Exposure on Mouse Mammary Gland. (Eng) Mori, T. (Zoological Inst., Faculty Science, Univ. Tokyo, Hongo, Tokyo 113, Japan); Mills, K. T.; Bern, H. A. *IRCS Med Sci (Cancer)* 7(4): 201; 1979.

When mammary glands of mice treated neonatally with 17β -estradiol or testosterone ($20 \mu\text{g/day}$, sc, for 5 days) were transplanted into untreated hosts of the same strain, they showed no significant differences from normal host mammary glands. When left in situ, the mammary glands of mice treated neonatally with the sex hormones showed marked alveolar development and a higher incidence of hyperplastic alveolar nodules at ≥ 7 mo of age. (6 refs)

- 79-4498 Transplantability and Sex Steroid Hormone Responsiveness of Cervicovaginal Tumors Derived from Female BALB/cCrgl Mice Neonatally Treated with Ovarian Steroids. (Eng) Jones, L. A. (Reproductive Endocrinology Center, Dept. Obstetrics, Univ. California, San Francisco, CA 94143); Pacillas-Verjan, R. *Cancer Res* 39(7, part 1): 2591-2594; 1979.

Vaginal and cervical segments from BALB/cCrgl mice exposed to progesterone ($100 \mu\text{g}$) and/or 17β -estradiol (5 or $20 \mu\text{g}$) for 5 days beginning within 36 hr after birth were transplanted into syngeneic hosts and examined. Cervicovaginal lesions were found in the remaining portions of the genital tracts in 17/28 neonatally treated mice; there was one frank tumor. Tumors developed in 6/28 hosts bearing genital tract transplants from these mice. All tumors were readily transplantable as solid fragments from host to host with a 100% take rate. Three tumors in mice bearing tissues from progesterone-treated mice had both squamous cell and glandular components; these tumors metastasized to the intestinal region in some transplant hosts. Two squamous cell tumors arose in mice bearing tissues from estrogenized mice. These tumors grew more slowly than did the progesterone-induced tumors, but they also metastasized to the intestinal region. The sixth tumor, a squamous cell carcinoma, arose in a mouse bearing a tissue segment from a donor treated with estrogen plus progesterone. After three transplant generations, it changed from a squamous cell pattern to one resembling that of a small cell carcinoma. This tumor was highly invasive, metastasizing to both the liver and intestinal region. All tumors were hormone dependent in the transplant hosts. The results support the idea that hyperplastic lesions in the cervicovaginal region of mice treated neonatally with steroids are potentially neoplastic. (9 refs)

79-4499 Cervicovaginal and Mammary Gland Abnormalities in BALB/cCrgl Mice Treated Neonatally with Progesterone and Estrogen, Alone or in Combination. (Eng) Jones, L. A. (Reproductive Endocrinology Center, Dept. Obstetrics, Gynecology, and Reproductive Sciences, Univ. California, San Francisco, CA 94143); Bern, H. A. *Cancer Res* 39(7): 2560-2567; 1979.

Female BALB/cCrgl mice (mammary tumor virus unexpressed) were given daily sc injections of 50 or 20 μ g 17 β -estradiol (ES) and/or 100 μ g progesterone (PR) in 0.02 ml Sesame oil for 5 days, beginning within 36 hr of birth. About one-half of each group was ovariectomized when 40 days old, and all mice were killed between 18.5 and 26 mo of age. Neonatal PR leads to ovary-dependent persistent vaginal cornification and hyperplasia. In addition, 16/24 PR-treated mice had genital tract lesions, and 4 of these showed predominantly glandular features. No such lesions were observed in oil-treated or untreated mice. Lesions were also observed in both intact and ovariectomized mice treated with ES and ES-PR combinations, but most of the lesions were not as severe as those seen in mice treated neonatally with PR alone, and they were predominantly squamous in appearance. Although mammary tumors were not observed in the controls or the neonatally steroid-treated intact mice, many in the latter groups exhibited hyperplastic alveolarlike mammary nodules and other abnormalities. (40 refs)

79-4500 Endometrial Histology and Biochemistry in Climacteric Women During Oestrogen and Oestrogen/Progestogen Therapy. (Eng) Whitehead, M. I. (Dept. Obstetrics and Gynaecology, King's Coll. Hosp. Medical Sch., London SE5, England); McQueen, J.; King, R. J.; Campbell, S. *J R Soc Med* 72(5): 322-327; 1979.

Endometrial proliferation was monitored during cyclic estrogen and sequential estrogen/progestogen therapy to determine the endometrial response to exogenous estrogens and the modifying effect of progestogens on this response. Endometrial biopsy was performed on 62 perimenopausal and 115 postmenopausal women. In the group receiving cyclical therapy (2-47 mo, mean 15.1 mo), hyperplasia was observed in 22/69 patients on high-dose regimens and in 6/33 patients on low-dose regimens. During sequential therapy (2-50 mo, mean 16.2 mo), hyperplasia was diagnosed in 2/46 patients given high-dose regimens and in 1/29 patients given low-dose regimens. Twenty of the 28 patients in whom hyperplasia was diagnosed during cyclical therapy subsequently received sequential regimens; in all but one normal endometrium was obtained at repeat biopsy. Two of the three patients in whom hyperplasia was diagnosed during sequential therapy subsequently received

a combined estrogen/progestogen regimen (norethisterone, 2.5 mg, combined with the estrogen); normal endometrium was obtained at repeat biopsy in both cases. During cyclic therapy, cytoplasmic progesterone receptor levels were comparable with the premenopausal proliferative stage, indicating that the endometrium was subjected to a potent estrogen stimulus. During sequential therapy, receptor levels increased from wk 2 to 3 but were depressed during the wk of norethisterone ingestion (wk 4) and the following wk. Estradiol 17 β -dehydrogenase levels increased during progestogen administration. The results indicate that progestogens protect the endometrium against estrogen-induced stimulation. (21 refs)

79-4501 Estrogen-Progesterone Therapy in Perimenopausal Women. (Eng) Budoff, P. W. (Dept. Family Medicine, State Univ. New York, Stony Brook, NY 11797); Sommers, S. C. *J. Reprod Med* 22(5): 241-247; 1979.

Seventy-four women who had received estrogen combined with progesterone over an av of 57 mo for treatment of menopausal symptoms were studied retrospectively. The results were compared with those in 473 controls given neither estrogen nor estrogen-progesterone therapy. Endometrial aspiration biopsies in 26 patients and 28 controls showed no significant differences in the total number of normal, inactive or atrophic, or hyperplastic readings. The most common finding in the treated patients was a secretory response, which suggested that progesterone exerts an antiestrogen effect even when estrogen is given on a continuing basis. Among all hormone-treated patients, weight decreased over time; blood pressure, blood sugar, and cholesterol levels remained virtually unchanged; and Papanicolaou smear classes improved. (9 refs)

79-4502 A Study of Endocrine and Metabolic Variables in Postmenopausal Women with Endometrial Carcinoma. (Eng) Lucas, W. E. (225 W. Dickinson St., San Diego, CA 92103); Yen, S. S. *Am J Obstet Gynecol* 134(2): 180-186; 1979.

Fasting plasma levels of growth hormone (GH), insulin, prolactin (PRL), follicle-stimulating hormone, luteinizing hormone, estrone (E), and estradiol (E2) were measured on 3 consecutive days in 16 nonobese postmenopausal women with endometrial carcinoma (EC) and in 16 age- and wt-matched cancer-free controls. The EC and control groups showed no significant differences in the fasting levels of any hormone tested. The changes in insulin, GH, and

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glucose following 25 mg of iv glucose were similar for the two groups, as were the changes in GH and insulin following arginine infusion. The peak hypoglycemic response in insulin (0.1 unit/kg) was identical in the two groups, but the recovery from hypoglycemia was faster among the controls than among the EC patients ($p < 0.05$). The two groups did not differ in GH response to hypoglycemia. Patients and controls who had received estrogen therapy did not differ significantly in any test from those who had not. No correlation was found between baseline levels of E and E2 and either GH or PRL in either group. (7 refs)

- 79-4503 Serious Liver Disease in Women on Oral Contraceptives. (Cze) Brodanova, M. (I. interní klinika fakultní nemocnice I s poliklinikou, KUNZ SKNV, U nemocnice 2, 128 08 Prague 2, Czechoslovakia); Balas, V.; Kordac, V. *Cas Lek Cesk* 118(1): 22-27; 1979.

Two case reports illustrating the serious hepatotropic side effects of oral contraceptives are presented. Exacerbation of Budd-Chiari syndrome as a consequence of the use of oral contraceptives for 7 mo was seen in a 22-yr-old woman. A 42-yr-old woman developed multiple benign hepatic adenomas of the liver after using oral contraceptives for 7-8 yr. (29 refs)

- 79-4504 The Induction, Purification and Characterization of 17β -Hydroxy- C_{19} -Steroid Dehydrogenase of the Female Guinea Pig Kidney. (Eng) Shen, C. C. (Lab. Experimental Endocrinology, Medical Center, Univ. Alabama in Birmingham, Birmingham, AL 35294); Kochakian, C. D. *J Steroid Biochem* 10(2): 187-193; 1979.

Kidney cytosol and microsomes from female guinea pigs contained little or no detectable NADP⁺- or NAD⁺-linked dehydrogenase activity for several hydroxy- C_{19} -steroids. Testosterone administration induced a gradual increase in cytosol β - C_{19} -steroid dehydrogenase activity to the level of the male, but this increase was not seen in the microsomal fraction. The purification and characterization of the induced enzyme are described. (14 refs)

- 79-4505 Chemical Carcinogenesis. (Eng) Calvin, M. (Lab. Chemical Biodynamics, Univ. Califor-

nia, Berkeley, CA 94720). *Prog Biochem Pharmacol* 14: 6-27; 1978.

Primary cellular change, which may be induced by physical, chemical, and/or biological agents, may be the factor that is common to all carcinogenesis. The nature of that primary change and how it may result from the action or interaction of viruses, chemicals, and radiation are discussed. (no refs)

- 79-4506 The Effect of Temperature on the Formation of Mutagens in Heated Beef Stock and Cooked Ground Beef. (Eng) Dolara, P. (Center Biology Natural Systems, Washington Univ., St. Louis, MO 63130); Commoner, B.; Vithayathil, A.; Cuca, G.; Tuley, E.; Madyastha, P.; Nair, S.; Kriebel, D. *Mutat Res* 60(3): 231-237; 1979.

Studies of the role of temperature in the production of mutagens in beef stock and in ground beef cooked by several procedures that differ in temperature are reported. The microsome-activatable mutagens [chromatographically distinguishable from benzo(a)pyrene and from the mutagens produced from pyrolyzed amino acids and proteins] previously found in beef extract and in bacterial nutrients that contain beef extract were produced in beef stock when it was heated. Reflux boiling of beef stock at 100 C resulted in a linear increase in mutagenic activity toward *Salmonella* strain TA1538. The rate of production of mutagenic activity at temperatures between 68 and 98 C conformed closely to the Arrhenius equation, yielding an activation energy of 23,738 calories per mole. Extrapolation from these data predicted a sharp rise in the rate of mutagen formation between 140 and 180 C. This prediction was confirmed when ground beef patties (hamburgers) were prepared in various conventional electrically heated appliances that operate at different cooking temperatures within this range. The mutagenic activity of hamburger cooked at high temperatures was limited to the surface layers; the temperature inside the hamburger did not exceed 100 C during cooking. No mutagenic activity was found in comparable samples of uncooked meat. The results indicate that the mutagens may be formed as a result of the temperatures encountered in certain conventional cooking procedures. (6 refs)

- 79-4507 Physical Factors Affecting the Mutagenicity of Fly Ash from a Coal-fired Power Plant. (Eng) Fisher, G. L. (Radiobiology Lab., Univ. California,

Davis, CA 95616); Chrisp, C. E.; Raabe, O. G. *Science* 204(4395): 879-881; 1979.

Respirable fly ash fractions from a coal-fired power plant smokestack were studied for their mutagenicity in *Salmonella typhimurium* strains TA1538, TA98, and TA100. Serum filtrates of all four size-classified, stack-collected fractions were mutagenic for TA1538. The finest fractions (3 and 4) were the most mutagenic, with fraction 3 being more mutagenic than the finest fraction, 4. The mutagenicities of all four fractions, especially 3, increased in the presence of S9 mix. Unsized, electrostatic precipitated fly ash was not mutagenic for TA1538. UV- or x-irradiation did not significantly affect the mutagenicity of serum filtrates of fraction 3 for any bacterial strain, but the mutagenic activity decreased at temperatures above 200 C. No mutagenic activity was observed with filtrates from fly ash heated to 350 C. (16 refs)

79-4508 The Use of Sister Chromatid Exchange Analysis to Detect Ambient Mutagens in Drinking Water (Meeting Abstract). (Eng) Guerrero, R. R. (Pasadena Foundation Medical Res., Pasadena, CA 91101); Rounds, D. E. *In Vitro* 15(3): 171-172; 1979 (no refs)

See also:

- *(Rev.): 79-4201, 79-4202, 79-4203, 79-4204, 79-4205, 79-4206, 79-4207, 79-4208, 79-4209, 79-4211, 79-4212, 79-4213, 79-4214, 79-4215, 79-4216, 79-4217, 79-4218, 79-4219, 79-4220, 79-4221, 79-4222, 79-4223, 79-4224, 79-4225, 79-4226, 79-4227, 79-4228, 79-4229, 79-4230, 79-4231, 79-4232, 79-4233, 79-4234, 79-4235, 79-4236, 79-4237, 79-4238, 79-4239, 79-4240, 79-4241, 79-4242, 79-4243, 79-4244, 79-4245, 79-4246, 79-4247, 79-4248, 79-4249, 79-4250, 79-4251, 79-4252, 79-4253, 79-4254, 79-4255, 79-4256, 79-4257, 79-4258, 79-4259, 79-4260, 79-4261, 79-4262, 79-4263, 79-4264, 79-4270, 79-4302, 79-4303, 79-4308, 79-4309, 79-4310, 79-4311, 79-4313, 79-4314, 79-4316, 79-4318, 79-4319, 79-4320, 79-4321, 79-4322, 79-4323, 79-4324, 79-4325.
- *(Phys.): 79-4521, 79-4529, 79-4535, 79-4538, 79-4539, 79-4540, 79-4542, 79-4543, 79-4544, 79-4546, 79-4550.
- *(Viral): 79-4603, 79-4633.
- *(Immun.): 79-4652, 79-4658, 79-4673, 79-4674, 79-4679, 79-4680.
- *(Epid.-Biom.): 79-4739, 79-4740, 79-4744, 79-4745, 79-4746, 79-4748, 79-4750, 79-4751, 79-4754, 79-4760, 79-4762, 79-4765, 79-4767, 79-4770, 79-4778, 79-4779, 79-4784.

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79-4509 Radon-222, a Potentially Harmful Agent in Certain Nonuranium Mines. (Fre) Weissbuch, H. (Institutul de igiena si sanatate publica, Str. Dr. Victor Babes nr 14, IASI, Romania); Botezatu, E.; Gradinaru, M. *Radioprotection* 14(1): 41-44; 1979.

Concentrations of ^{222}Rn were determined in the air inside seven nonuranium mines. The mean concentrations, expressed as picocuries/liter, were 13.1-1,278, and they ranged from 3.5 to 1,866. In all mines but one, the concentrations exceeded both the max allowable concentration established for the general population and the max allowable concentration established for work areas. The findings indicate that nonuranium miners have a potential cancer risk due to the presence of ^{222}Rn in the air. (8 refs)

79-4510 Autopsy Studies of Hashimoto's Thyroiditis in Hiroshima and Nagasaki (1954-1974): Relation to Atomic Bomb Radiation. (Eng) Asano, M. (Dept. Pathology, Radiation Effects Res. Foundation, 164 Sakurabacho, Nagasaki 850, Japan); Norman, J. E.; Kato, H.; Yagawa, K. *J Radiat Res (Tokyo)* 19(4): 306-318; 1978.

The late effects of atomic bomb radiation on the prevalence of Hashimoto's thyroiditis (HT) were assessed by studying the 1954-1974 autopsy records of A-bomb survivors and unexposed controls in Hiroshima and Nagasaki. HT was classified as lymphoid (71 cases), diffuse (6), or fibrous (78). The radiation doses for A-bomb survivors were estimated in rads based on air-decay curves, shielding effects, and data collected from each survivor. The number of cases of HT in the autopsy cases increased from 0.20% in 1956 to 4.93% in 1974, but the difference was not significant. There was no association between HT type and age at time of exposure or dose. The male:female ratio (0.24) in the low-dose HT patients (not in the city at the time of the bomb) was as expected, but the ratio for patients with diffuse or fibrous types in the high-dose group (100+ rads) was reversed (1.6). However, this difference was not statistically significant. There was no significant pattern of variation of thyroid wt with radiation dose or type of thyroiditis. The following cancers were observed in the HT patients: stomach cancer (8 cases), bronchogenic cancer (6), breast cancer (3), and ovarian cancer (3). The prevalence of ovarian cancer was significantly higher than that expected, but the prevalence of stomach cancer and all cancers was lower. The results suggest there was no late effect of A-bomb radiation in this series. (29 refs)

79-4511 The Calculation of Annual Limits of Intake for Plutonium-239 in Man Using a Bone Model Which Allows for Plutonium Burial and Recycling. (Eng) Priest, N. D. (Natl. Radiological Protection Board, Harwell, Didcot, Oxon. OX11 0RQ, England); Hunt, B. W. *Phys Med Biol* 24(3): 525-546; 1979.

Annual limit of intake (ALI) values for plutonium-239 in humans were calculated with the use of committed dose equivalent limits and a multicompartment bone model that allows for Pu burial and recycling in the skeleton. In one skeletal compartment, the growing surfaces of cortical bone, it was assumed that Pu deposits are retained and are not subject to resorption or recycling. In the trabecular bone compartment, Pu was taken to be resorbed with either subsequent redeposition onto bone surfaces or retention in the bone marrow. ALI's for ^{239}Pu were calculated assuming a range of rates of bone accretion ($0-32 \mu\text{m yr}^{-1}$), different amounts of Pu retained in the marrow (0%-60%), and a 20%, 45%, or 70% deposition of Pu in the skeleton from the blood. The calculations made using this bone model suggest that 750 Becquerels (Bq; 20 nanocuries) is an appropriate ALI for the inhalation of class W and Y Pu compounds and that 830 kBq and 5 MBq (23 and 136 μCi) are the appropriate ALI's for the ingestion of soluble and insoluble forms of Pu, respectively. (33 refs)

79-4512 Relationship Between Age and Type of Exposure to ^{90}Sr and Tumor Development in Rats. (Rus) Lavrent'ev, L. N. (Res. Inst. Radiation Hygiene, Leningrad, USSR). *Vopr Onkol* 25(4): 38-41; 1979.

^{90}Sr -induced tumorigenesis was studied in 3- to 4-mo-old and 8- to 10-mo-old albino rats. The animals were given chronic doses ($0.002 \mu\text{Ci/g/day po}$, for 720 days) or a single dose ($0.35 \mu\text{Ci/g po}$) of the isotope. After chronic exposure, tumors developed in 17/75 3- to 4-mo-old rats and in 22/112 8- to 10-mo-old rats; the incidence of osteosarcomas depended upon the age of the rats (5/17 younger rats had osteosarcomas, compared with 0/14 older animals). After a single administration of isotope, tumors were detected in 32/70 younger rats (all had osteosarcomas) and in 7/45 older rats (only 1 had an osteosarcoma). The av latent period of osteosarcoma development was significantly shorter in the 3- to 4-mo-old rats (193-316 days, vs 487-654 days for the 8- to 10-mo-old rats). (9 refs)

- 79-4513 Lung Tumorigenesis in the Syrian Hamster from Particulate Sources of ^{147}Pm β Radiation. (Eng) Anderson, E. C. (Health Div., Los Alamos Scientific Lab., Univ. California, Los Alamos, NM 87545); Holland, L. M.; Prine, J. R.; Smith, D. M. *Radiat Res* 79(2): 349-367; 1979.

Syrian hamsters were exposed to ^{147}Pm β radiation from ceramic microspheres permanently lodged in the capillary bed of the lung. Lung burdens ranged from 6,000 to 152,000 spheres, with activities of either 70 or 450 picocuries (pCi)/sphere. Lung burdens of 0.4 to 16 μCi gave initial dose rates of 0.65 to 20.5 krad/yr. No life shortening was observed. Tumor incidence increased with the logarithm of dose rate and reached 30% at about 15 krad/yr. Tumor induction time was about 300 days; the incidence rate reached a max at about 600 days postexposure. At all dose levels, the distribution of tumor types was 35% adenomas, 41% adenocarcinomas, and 24% epidermoid carcinomas. Attempts to identify certain lesions as precancerous were unsuccessful. In particular, the bronchiolar adenomatoid lesion (BAL), which is indistinguishable from a variety of inflammatory processes in the lung, was found at significant levels in controls; and BAL incidence did not correlate with subsequent incidence of malignancy in treated animals. (44 refs)

- 79-4514 Development of Multiple Myeloma in Long Term Survivors of Breast Cancer. (Eng) Yeh, G. K. (275 Hospital Parkway, Suite 350, San Jose, CA 95119); Axelrod, M. R. *Clin Oncol* 5(2): 175-177; 1979.

Two breast patients (black women aged 28 and 81 yr) developed multiple myeloma 6 yr after successful treatment of their primary malignancy. Both primary tumor specimens revealed lymphocytic infiltration, and both patients had received postoperative regional radiotherapy with ^{60}Co (6,000 rads over a 4-wk period). Both patients had severe bone pain, lytic bone lesions, M protein, anemia, and plasma cell infiltration of the marrow. The sequential development of these two malignancies may be more than coincidental. The possibility of multiple myeloma should be entertained in cases in which extensive lytic bone lesions develop years after successfully treated breast cancer. (8 refs)

- 79-4515 Response of Swine Skin to Acute Single Exposures of X-Rays: Quantification of the Epidermal Cell Changes. (Eng) Archambeau, J. O. (Div. Radiation Oncology, City of Hope Natl. Medical Center, Duarte, CA 91010); Bennett, G. W.; Abata, J. J.; Brenneis, H. J. *Radiat Res* 79(2): 298-337; 1979.

Three shoulder, chest, and ham skin fields (10 cm in diameter) of 24 swine were irradiated with acute single ex-

posures of 1,700, 2,300, and 2,700 R. The animals were anesthetized, biopsied, and sacrificed at 1- to 70-day intervals post-irradiation. The observed histologic changes occurred in two phases: the degenerative phase was characterized by progressive cell loss (similar for all fields), an increase in nuclear volume, and a decrease in mitotic index; and the regenerative phase was characterized by the appearance of discrete islands of normal-appearing cells. These regenerating islands, which were classified based on chord length as small, large, or giant, increased progressively in size until the field was re-epithelialized. The mitotic index was as high as 6%, with a tritiated thymidine labeling index between 30% and 70%. The estimated cell cycle time was 15 hr. The number of islands was dose-dependent at 17-28 days postirradiation, with D_0 of 272 R for small islands, 568 R for large islands, 1,620 R for giant islands and 337 R for the combined data. Re-epithelialization was attributed to proliferation of cells in giant islands, while cells from small islands contributed little. (21 refs)

- 79-4516 Dose-Response Relations for Dicentric Yields in G_0 Lymphocytes of Man and a Crab-eating Monkey Following Acute and Chronic γ -Irradiations. (Eng) Takahashi, E. (Div. Genetics, Natl. Inst. Radiological Sci., Anagawa, Chiba 260, Japan); Hirai, M.; Tobari, I.; Nakai, S. *Mutat Res* 60(3): 357-365; 1979.

The dicentric yields in the G_0 lymphocytes of a 29-yr-old male volunteer and a crab-eating monkey, *Macaca fascicularis*, were compared after acute and chronic γ -irradiation. There was no significant difference in dicentric yield between the two species, with acute irradiation (49.6 rads/min), but there was a significant difference with chronic irradiation (17.1 rads/hr). When the dose-response relationships were fitted to a linear-quadratic model ($Y = \alpha D + \beta D^2$), the species difference was found to be almost entirely due to a change in the value of β . After chronic irradiation, the β value for the monkey was almost negligible, but that for the man was significant. A postirradiation incubation experiment showed that cells with dicentrics were partly eliminated during chronic irradiation, because there were appreciable reductions in the dicentric yield (approx 25% for both man and monkey at 400 rads and mitotic index (approx 30% and 60% for man and monkey, respectively, at 400 rads). Accordingly, it would be reasonable to postulate that a G_0 repair mechanism for dicentrics, other than selective elimination, must play a major role in mediating the effects of low-dose radiation. The capacity for the G_0 repair of chromosome damages leading to dicentrics may differ among various primate species. (22 refs)

- 79-4517 Response of an *Oedionychina* (Coleoptera) Karyotype to Acute Gamma Radiation. (Eng)

Virkki, N. (Agricultural Experiment Station, Mayaguez Campus, Univ. Puerto Rico, Rio Piedras, Puerto Rico). *J Agric PR* 63(2): 116-145; 1979.

Acute exposure to ^{60}Co radiation at 250 and 500 rads produced chromosome aberrations in practically all treated male fleabeetles (*Omophoita cyanipennis*). Spermatogonia were unaffected. In spermatogenesis, aberrations during metaphase I (MI) became recognizable 4-6 hr after radiation. Although presumably induced in a later phase than chromosome gaps, chromosome translocations reached their MI peak simultaneously with the gaps, about 24-50 hr after radiation. This was due to a marked delay in the development of cells irradiated during the diffuse stage. The late prophase effects manifested at MI were sticky interchromosomal contacts and subchromatid interchanges. Both gap and translocation yields increased with dose; 3,000 and 12,000 rads produced pulverization and clumping of chromosomes. The long sex chromosomes appeared to be the most probable exchange partners for any *O. cyanipennis* chromosomes. Interautosomal exchanges comprised only 6.8% of all exchanges. Aberrations caused irregular chromosome segregation and fragments both at anaphase I and anaphase II, resulting in undersized and supernumerary supermatid nuclei, and a great variation of chromatin content even in those nuclei that had a normal appearance. (41 refs)

79-4518 Radiation Induced Chromosome Aberrations and the Poisson Distribution. (Eng) Edwards, A. A. (Natl. Radiological Protection Board, Harwell, Didcot, Oxon. OX11 0RQ, England); Lloyd, D. C.; Purrott, R. J. *Radiat Environ Biophys* 16(2): 89-100; 1979.

The distribution of dicentrics and acentrics that occurs when human lymphocytes are cultured for 48 hr after irradiation by x-rays, γ -rays, or neutrons was studied. For dicentrics, the observed distribution after treatment with x-rays, γ -rays, and fission neutrons could be described by Poisson statistics. For higher energy neutrons, however, overdispersion was observed. The phenomenon of overdispersion was also observed for acentrics, irrespective of the type of radiation used. Both acentrics and dicentrics showed a greater dispersion when the blood was irradiated with neutrons than when it was irradiated with γ -rays. The possibility that overdispersion results from variations in dose to sensitive sites led to the conclusion that for dicentrics, the site size is considerably larger than the 1-2 μm diameter derived by applying the dual action theory to the dose-effect relationships. This larger site may be the cell nucleus. (17 refs)

79-4519 Re: Radiation-induced Bladder Tumors (2 Letters to Editor). (Eng) Fokkens, W. (Dept. Documentation and Epidemiology, Rotterdamsch Radio-

Therapeutisch Instituut, Rotterdam, Netherlands); Hop, W. C.; Duncan, R. E.; Bennett, D. W.; Evans, A. T.; Aron, B. S.; Schellhas, H. F. *J Urol* 121(5): 690; 1979.

A previous conclusion that patients irradiated for cervical carcinoma had subsequent primary bladder cancers (BIC's) 57 times more often than expected is criticized because of the way in which the age of the patients was taken into account. By calculating the expected number of BIC's based on a female population >50 yr old, the actual risk of BIC is much less. In a reply, the original authors feel that this calculation of expected BIC's introduces an artificial distribution of patients for statistical comparison. An independent consultant suggests that both points are valid. When both are considered in the calculations, the actual risk is eight times greater than that expected, still large enough to suggest some risk from radiation. (1 ref)

79-4520 Potential Health Effects of Low-Level Irradiation During Development in the Dog (Meeting Abstract). (Eng) Benjamin, S. A. (Fort Collins, CO); Angleton, G. M.; Hargis, A. M.; Jaenke, R. S.; Lee, A. C.; Miller, C. W.; Norrdin, R. W.; Phemister, R. D. *J Am Vet Med Assoc* 174(9): 953; 1979 (no refs)

79-4521 Early Changes in the Rat Colon after Treatment with X-Radiation and 1,2-Dimethylhydrazine. (Eng) Denman, D. L. (Univ. Iowa, Iowa City, IA). *Diss Abstr Int [B]* 39(8): 3714-3715; 1979 (no refs)

79-4522 Ionizing Radiation and Laryngeal Carcinoma. (Ger) Martin, G. (HNO-Klinik, Philipps-Universitat Marburg a.d.L., Deutschhausstrasse 3, 3550 Marburg a.d.L., W. Germany); Glanz, H.; Kleinsasser, O. *Laryngol Rhinol Otol (Stuttg)* 58(3): 187-195; 1979.

Case report of the relationship between ionizing radiation and laryngeal carcinoma are presented. A large epiglottic carcinoma was discovered in a 72-yr-old man who had received x-ray therapy for edema of the cervical lymph nodes at age 19 yr and again at age 35 yr. The occurrence of a second laryngeal carcinoma >5 yr after diagnosis of the first was studied in a series of 109 patients who had undergone radiotherapy with or without surgery for laryngeal carcinoma. Fifty-one of the 109 patients were followed for >10 yr, a few for up to 17 yr. Eight second laryngeal carcinomas were found 6-15 yr after radiotherapy (3/48 after radiotherapy and surgery, 2/26 after telecobalt therapy, and 3/25 after cobalt contact therapy). The eight cases represent a considerably high percentage, because the number of patients decreases over the years. The findings indicate the potential hazards of irradiation of the laryngeal area, especially in young patients. (91 refs)

- 79-4523 Basal-Cell Tumors of the Lumbar Skin after Radiotherapy for Arthrosis. (Eng) Di Pietro, S. (Istituto Nazionale Tumori, Via G. Venezian, 1, 20133 Milan, Italy); Milani, A.; Volterrani, F. *Tumori* 65(1): 127-132; 1979.

Twenty-three patients (10 men, 13 women 46-79 yr old) with basal-cell tumors of the skin arising in the lumbar-sacral region after repeated irradiation for arthrosis are described. All of the patients had been submitted to 2-10 cycles of irradiation, with each cycle consisting of an av of six doses of 100 R each. The total dose absorbed at skin level was estimated at 14.4-72.0 Gray units administered over a 2- to 6-yr period. Thirteen to 30 yr (median, 19 yr) had elapsed since the end of the irradiations, but in some cases the lesions had been in existence for a number of years. The fact that basal cell tumors are relatively rare on the lumbar-sacral skin supports the hypothesis of a cause-effect relationship between radiation and these tumors. The observations confirm the possibility of skin cancer occurring after many years as a consequence of low-dose radiation administered for anti-inflammatory purposes. (13 refs)

- 79-4524 Carcinogenic Effect of Combined Whole-Body External and Local Internal ^{131}I Radiation in Rats. (Rus) Vasil'eva, L. A. (Res. Inst. Vocational Hygiene and Occupational Diseases, Moscow, USSR); Burykina, L. N.; Likhachev, I. P. *Vopr Onkol* 25(4): 35-38; 1979.

Random-bred albino rats were divided into four groups: (1) controls; (2) ^{131}I po for 10 days (total dose 20 $\mu\text{Ci}/\text{rat}$); (3) whole-body irradiation with x-rays (300 R); and (4) combined exposure to ^{131}I and x-rays. The animals were followed until death. The life-span of Groups 3 and 4 rats was decreased (561 and 556 days, respectively) compared with that of Group 1 and 2 (645 and 635 days, respectively). The number of tumor-bearing animals was significantly increased in Groups 3 and 4 (75.0% and 69.0%, respectively, compared with 33.8% in Group 1 and 40.0% in Group 2). The incidence of malignant tumors was 18.8% in Group 1, 12.0% in Group 2, 42.7% in Group 3, and 50.0% in Group 4. The incidence of thyroid tumors was significantly increased in Groups 2, 3, and 4 (17.7%, 17.5%, and 16.6%, vs 7.9% in controls). (4 refs)

- 79-4525 Anaplastic Carcinoma of Thyroid: Radiation-associated. (Eng) Shimaoka, K. (Dept. Medicine B, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263); Getaz, E. P.; Rao, U. *NY State J Med* 79(6): 874-877; 1979.

A review of 316 patients admitted to a cancer research institute during 1956-1975 with diagnosis of thyroid car-

cinoma resulted in a confirmed diagnosis of anaplastic or poorly differentiated thyroid carcinoma in 41 patients. Three (all white men) of the latter had a history of radiation therapy: two had received radiation for Hodgkin's disease, 44 and 19 yr, respectively, before diagnosis of thyroid carcinoma; the third had received radiation therapy for a chromophobe adenoma of the pituitary 5 yr earlier. A search of the literature identified 12 additional cases of anaplastic carcinoma associated with previous radiation exposure. Among the total 15 patients, all 5 who were treated for thyroid conditions probably received >2,000 rads. Of the remaining 10 patients, 4 were treated for malignant lymphoma, and at least 3 of these received high doses. Only four patients were <40 yr old at the time of diagnosis of anaplastic thyroid carcinoma. At $\geq 2,000$ rads, there appears to be a gradual decrease in risk of developing thyroid carcinoma. However, this may be largely due to the decrease of well-differentiated thyroid carcinoma, and the risk of developing anaplastic carcinoma may actually increase at higher radiation doses. (45 refs)

- 79-4526 Significance of Non-palpable "Cold" Scintigraphic Thyroid Defects in Patients Following Childhood Neck Irradiation (Meeting Abstract). (Eng) Okerlund, M. D. (Univ. California, San Francisco, CA); Sommers, J.; Sakmar, T.; Galante, M.; Hunt, T.; Clark, O. *J Nucl Med* 20(6): 678; 1979 (no refs)

- 79-4527 Radiation-induced Cancer of Pharynx and Esophagus. Report of Three Cases. (Fre) Charles, J. (SSM AI, 20 rue de Tinchon, F-59300 Valenciennes, France); Fiasse, R.; Pringot, J.; Heller, F. *Acta Gastroenterol Belg* 42(1/2): 7-29; 1979.

Three cases of esophageal cancer that developed following x-ray treatment of benign thyroid diseases are reported. A poorly differentiated malpighian epithelioma was found in a 77-yr-old woman 50 yr after radiotherapy for Graves' disease [160 kilovolts (kV), left and right cervical fields; 9 sessions in 6 mo, total dose 3,150 R/field or a total skin dose of 6,300 R (2,100 rads); second treatment 5 wk later consisting of 15 sessions in 6 mo; total dose 5,250 R/field corresponding to a total skin dose of 10,500 R (6,200 rads)]. A well-differentiated malpighian epithelioma of the cervical esophagus was found in a 42-yr-old woman 21 yr after low-voltage x-ray therapy for a benign goiter (200 kV, 2 cervical fields, 500 rads/field, 8 weekly sessions). A differentiated malpighian epithelioma of the cervical esophagus was diagnosed in a 66-yr-old woman 52 yr after radiotherapy (probably low-voltage) for benign goiter. (16 refs)

- 79-4528 Sister-Chromatid Exchange and Phototherapy. (Eng) Hatcher, N. H. (Birth Defects

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Inst., Div. Lab. and Res., New York State Dept. Health, Albany, NY 12237); Risemberg, H. M.; Powers, M. M.; Hook, E. B. *Mutat Res* 60(3): 401-403; 1979.

The frequency of sister-chromatid exchanges (SCE) in peripheral blood samples from 11 infants who had undergone phototherapy for hyperbilirubinemia was compared with that in blood samples from 29 control infants. The results indicated no association between phototherapy and SCE frequency. This negative finding could not be explained by changes following termination of phototherapy, since SCE values determined in the cord blood of six infants prior to phototherapy were similar to posttherapy values. (3 refs)

- 79-4529 In Vivo Sister-Chromatid Exchange: A Sensitive Measure of DNA Damage.** (Eng) Nakanishi, Y. (Section on Cellular Aging and Genetics, Lab. Cellular and Molecular Biology, Gerontology Res. Center, Natl. Inst. Aging, NIH, Baltimore, MD 21224); Schneider, E. L. *Mutat Res* 60(3): 329-337; 1979.

A variety of chemical agents and x-irradiation were examined for their abilities to induce sister-chromatid exchanges (SCE) in male C57BL/6J mice. SCE were counted in metaphase bone-marrow cells following iv bromodeoxyuridine treatment and x-irradiation or iv administration of the test drug. The highest level of SCE was produced by cyclophosphamide (CPA), but the most potent inducer of SCE was mitomycin C (MMC). Other potent inducers were 4-nitroquinolone-1-oxide (4NQO), N-methyl-N'-nitro-N-nitrosoguanidine, adriamycin, and streptonigrin. Ethyl methanesulfonate, daunomycin, dibromomannitol, and x-rays were moderate inducers of SCE; aflatoxin B₁, tris-(2,3-dibromopropyl)phosphate, N-acetoxy-2-acetylaminofluorene, and caffeine were weak inducers. Hoechst 33258 and proflavine induced no SCE. For CPA, MMC, adriamycin, and x-rays, SCE induction was a linear function of dosage. At levels of x-rays needed to induce significant levels of SCE, chromosome aberrations were too numerous to quantify. MMC, CPA, and 4NQO produced the highest ratios of SCE to chromosome aberrations, whereas daunomycin and adriamycin yielded the smallest ratios. X-rays markedly inhibited cellular replication in vitro, as did daunomycin. CPA and adriamycin had relatively little effect on cellular replication. The results indicate that SCE are more sensitive than chromosome aberrations as indicators of chemically induced DNA damage. (35 refs)

- 79-4530 Effect of Ultrasound on Human Leucocytes. Sister Chromatid Exchange Analysis.** (Eng) Morris, S. M. (Dept. Medical Genetics, Univ. Sch. Medicine, 1100 W. Michigan St., Indianapolis, IN 46223);

Palmer, C. G.; Fry, F. J.; Johnson, L. K. *Ultrasound Med Biol* 4(3): 253-258; 1979.

The bromodeoxyuridine-Giemsa method indicated that ultrasound (15.3-36 W/cm²) did not increase the rate of sister chromatid exchanges in human WBC irradiated in the G₀ stage. (28 refs)

- 79-4531 Comparison of Micronuclei Induction for X-Ray and Ultrasound Exposures of *Vicia faba* Root Meristem Cells.** (Eng) Miller, M. W. (Dept. Radiation Biology and Biophysics, Sch. Medicine and Dentistry, Univ. Rochester, Rochester, NY 14642). *Ultrasound Med Biol* 4(3): 263-267; 1978.

X-radiation (200 R), but not ultrasound at a clinically used intensity and frequency (1.1 megahertz, 8 watts/cm² peak, 1-min continuous exposure) induced micronuclei formation in the root meristem cells of *Vicia faba* 6-36 hr after exposure. (24 refs)

- 79-4532 Radiation-induced Osteogenic Sarcoma of C3H Mouse: Effects of *Corynebacterium parvum* and WBI on Its Natural History and Response to Irradiation.** (Eng) Choi, C. H. (Edwin L. Steele Lab. Radiation Biology, Dept. Radiation Medicine, Massachusetts General Hosp., Boston, MA 02114); Sedlacek, R. S.; Suit, H. D. *Eur J Cancer* 15(4): 433-442; 1979.

An osteogenic sarcoma, which appeared at 316 days following a single 5,000 rad dose to the leg of a 100-day-old C3Hf/Sed mouse, was studied as second and third generation transplants (F2 and F3) in syngeneic hosts with respect to local growth, metastasis, and response to local irradiation in normal, immunopotentialized (350 µg *Corynebacterium parvum* iv 96 hr after tumor transplant), and immunosuppressed (whole body irradiated; WBI) hosts. Mean survival after transplantation was 126 days, 115 days, and 173 days for control, WBI, and *C. parvum*-treated mice respectively and was directly correlated with the frequency of spontaneous pulmonary metastases. Incidences of pulmonary metastasis were 95%, 100%, and 58% in control, WBI, and *C. parvum* groups, respectively. In the *C. parvum*-treated group, regression was observed in 10/19 tumors; 3/19 mice were cured of their osteosarcomas and were alive free of disease 12 mo after treatment. Although 5,000 rad local irradiation resulted in destruction of 100% of the leg tumors, 49% of the animals died of metastatic tumor to the lung. In *C. parvum*-treated mice, 16% died of metastatic tumor. Control of half of the irradiated 8 mm diameter tumors was achieved with 4,350 rad and with 3,600 rad for normal and *C. parvum*-treated hosts, respectively. Whole body irradiation (600 rad) given 24 hr before tumor transplant had a small adverse effect. (17 refs)

79-4533 The Ozone Shield and Skin Cancer (Meeting Abstract). (Eng) De Gruijl, F. R. (Inst. Dermatology, State Univ. Utrecht, Utrecht, Netherlands); Van Der Leun, J. C. *J Invest Dermatol* 72(5): 275-276; 1979 (no refs)

79-4534 Light-induced Skin Cancer and Prolonged UV-Erythema (Meeting Abstract). (Eng) Jung, E. G. (Dept. Dermatology, Stadt Krankenanstalten, Postfach 23, D 6800 Mannheim, W. Germany); Furtwangler, M.; Klostermann, G.; Bohnert, E. *J Invest Dermatol* 72(5): 272; 1979 (no refs)

79-4535 Topical Retinoic Acid (RA) and Ultraviolet (UV) Carcinogenesis (Meeting Abstract). (Eng) Epstein, J. H. (Dept. Dermatology, Univ. California, San Francisco, CA); Grekin, D. A. *J Invest Dermatol* 72(5): 272; 1979 (no refs)

79-4536 Stages of Connective Tissue Alteration and Tumor Development Under UVA Radiation in Mice Skin (Meeting Abstract). (Eng) Mahrle, G. (Universitäts-Hautklinik, Göttingen, W. Germany); Berger, H.; Kolmel, K. *J Invest Dermatol* 72(5): 272; 1979 (no refs)

79-4537 Detection of Tumor-associated Cell Membrane Antigens in a UV-induced Mouse Sarcoma by the Ferritin-Hybrid Antibody Method. (Ger) Lageman, A. (Institut für Allgemeine Pathologie und Pathologische Anatomie, Medizinische Akademie, Nordhauser Strasse 74, DDR-50 Erfurt, E. Germany); Pasternak, L.; Pasternak, G.; Dietz, W. *Acta Biol Med Ger* 37(11/12): 1729-1734; 1978.

The demonstration of tumor-associated membrane antigens in a UV-induced mouse sarcoma by the ferritin-hybrid antibody (FHA) technique and by immunofluorescence is described. The sarcoma (UV 15264) was induced in inbred XVII/B1n mice by long-term UV irradiation, and it was transplanted as an ascites tumor by ip application of tumor cells from an im graft. Membrane immunofluorescence was noted in 90% of the cells with immune serum, in 50% with normal serum, and in 15% with conjugated fluorescein isothiocyanate-labeled anti-mouse IgG. The FHA technique showed two forms of ferritin deposition: in a single layer at equal distances from the cell membrane and in the electron-dense, cloudy material on the cell membrane. The first form of ferritin deposition was apparently due to reaction with tumor-associated antigens, as it occurred only after incubation of the cells with

immune serum. The second form of ferritin deposition was seen after incubation of the cells with normal serum, hybrid antibodies, or with ferritin alone; ie, this deposition corresponded to the membrane fluorescence seen in control experiments. The results show that UV-induced sarcoma cells adsorb immunoglobulins, specific antibodies, or antigen-antibody complexes in vivo. The FHA technique thus permits reactions with normal sera and with immune sera to be distinguished clearly. (8 refs)

79-4538 Epidermal Dystrophy: Occurrence after Psoriasis Therapy with Psoralen and Long-Wave Ultraviolet Light. (Eng) Cox, A. J. (Dept. Dermatology, Stanford Univ. Medical Center, 300 Pasteur Drive, Stanford, CA 94305); Abel, E. A. *Arch Dermatol* 115(5): 567-570; 1979.

Thirty-seven patients receiving psoralen and long-wave UV radiation (PUVA) therapy for psoriasis were studied over a 1-yr period. Punch biopsy specimens were obtained from clinically uninvolved and involved skin of a sunlight-protected part of the body prior to the onset of therapy and after 1 yr. After 1 yr, biopsy specimens were also obtained from sunlight-exposed clinically uninvolved skin. In 19/37 of the 1-yr specimens from sunlight-protected skin, there were dystrophic epidermal changes consisting of irregularly scattered small foci of abnormal keratinocytes that were distinctly different from the irregular cells sometimes present in pretreatment control specimens. Abnormally large hyperchromatic nuclei occurred in single cells or small cell clusters, and there was focal disorientation of keratinocytes with respect to the usual progression of maturation. Occasionally, a large cell was in mitosis. Epidermal dystrophy was present in regions of regressed psoriatic lesions, but it was usually less prominent than that in clinically uninvolved skin. The specimens from sunlight-protected, clinically uninvolved skin procured at the time the lesions had first cleared under PUVA therapy showed changes similar to those seen at 1 yr. No relationship was identified between degree of epidermal change and total UV dose, skin type, treatment schedule, age, sex, or response of the psoriasis to therapy. The epidermal changes may represent transient effects, but it is also possible that the cells have been altered genetically by a somatic mutation that may result in skin cancer. (19 refs)

79-4539 Photochemotherapy of Psoriasis Using a New Mono Functional Psoralen Noncarcinogenic in Mice (Meeting Abstract). (Eng) Dubertret, L. (Hopital Mondor, Creteil, France); Averbeck, D.; Bisagni, E.; Touraine, R. *J Invest Dermatol* 72(5): 278-279; 1979 (no refs)

79-4540 Measurement of DNA Crosslinks by S_1 Nuclease: Induction and Repair in Psoralen-Plus-360 nm Light Treated *Escherichia coli*. (Eng) Ben-Hur, E. (Nuclear Research Centre-Negev., P.O. Box 9001, Beer-Sheva, Israel); Prager, E.; Riklis, E. *Photochem Photobiol* 29(5): 921-924; 1979.

DNA crosslinks in *Escherichia coli* cells exposed to 4,5',8-trimethylpsoralen (TMP) plus near UV light (360 nanometers) were measured by a rapid and sensitive new method. The approach is based on the specificity of S_1 nuclease from *Aspergillus oryzae* to resolve single from double-stranded DNA. Bacterial cells were lysed and the DNA denatured by alkali. Following acid neutralization, crosslinked DNA undergoes spontaneous renaturation and is rendered S_1 -nuclease-resistant and, therefore, acid-precipitable. The single-stranded fraction remaining after alkali denaturation decreases with increasing near UV light in the presence of TMP, the decrease following first-order kinetics. The reaction rates were faster when exposure was at 4 C rather than at 20 C, which suggested that excision of crosslink occurs during exposure at the higher temperature. Since the rate of DNA crosslinking in an excision-deficient *uvr B* mutant was higher than in wild-type bacteria at 4 C, some excision must have occurred even at 4 C. DNA from excision-proficient cells incubated at 37 C following exposure to EMP-plus-near UV at 4 C showed a greater single-stranded fraction than that from nonincubated cells. This indicates repair of DNA crosslinks, which proceeded with a half-time of 8 min at 37 C and was unaffected by substitution of thymine in DNA by 5-bromouracil. (22 refs)

79-4541 Age-related Decrease of Ultraviolet Light-induced DNA Repair Synthesis in Human Peripheral Leukocytes. (Eng) Lambert, B. (Dept. Clinical Genetics, Karolinska Hosp., S-104 01 Stockholm 60, Sweden); Ringborg, U.; Skoog, L. *Cancer Res* 39(7, part 1): 2792-2795; 1979.

The capacity for UV light-induced DNA repair synthesis, studied in peripheral WBC from 58 healthy subjects 13-94 yr old, was found to vary greatly between individuals. A negative, statistically significant correlation was obtained between age and DNA repair synthesis, indicating a decrease in repair capacity with age. An age-related decrease in DNA repair may increase the susceptibility of cells to agents causing DNA damage, ie, carcinogens and certain cytostatic drugs. (26 refs)

79-4542 Overlapping Pathways for Repair of Damage from Ultraviolet Light and Chemical Carcinogens in Human Fibroblasts. (Eng) Brown, A. J. (Univ. Tennessee-Oak Ridge Graduate Sch. Biomedical Sciences, Oak Ridge, TN 37830); Fickel, T. H.; Cleaver, J. E.;

Lohman, P. H.; Wade, M. H.; Waters, R. *Cancer Res* 39(7): 2522-2527; 1979.

DNA excision repair was measured in cultured human fibroblasts after single or dual treatments with UV radiation, 4-nitroquinoline 1-oxide (4NQO), or N-acetoxy-2-acetylaminofluorene (AAAF). Three approaches were used to monitor repair: unscheduled DNA synthesis, measured by autoradiography; repair replication, measured by the incorporation of a density-labeled DNA precursor into repaired regions; and excision of UV endonuclease-sensitive sites. When a single repair-saturating dose of one of the three carcinogens was administered, little stimulation of unscheduled DNA synthesis or repair replication occurred in response to additional treatment with one of the other carcinogens. In no instance was total additivity of repair observed. These observations were confirmed when the excision of endonuclease-sensitive sites produced by UV damage (ie, pyrimidine dimers) was inhibited by exposure to 4NQO and AAAF. The data indicate that the repair of lesions induced by these agents may have common rate-limiting steps, a conclusion supported by the repair deficiency in xeroderma pigmentosum cells in which a single mutation eliminates the repair of damage caused by each of the agents. (28 refs)

79-4543 Mutagenicity of 8-Methoxypsoralen and Long-Wave Ultraviolet Irradiation in V-79 Chinese Hamster Cells. A First Approach to a Risk Estimate in Photochemotherapy. (Eng) Burger, P. M. (Dept. Dermatology, Univ. Medical Centre, Leiden, Netherlands); Simons, J. W. *Mutat Res* 60(3): 381-389; 1979.

The effect of 8-methoxypsoralen (8-MOP) and long-wave UV irradiation (UVA) on cell killing and mutation induction was studied in V-79 Chinese hamster cells. No effect was observed after treatment of the cells with 8-MOP alone ($50 \mu\text{g/ml}$, 4 hr), UVA alone ($9,000 \text{ joules/m}^2$), or 8-MOP metabolized by rat liver microsomes. Combined treatment with 8-MOP and UVA induced both cell killing and mutation. These effects were also observed under conditions approaching those used to treat patients with psoralen + UVA (PUVA) photochemotherapy, with respect to the concentration of 8-MOP in the skin and the amount of UVA received by the epidermal cells. A simple relation proved to apply to mutation induction under different treatment conditions: 5.5×10^{-8} per J/m^2 per μg 8-MOP/ml. On this basis, the mutation induction in dividing cells per session of PUVA photochemotherapy amounts to 12.4×10^{-5} , which is probably an overestimation because of differences between hamster and human cells with respect to excision repair capacity. In addition, most cells are in G_0 in vivo, and thus more time is available for excision repair. (36 refs)

79-4544 Biological Effects of Near-UV Radiation 2'-Acetylformanilide-sensitized Formation of Hydrogen Peroxide from Nucleosides and Nucleotides. (Eng) McCormick, J. P. (Dept. Chemistry, Univ. Missouri, Columbia, MO 65211); Oczos, A. *Photochem Photobiol* 29(5): 1041-1044; 1979.

2'-Acetylformanilide has been found to be an effective, near-ultraviolet (300-380 nanometers) sensitizer for the photooxidation of nucleosides and nucleotides in aqueous soln, with hydrogen peroxide being formed in high yield. The decreasing order of hydrogen peroxide formation and substrate destruction was found to be: guanosine, adenosine, thymidine, uridine and cytidine. The process was highly pH dependent, low pH being most favorable for photooxidation. Experiments using deuterium oxide and superoxide dismutase indicate that both singlet oxygen and superoxide ion can be involved in hydrogen peroxide formation. (23 refs)

79-4545 Repair of Near (365 nm)- and Far (254 nm)-UV Damage to Bacteriophage of *Escherichia Coli*. (Eng) Tyrrell, R. M. (Instituto de Biofisica, Centro de Ciencias da Sauder, Universidade Federal do Rio Janeiro, Rio de Janeiro, Brazil). *Photochem Photobiol* 29(5): 963-970; 1979.

Intact *Escherichia coli* bacteriophage T4 and its three repair-defective mutants were irradiated at 365 nanometers (nm) or at 254 nm and then analyzed for DNA photoproducts, or they were injected into their bacterial host to test susceptibility of UV damage to phage and host-cell mediated repair systems. Both thymine dimers and single-strand breaks were induced in the phage DNA by 365 nm radiation. The dimers appeared to be the major lethal lesion (approx 2 dimers/lethal event) in repair-deficient bacteriophage T4 and in bacteriophage λ after irradiation with 254 nm or 365 nm radiation. Damage induced in T4 by either wavelength was equally susceptible to x -gene reactivation (repair sector of approx 0.5); v -gene reactivation acted on a larger fraction of the near-UV damage (repair sector of 0.82 at 365 nm vs 0.66 at 254 nm). The host-cell mediated photoreactivation system was only slightly less effective for near-UV damage, but host-cell reactivation (as measured by comparing survival of phage λ on a uvr^+ and a uvr^- host) was effective against a far smaller sector of near-UV damage (0.35) than far-UV damage (0.85). Weigle-reactivation (far-UV induced) of near-UV damage to phage λ was not observed. The results suggest that unless the near-UV damaged phage DNA is repaired immediately after injection, the lesions rapidly lose their susceptibility to repair with a consequent loss of activity of the phage particles. (28 refs)

79-4546 Netropsin: Interaction with Ultraviolet Irradiated DNA. (Eng) Sutherland, J. C.

(Biology Dept., Brookhaven Natl. Lab., Upton, NY 11973); Duval, J. F.; Farland, W. H.; Griffin, K. P. *Photochem Photobiol* 29(5): 943-949; 1979.

Ultraviolet irradiation of double-stranded DNA reduces the circular dichroism (CD, $\lambda > 300$ nm) induced when the basic peptide antibiotic netropsin (Nt) is added to DNA subsequent to the irradiation, compared with the CD induced by the same concentrations of Nt added to unirradiated DNA. Nt is known to bind A-T base pairs in duplex DNA, but it will not bind to single-stranded DNA. The reduction in the max induced CD observed with saturating concentrations of Nt is a linear function of the concentration of pyrimidine dimers that, along with other dinucleotide photoproducts, form short disrupted regions in duplex DNA. The decrease in the CD of Nt bound to irradiated DNA could be due to elimination of potential Nt sites in the vicinity of a dimer, reduction in the av magnitude of the CD of Nt bound near a dimer, or various combinations of these effects. In addition, there is a reduction in the av binding constant for Nt bound to irradiated DNA, compared with unirradiated DNA, suggesting that formation of dinucleotide photoproducts tends to eliminate preferentially the tighter binding sites or that tighter sites are converted to weaker ones. A simple model suggests that no more than one-third to one-half of the pyrimidine dimers formed in DNA completely eliminate a Nt site. (20 refs)

79-4547 Effect of Intensity and Wavelength of Fluorescent Light on Chromosome Damage in Cultured Mouse Cells. (Eng) Parshad, R. (Dept. Pathology, Coll. Medicine, Howard Univ., Washington, DC 20059); Sanford, K. S.; Taylor, W. G.; Tarone, R. E.; Jones, G. M.; Baeck, A. E. *Photochem Photobiol* 29(5): 971-975; 1979.

A single 3- to 20-hr exposure of line NTC 9266 mouse cells to cool-white fluorescent light [4.6 watts (W)/m²] produced chromatid breaks and exchanges. The effective wavelength was in the visible range and coincided with the mercury emission peak at 405 nanometers. Increasing the light intensity from 4.6 W to 15.3 W/m² for 20 hr caused a concomitant increase both in chromosome damage and formation of hydrogen peroxide (H₂O₂) in the serum-free medium. Cells washed free of medium and illuminated in saline for 3 hr showed the same extent of chromosome damage as cells illuminated in culture medium. Addition of catalase during the 3-hr exposure period eliminated the light-induced damage. It is concluded that light-induced chromatid breaks and exchanges result from H₂O₂ production within the cell and that exogenous catalase can enter the cell and prevent this damage. (17 refs)

79-4548 Cell Cycle Mapping by Irradiating Cells with Bromosubstituted DNA Segments. (Eng) de

PHYSICAL CARCINOGENESIS

La Torre, C. (Instituto de Biología Celular (CSIC), Velazquez, 144, Madrid-6, Spain); Gonzales-Fernandez, A. *Photochem Photobiol* 29(5): 977-981; 1979.

The role of chromatin replicated at restricted S intervals in further interphase progression was analyzed by 5-bromodeoxyuridine substitution by black light irradiation (313 nanometer peak) in a naturally synchronous population rendered binucleate by a caffeine pulse in *Allium cepa* L. root meristems. Interphase completion is preferentially halted when irradiating cells with bromosubstituted chromatin that is replicated at the 3rd and 4th interval of an S period artificially divided into 5 portions. The treatment apparently inhibits transcription of the bromosubstituted DNA segments. The failure in the transcription of these segments may be related to the transition point located at early G₂ in these cells, when protein synthesis is needed in order to reach mitosis. (24 refs)

79-4549 Saturation of DNA Repair in Mammalian Cells. (Eng) Ahmed, F. E. (Dept. Biology, Brookhaven Natl. Lab., Upton, NY 11973); Setlow, R. B. *Photochem Photobiol* 29(5): 983-989; 1979.

Excision repair was measured in normal human fibroblasts up to 80 joules (J)/meter² (m²). The four techniques used (unscheduled DNA synthesis, photolysis of bromodeoxyuridine incorporated during repair, loss of sites sensitive to a UV endonuclease from *Micrococcus luteus*, and loss of pyrimidine dimers from DNA) showed little difference between the two doses. Moreover, the loss of endonuclease sites in 24 hr following two 20 J/m² doses separated by 24 hr was similar to the loss observed following one dose. It was concluded that the observed plateau in excision repair is real and does not represent some inhibitory process at high doses but a true saturation of one of the rate limiting steps in repair. (34 refs)

79-4550 Morphological Transformation, DNA Damage, and Chromosomal Aberrations Induced by a Direct DNA Perturbation of Synchronized Syrian Hamster Embryo Cells. (Eng) Tsutsui, T. (Natl. Inst. Environmental Health Sciences, P.O. Box 12233, Research Triangle Park, NC 27709); Barrett, J. C.; Ts'o, P. O. *Cancer Res* 39(7): 2356-2365; 1979.

The cellular effects of a direct perturbation of DNA during various portions of the DNA synthesis period (S phase) were examined. Early-passage Syrian hamster embryo cells (HEC) were synchronized by growth in medium containing 1% serum, followed by hydroxyurea treatment. This method resulted in about 80% of the cells entering S phase synchronously and did not induce any detectable chromosome abnormalities. Cells at different periods in the S phase were treated for 1 hr with 5-bromodeoxyuridine

(BUdR), followed by irradiation with near UV. This treatment induced chromosome aberrations and DNA damage, as measured by changes in sedimentation profile in alkaline sucrose gradients. No specific period during S phase was significantly more sensitive to treatment with respect to cell survival, chromosome aberrations, or DNA damage and repair. The induction of morphological transformation was also cell phase dependent, occurring only in cells synthesizing DNA. However, the incidence of morphological transformation was dependent on the portion of the S phase during which treatment was administered. The frequency of morphological transformation was highest in cells treated in early to middle S phase, particularly in the second hr of S phase. No transformation was observed in the late S, G₁-S boundary, and G₂ phases. BUdR treatment or irradiation alone induced no changes. These results suggest that certain region(s) in the DNA of Syrian HEC, designated by their specific temporal relationship in the S phase, may be the most sensitive targets for perturbation by BUdR treatment + near-UV irradiation and subsequent initiation of morphological transformation. (45 refs)

79-4551 Transport of Asbestos in Cultured Tomato Root Tips (Meeting Abstract). (Eng) Risner, R. J. (Ohio State Univ., Columbus, OH 43210); Sharp, W. R. *In Vitro* 15(3): 229-230; 1979 (no refs)

79-4552 Analysis of the Cores of Ferruginous (Asbestos) Bodies from the General Population. III. Patients with Environmental Exposure. (Eng) Churg, A. M. (Dept. Pathology, Univ. California at San Francisco, Sch. Medicine, San Francisco, CA 94143); War-nock, M. L. *Lab Invest* 40(5): 622-626; 1979.

The cores of asbestos (AS) bodies obtained from individuals with low AS body counts were analyzed. Typical AS bodies visible by light microscopy were isolated from the lungs of 29 persons with < 100 such bodies per gram of lung, a level considered indicative of environmental rather than occupational AS exposure. Of 144 bodies examined by electron diffraction, 143 contained an amphibole AS core and 1 contained a chrysotile AS core. Thirty-five bodies from 21 patients were also analyzed by electron microprobe. Of these, 21 were chemically consistent with amosite or crocidolite AS, 13 with anthophyllite AS, and 1 with tremolite AS. Certain differences in chemical fiber types between men and women became apparent. Although cores of amosite and crocidolite predominated in men (12/14, 86%) anthophyllite and tremolite comprised 57% (12/21) of the cores found in women, a statistically significant difference. These differences suggest that the major commercial varieties of amphibole AS (amosite and crocidolite) are the source of the fibers in men, whereas in women a major source may be cosmetic talc, which is often contaminated with anthophyllite and tremolite. On the

basis of this study and previous studies, it is concluded that almost all typical AS bodies from the lungs of the general population contain an amphibole AS core. (17 refs)

79-4553 Trauma and Hodgkin's Disease. (Eng) Bichel, J. (Siriusvej 1A, DK-8270 Højbjerg, Denmark). *Acta Med Scand* 205(4): 347-349; 1979.

The development of Hodgkin's disease (HD) following trauma in two patients is reported. A 37-yr-old man developed a tumor in the region of the upper sternum where he had received a blow with a fist 1 mo earlier. The histologic appearance was that of HD. A 53-yr-old man developed lymphogranulomatosis between the first and fourth lumbar vertebrae 1 yr after falling from a ladder and striking his back on the ground. Postmortem examination showed enlarged lymph nodes along the spine and infiltrations in the liver, spleen, and right kidney. These cases are suggestive of a causal relationship between trauma and

HD. However, the possibility exists that these patients may have suffered from latent HD before they sustained trauma, with the trauma being an HD-localizing factor. (7 refs)

See also

- *(Rev.): 79-4207, 79-4209, 79-4210, 79-4220, 79-4265,
79-4266, 79-4267, 79-4268, 79-4269, 79-4270,
79-4271, 79-4272, 79-4273, 79-4274.
- *(Chem.): 79-4389, 79-4415, 79-4475.
- *(Viral): 79-4595, 79-4641, 79-4683.
- *(Epid.-Biom.): 79-4739, 79-4743, 79-4747, 79-4749,
79-4756, 79-4758, 79-4759, 79-4763,
79-4764, 79-4773, 79-4777, 79-4781.

VIRAL CARCINOGENESIS

79-4554 Hydroxyapatite Chromatographic Detection of Tumor Virus Specific Genes (Meeting Abstract). (Eng) Scola-Nagelschneider, G. (Dept. Dermatology, Univ. Munich, Munich, W. Germany); Balda, B. R. *J Invest Dermatol* 72(5): 269; 1979 (no refs)

79-4555 Electron Microscope Analysis of DNA Products of Reverse Transcription. (Rus) Ryn-dich, A. V. (Inst. Molecular Biology and Genetics, Kiev, USSR); Maniakov, V. F.; Mazaev, A. G.; Hahn, F.; Hunger, H. D.; Samarina, O. P.; Kavan, V. M. *Mol Biol (Mosk)* 13(2): 337-346; 1979.

Electron microscopy was used to analyze the length of the DNA products of reverse transcription (complementary DNA, cDNA). The pre-messenger RNA (pre-mRNA) from rat liver and rabbit erythroid bone marrow cells was used. Rabbit globin mRNA was used as a template, and the RNA-dependent DNA polymerase was isolated from chicken myeloblastosis virus. It was found that the rabbit globin mRNA consisted of two types of molecules (av length 0.1 and 0.2 μ m, respectively). The number of short molecules was almost two times greater than the number of long ones. The DNA product of the reverse transcription of rabbit globin mRNA was also represented by two types of molecules, and their lengths were the same as those comprising the template. The cDNA synthesized in the presence of actinomycin D had a single-stranded structure, but the cDNA synthesized in the absence of actinomycin D on the pre-mRNA template (ie, without the inhibition of synthesis of the second strand) contained both single- and double-stranded molecules. Approx 10% of the double-stranded molecules had a branched structure. (27 refs)

79-4556 Primer tRNA^{Trp} Enhances the Inhibition of Avian Myeloblastosis Virus Reverse Transcriptase by Pyridoxal-5'-phosphate. (Eng) Araya, A. (Departement de Biochemie, UER-BBC, Universite de Bordeaux II, 3561 Cours de la Liberation, 33405 Talence, France); Labouesse, J.; Litvak, S. *Biochem Biophys Res Commun* 88(1): 9-15; 1979.

The effect of primer tryptophan transfer RNA (tRNA-^{Trp}) on the inhibition of avian myeloblastosis virus (AMV) reverse transcriptase (RT) by pyridoxal-5'-phosphate (PLP) was studied. RT was inactivated by preincubation with PLP, about 70% inactivation being obtained with 2 mM. In the presence of beef tRNA-^{Trp}, the same degree of inactivation was achieved with 0.6 mM PLP. The α subunit of

AMV RT could also be inhibited by PLP, but tRNA-^{Trp} did not enhance the effect. The inactivation of AMV RT in the presence of 1.2 mM PLP was a function of tRNA-^{Trp} concentration, a plateau of 90% inhibition being attained between 2 and 5 μ M tRNA. tRNA did not affect the activity of AMV RT when poly A-dT₁₂ was used as template. Thymidine triphosphate (10 mM) prevented the inhibition by PLP, but it only partially prevented the inhibition by PLP plus tRNA-^{Trp}. Yeast tRNA-^{Trp} did not enhance PLP-induced inhibition as effectively as beef tRNA-^{Trp}, and beef cellular valine tRNA had no effect at all. In the presence of beef tRNA-^{Trp}, two additional lysines were titrated with PLP as measured by reduction of the enzyme-PLP complex with ³H-NaBH₄. The β subunit of AMV RT appeared to be involved in the primer-enzyme interaction. (23 refs)

79-4557 Isolation and Characterization of a Large 'Hairpin' Segment from Avian Retrovirus RNA. (Eng) Perdue, M. L. (Dept. Pathology, Div. Experimental Pathology, Univ. Kentucky, Lexington, KY 40506); Wunderli, W.; Joklik, W. K. *Virology* 95(1): 24-35; 1979.

Avian retrovirus RNA (both 70S and 35S) possesses several attributes of double-stranded (ds) RNA's, including an affinity for hydroxyapatite equal to that of authentic ds RNA's and exceeding that of any other single-stranded RNA tested except heterogenous nuclear RNA. Some of its sequences also exhibit an unexpectedly high melting temperature, which indicates the presence of intramolecular base-paired regions. Limited digestion with pancreatic RNase A of the Prague C strain of Rous sarcoma virus (RSV), B77 virus or Rous-associated virus 2 35S RNA yielded a product that accounted for about 7% of the total viral RNA, behaved like reovirus ds RNA when chromatographed in hydroxyapatite, possessed a T_m that was similar to that of reovirus ds RNA, was almost as susceptible to RNase III as reovirus ds RNA under conditions when reovirus messenger RNA was completely resistant, and could be isolated as a relatively homogeneous component following centrifugation in sucrose density gradients or electrophoresis in formamide-containing polyacrylamide gels. These properties indicated that the product is a highly (but not perfectly) base-paired hairpin about 350 base pairs long. It was mapped by determining which of various size classes of poly(A)-containing fragments of viral RNA contained it. It was located in the region between 5,000 and 6,000 nucleotides from the 3'-terminus of nondefective viral RNA; this region is at, or close to, the junction of the *pol* and *env* genes. The fact

that the RNA of the helper virus free Bryan high-titer strain of RSV, which lacks most of the *env* gene, did not yield such a hairpin fragment agrees with this conclusion. (44 refs)

- 79-4558 Glucose-specific Cytochalasin B Binding Is Increased in Chicken Embryo Fibroblasts Transformed by Rous Sarcoma Virus.** (Eng) Salter, D. W. (Dept. Microbiology, Univ. Illinois, Urbana, IL 61801); Weber, M. J. *J Biol Chem* 254(9): 3554-3561; 1979.

Radiolabeled cytochalasin B was used to determine if the increased hexose transport rate in transformed chicken embryo fibroblasts is due to an increased number of hexose carriers. The difference in glucose-specific cytochalasin B binding between normal and transformed chicken embryo fibroblasts was found to correlate closely with the difference in the hexose transport rate. (57 refs)

- 79-4559 Continuous Lines of RSV-transformed Embryo Cells and Peritoneal Macrophages of Japanese Quails.** (Eng) Yamanouchi, K. (Dept. Measles Virus, Natl. Inst. Health, Musashi-Murayama, Tokyo 190-12, Japan); Yoshikawa, Y.; Hayami, M.; Hishiyama, M.; Boschek, C. B. *Jpn J Med Sci Biol* 32(1): 19-35; 1979.

The establishment and characterization of four continuous lines of Rous sarcoma virus-transformed quail cells, two derived from quail embryo cells (QERC-31F and QERC-31N) and two derived from adult quail peritoneal macrophages (PERP and PERY) are described. A marked morphological difference was noted between QERC-31F and QERC-31N: the former had a fusiform shape and the latter a nodular shape. Both PERP and PERY had a macrophage like morphology and phagocytic capacity. All four cell lines contained group-specific antigen and an 85,000-dalton virion envelope glycoprotein (gp85). Production of transforming virus was found in QERC-31N, PERP, and PERY. Although it did not produce transforming virus, QERC-31F was demonstrated to produce C-type particles by electron microscopy and to contain tumor-specific surface antigen by in vivo immunization and in vitro microcytotoxicity tests. (22 refs)

- 79-4560 Partial Structure of a Membrane Glycopeptide from Virus-transformed Hamster Cells.** (Eng) Santer, U. V. (Joseph Stokes Jr. Res. Inst., Children's Hosp., Philadelphia, PA 19104); Glick, M. C. *Biochemistry* 18(12): 2533-2540; 1979.

The isolation, purification, and partial structure of one of the major glycopeptides from the surface of a clone of Rous sarcoma virus-transformed baby hamster kidney cells

(C₁₃/B₄) are reported. The cells were metabolically labeled with L-[¹⁴C]fucose. This glycopeptide represented 19% of the total radioactivity removed by trypsin from the cell surface of the transformed fibroblasts, and it was more abundant in the transformed cells than in their normal counterparts. Purification of the glycopeptide after digestion with Pronase was accomplished by successive chromatography on diethylaminoethylcellulose and Sephadex G-50. The monosaccharide content of the glycopeptide was 42, 127, 138, 114, and 243 nanomoles of fucose, sialic acid, galactose, mannose, and glucosamine, respectively. A partial structure of the glycopeptide was proposed from the results of sequential enzymatic degradation coupled with gas-liquid chromatographic analysis of the resultant monosaccharides. All of the enzymes used were purified and pretested on natural substrates and found to remove terminal monosaccharides of the correct configuration quantitatively. The purification and properties of an α -L-fucosidase from rat testes are described. All of the radioactivity in the glycopeptide, recovered as fucose, was present at the core and was removed by treatment with this α -L-fucosidase. The proposed structure is a triantennary, completely sialylated, complex glycopeptide containing a core region of β -D-mannose, β -D-N-acetylglucosamine, and α -L-fucose. (56 refs)

- 79-4561 Membrane Glycopeptides from Virus-transformed Hamster Fibroblasts and the Normal Counterpart.** (Eng) Glick, M. C. (Dept. Pediatrics, Sch. Medicine, Univ. Pennsylvania, Philadelphia, PA 19104). *Biochemistry* 18(12): 2525-2532; 1979.

The membrane glycopeptides from baby hamster kidney fibroblasts (BHK₂₁/C₁₃) and from a clone transformed by Rous sarcoma virus (C₁₃/B₄) were compared with the use of cells metabolically labeled with radioactive D-glucose and L-fucose. Most of the glycopeptides were metabolically labeled with both the general and specific glycoprotein precursors. The glycopeptides obtained from the cell surface by controlled trypsinization were representative of the surface membrane, as shown by comparisons with those of purified membrane preparations. The trypsin-removable glycopeptides from both cell types were further processed and examined by successive chromatography on Sephadex G-50 and diethylaminoethylcellulose. The chromatographic distribution patterns showed that each cell type had glycopeptides of similar characteristics, but dramatically different proportions. After transformation, there was an increase in the larger, more highly charged glycopeptides. This was verified by the increased sialic acid content in these glycopeptides. Some of the glycopeptides were homogeneous after the size and charge separations, since a variety of procedures did not separate them further. The apparent homogeneity and reasonably few species obtained may be due to the isolation methods, which may select particular glycopeptides from the external portion of the membrane. The results corroborate this concept and

show for the first time that virus transformation is accompanied by an increase in, rather than de novo synthesis of, certain species of glycopeptides. (34 refs)

- 79-4562** Transfection Studies in Vitro and In Vivo with Isolated Marek's Disease Virus DNA. (Eng) Kaaden, O. R. (Institut für Virologie, Tierarztl. Hochschule, Bunteweg 17, 3000 Hannover, 71, W. Germany). *IARC Sci Publ* 24(II): 627-633; 1978.

For transfection studies in vitro and in vivo, infectious Marek's disease virus (MDV) DNA was isolated from purified virus particles. Its sedimentation coefficient in neutral sucrose gradients was found to be $53 \pm 2S$ and the buoyant density in cesium chloride corresponded to 1.707 g/cm^3 . The virus DNA was also analyzed by using the restriction endonucleases *EcoRI* and *HindIII*. High mol wt MDV DNA precipitated by the calcium phosphate technique was shown to be infectious in cultivated chicken embryo fibroblasts and newly hatched antibody-free chickens. Three out of eight chickens receiving between 0.5 and $2 \mu\text{g}$ MDV DNA by the intraabdominal route produced precipitating A-antigens in the feather follicles and/or lymphoproliferative tumors in the visceral organs. Infectious MDV was also detected in spleen and blood lymphocytes. Transformation experiments in vitro performed with isolated thymus cells, however, have so far produced negative results. The induction of Marek's disease and tumors by purified MDV DNA also indicates that the presence of contaminating avian oncornaviruses in the inoculum is not a necessary condition for the initiation of an MDV infection. (4 refs)

- 79-4563** Effect of Cyclic Nucleotides on the Response of Cells to Infection by Various Herpesviruses. (Eng) Newton, A. A. (Dept. Biochemistry, Univ. Cambridge, Cambridge, England). *IARC Sci Publ* 24(I): 381-387; 1978.

The effects of varying intracellular cyclic nucleotide concentrations on the growth of herpes simplex virus type 1 in mouse fibroblasts (strain L) and on the growth of Marek's disease virus in duck embryo fibroblasts were studied. In each case, agents that elevated the intracellular cyclic AMP levels [ie, prostaglandin E_1 , low (10%) serum, theophylline] increased the yield of infectious virus or viral DNA, but inhibited cell division. Cyclic guanosine monophosphate enhanced the synthesis of viral DNA but allowed the continued synthesis of cell DNA. (6 refs)

- 79-4564** Altered Biological and Biochemical Properties of a Phosphonoacetate-resistant Mutant of Herpesvirus of Turkeys. (Eng) Lee, L. F. (Science and

Education Administration-Federal Res., Regional Poultry Res. Lab., U.S. Dept. Agriculture, East Lansing, MI); Nazerian, K.; Witter, R. L.; Leinbach, S. S.; Boezi, J. A. *IARC Sci Publ* 24(I): 253-260; 1978.

A phosphonoacetate (PA)-resistant mutant of the herpesvirus of turkeys (HVT) was isolated and characterized. The mutant (HVTpa) replicates in growth medium containing $300 \mu\text{g/ml}$ of PA and shows in vitro temperature sensitivity at 41°C (its $37^\circ\text{C}/41^\circ\text{C}$ efficiency of replication is about 5). HVTpa replicates poorly in chickens and fails to provide complete protection against MDV challenge. The HVTpa-induced DNA polymerase has an apparent inhibition constant for PA 10 times as great, an apparent inhibition constant for pyrophosphate twice as great, and an apparent Michaelis constant for deoxycytidine triphosphate 2.5 times as great as the respective figures for the wild-type HVT-induced enzyme. The HVTpa-induced enzyme is also more thermolabile. (8 refs)

- 79-4565** An MSV-specific Subgenomic mRNA in MSV-transformed G8-124 Cells. (Eng) Donoghue, D. J. (Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA 02139); Sharp, P. A.; Weinberg, R. A. *Cell* 17(1): 53-63; 1979.

An intracellular subgenomic RNA species from murine sarcoma virus (MSV)-transformed G8-124 cells was characterized by electron microscopy of RNA: complementary DNA (cDNA) heteroduplexes using long cDNA's of both MSV and murine leukemia virus (MuLV). This subgenomic RNA, 3.1 kilobases (kb) long, consisted of 5'-derived sequences of about 0.4 kb joined to 2.7 kb of RNA derived from the 3' end of the RNA genome. These 3'-derived sequences included the residual sequences from the MuLV *pol* region and the acquired cellular sequences of MSV. The MSV genome was shown to retain approx 0.13 kb from the 5' end of the MuLV *env* region, including sequences that span the splice point in the MuLV *env* messenger RNA (mRNA). However, no subgenomic MSV RNA could be detected that consisted of a 5'-derived leader sequence spliced to the retained *env* region sequences, nor could a subgenomic MSV RNA be detected in which a 5'-derived leader sequence was joined directly to the acquired cellular sequences. Although its translation products are unknown, the subgenomic MSV RNA was present in preparations of poly(A) + polysomal RNA, consistent with this RNA functioning as a messenger. The structure of this 3.1-kb MSV subgenomic RNA suggests that it functions as an mRNA in the expression of 3'-encoded MSV information, possibly including transformation-specific sequences. (35 refs)

- 79-4566** Fusion Activity of Virions of Murine Leukemia Virus. (Eng) Zarling, D. A. (Dept.

Pathology, Univ. Wisconsin, Madison, WI 53706); Keshet, I. *Virology* 95(1): 185-196; 1979.

The fusion activity associated with many strains of murine leukemia virus (MLV) is commonly used to measure MLV infectivity. Other MLV strains lack fusion activity, although some can produce variants that have fusion activity. The mechanism of MLV-induced fusion was investigated by the SC-1/uv-XC fusion assay, which measures the infectivity of MLV grown in SC-1 cells by the XC cell fusion activity of the progeny virus. The fusion activity of parental MLV in XC cells was measured directly within a few hours after infection. No new macromolecular synthesis (early after infection) was required for MLV-induced fusion. Treatment of MLV virions with proteolytic enzymes destroyed their ability to fuse XC cells. The fusion activity of MLV, like the infectivity of MLV, was specifically inhibited by anti-MLV gp70 serum but not by antiserum against other MLV polypeptides. MLV infectivity was much more susceptible to UV irradiation than was viral fusion activity. Noninfectious (UV irradiated) MLV also caused XC cell fusion, suggesting that fusion activity was associated with a virion component. In MLV virions disrupted with NP-40 detergent, fusion activity was restored after high-speed centrifugation and dialysis of the detergent. The fusion activity of NP-40 disrupted MLV (after high-speed centrifugation and dialysis) was inhibited by antiserum against MLV gp70. Thus, MLV fusion activity is associated with the virion envelope glycoprotein. (31 refs)

79-4567 AKR Leukemogenesis: Identification and Biological Significance of Thymic Lymphoma Receptors for AKR Retroviruses. (Eng) McGrath, M. S. (Lab. Experimental Oncology, Dept. Pathology, Stanford Univ. Sch. Medicine, Stanford, CA 94305); Weissman, I. L. *Cell* 17(1): 65-75; 1979.

A quantitative virus-binding assay was used to determine whether in vivo thymocytes of preleukemic and leukemic AKR mice bear receptors for XC+ N-ecotropic viruses, MCF viruses (recombinant retroviruses causing mink fibroblast foci), and XC- SL viruses (retroviruses that do not cause XC plaques or mink fibroblast foci). Within the normal thymus, 0.5%-2.5% of the cells bore receptors for murine leukemia virus (MuLV). Each MuLV-induced T cell lymphoma was made up of cells that bore receptors specific for the inducing MuLV; in two such lymphomas, the relative binding efficiency was $SL > MCF-247 \gg$ AKR N-tropic viruses. In the preleukemic period, most thymocytes produced MuLV proteins and expressed them as antigens. Only a small percentage of cells expressed MuLV receptors. On transfer to syngeneic mice, virus antigen-positive AKR retrovirus receptor-negative cells were not tumorigenic, whereas AKR retrovirus receptor-positive cells were tumorigenic. It is concluded that the presence of specific cell surface receptors for lymphoma

cell-produced and recombinant AKR retroviruses is a marker for leukemia in these hosts. (32 refs)

79-4568 Effect of Elevated Temperature on AKR Leukemia Virus Production in AKR Mouse Cells. (Eng) Tomita, Y. (Dept. Microbiology, Sch. Medicine, Chiba Univ., Chiba, Japan); Kuwata, T.; Takayama, N. *Microbiol Immunol* 23(1): 45-50; 1979.

The effect of elevated temperature (40 C) on the multiplication of AKR murine leukemia virus (AKR-MuLV) in AKR mouse cells and of Rauscher (R)-MuLV in BALB/c mouse cells (JLS-V9) and C57BL/6 mouse cells (R-17) was studied. The K3b cell line (derived from a Rous sarcoma virus-induced sarcoma in an AKR mouse) and the K3b4Or line (a variant that grows well at 40 C) were used. As determined by assay of virion-associated reverse transcriptase (RT), production of AKR-MuLV at 40 C was reduced to < 34% and < 20% of control levels at 24 and 48 hr, respectively. R-MuLV production remained at > 86% of the control level at 24 hr. Suppression of AKR-MuLV production in K3b4Or cells was reversed in a time-dependent fashion after a shift from 40 C to 37 C. The decrease in RT activity at 40 C did not appear to be due merely to heat inactivation. A few virus particles were produced and released into the medium of K3b4Or cells even at 40 C. This temperature was also nonpermissive for AKR-MuLV replication in AKR 2B cells. Despite the presence of a thermosensitive step(s) during AKR-MuLV multiplication, the complete viral genome was stable at 40 C once it had been integrated into the host cell DNA. (15 refs)

79-4569 Expression of Endogenous Murine Leukaemia Viruses in AKR/J Streaker Mice. (Eng) Bedigian, H. G. (Jackson Lab., Bar Harbor, ME 04609); Shultz, L. D.; Meier, H. *Nature* 279(5712): 434-436; 1979.

The expression of murine leukemia viruses in normal and athymic mutant (streaker) AKR/J mice was studied. Congenital thymic aplasia in AKR/J mice abolishes the occurrence of typical lymphomas and permits the development of tumors normally rare or absent in this strain, ie, reticulum cell sarcomas and granulocytic leukemias. Streaker mice produced both ecotropic and xenotropic AKR/J leukemia viruses, but not a recombinant virus, designated mink cell focus-forming virus (MCF), which has been isolated from the thymuses of late preleukemic and leukemic AKR/J mice. Beginning at age 5 mo, there was also a significant difference in the expression of xenotropic virus between the normal and streaker mice. Ecotropic and xenotropic viruses were found in several tissues from streaker mice with reticulum cell sarcomas, but no dual-tropic virus was isolated and none of the isolates induced morphological changes on mink lung cells. The data suggest that the thymus or a particular thymic cell population

is necessary for leukemogenesis and for the isolation of MCF virus in AKR mice. High virus titers may also be necessary for the isolation of recombinant viruses. (18 refs)

- 79-4570 Immunoprevention of Leukemia in AKR Mice by Type-specific Immune Gamma Globulin (IgG).** (Eng) Huebner, R. J. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD 20014); Price, P. J.; Gilden, R. V.; Toni, R.; Hill, R. W.; Fish, D. C. *Prog Biochem Pharmacol* 14: 151-156; 1978.

The use of type-specific IgG to prevent leukemia in AKR mice, 80%-90% of which normally die of spontaneous lymphocytic leukemia by 12 mo of age, was studied. Newborn mice were given four injections of IgG containing specific antibodies for C-type AKR virus during the first 14 days of life. Suppression (by 4 logs over controls) of AKR virogene expression was observed up to 34 days of age, and partial suppression persisted beyond 200 days of age. By 365 days, 6/30 immunized mice had developed fatal leukemias compared with 20/24 controls. In another experiment, four IgG immunizations were given between 0 and 20 days of age, killed banded Gross leukemia virus (GLV) vaccine was then given three times at 14-day intervals, and a single injection of murine sarcoma virus (GLV) was given 10 days later. At 300 days of age, 30/50 controls had died of leukemia compared with only 1/24 immunized mice. The data provide definitive evidence establishing the genetically transmitted AKR virogene expressions as the endogenous phenotypic cause of leukemia in the AKR mouse. (12 refs)

- 79-4571 Studies on the Target Cell for the Friend Virus (FV-P Strain) Using the CFU-E Technique.** (Eng) Opitz, U. (Abteilung für Klinische Physiologie der Universität Ulm, Oberer Eselsberg, D-7900 Ulm/Donau, W. Germany); Seidel, H. J. *Blut* 37(4): 183-192; 1978.

The target cell for polycythemia-inducing Friend virus (FV-P) was studied using female NMRI and DBA/2 mice. Colony-forming unit erythropoietic (CFU-E) cultures were set up with or without erythropoietin (EP). After FV-P virus infection, colonies appeared in the absence of EP. These EP-independent colonies were taken to be markers of leukemia. Their number was correlated with the compartment size of pluripotent, granuloid-committed, and erythroid stem cells at the time of infection. The development of Friend leukemia did not require the actual presence of CFU-E, as seen in experiments with actinomycin D, and it was not correlated with the number of pluripotent or granuloid stem cells, as seen after busulfan treatment. It was, however, dependent on the erythropoietic state of the animal, as seen in plethoric mice (hypertransfused with RBC) and in mice that were bled. It is concluded that the

target cell for FV-P is located within the EP-responsive cell compartment, between early and late (CFU-E) erythroid precursor cells. (23 refs)

- 79-4572 Elevation of Lysosomal DNase Activity in Mouse Liver During Friend Virus Leukemogenesis.** (Rus) Drozhennikov, V. A. (Res. Lab. Experimental Immunobiology, Moscow, USSR); Perevezentseva, O. S.; Orlova, E. B. *Biokhimiia* 44(4): 649-657; 1979.

DNase I and DNase II activities in the livers of mice with Friend leukemia (FL) were evaluated. DBA/2 and BALB/c mice were inoculated ip with a 10% homogenate of the spleen of FL-bearing mice. At different times postinoculation, the mice were sacrificed, and their livers were perfused in situ and homogenized. Then, liver specimens were centrifuged and the activity of enzymes in various fractions was determined. Mitochondrial DNase I activity in the liver of leukemia-bearing mice did not differ from that in controls. There was an increase in the activity of lysosomal DNase II that started on day 12 postinoculation and reached a max on days 16 and 17. Electron microscopic examination of liver specimens showed that there was a significant increase in the number of primary lysosomes in Kupffer's and endothelial cells. (26 refs)

- 79-4573 Two-dimensional Analysis of Murine Leukemia Virus gag-Gene Polyproteins.** (Eng) Ledbetter, J. A. (Stanford Univ. Sch. Medicine, Dept. Genetics, Stanford, CA 94305). *Virology* 95(1): 85-98; 1979.

The processing of *gag* translational products in a Gross murine leukemia virus-induced leukemia [E (male) G2] was studied by two-dimensional gel electrophoresis, which combines separation based on charge in the first dimension and separation based on size in the second dimension. In most experiments, the *gag* species were compared with the *env* species; *gag* species were precipitated from labeled cells or virus with antisera to the virion *gag* proteins p30 or p10, whereas *env* species were precipitated from labeled cells or virus with anti-gp70 serum. Three viral proteins were detected on the surface of E (male) G2 cells labeled with ¹²⁵I-lactoperoxidase; these included gp70 and two glycosylated *gag* gene species (gpP95 *gag* and gpP85 *gag*). Neuraminidase treatment of ¹²⁵I-lactoperoxidase-labeled cells did not affect the antigenicity of gp70, gp95 *gag*, or gpP85 *gag*. However, the treatment caused gp70, gp95 *gag*, and gpP85 *gag* to migrate as more basic species, indicating that all three glycoproteins contain terminal sialic acid. The cytoplasmic *gag*-gene products were studied by ³⁵S-methionine labeling of E (male) G2 cells; seven relatively stable *gag* species were identified. In general, the *gag* intermediates exhibited multiple, specific modifications that

resulted in complex yet reproducible patterns in the two-dimensional gel system. The core polyproteins Pr75gag and Pr65gag formed rapidly after pulse-labelings, with Pr65gag being processed into Pr55gag through cleavage of p10. The smaller gag species (Pr45gag and p30) also appeared to result from processing of Pr65gag. In contrast, Pr75gag was directly processed to form gpP95gag. A protein of approx 58,000 daltons, designated P58gag, qualified as a gag species since it was specifically precipitated by anti-p30 serum. However, P58gag did not appear to be a precursor of p30 since it was long-lived in the cytoplasm. Both the cytoplasm and the virion contained several unique forms of p30. Comparisons of the gag species from several AKR leukemias indicated that similar, but not identical, gag gene products are present in the various murine leukemias. (31 refs)

- 79-4574 DNA Polymerase from Gross Murine Leukemia Virus.** (Eng) Bartnikowa, W. (Dept. Analytics and Clinical Biochemistry, Gliwice Branch Inst. Oncology, P.O. Box 201, Gliwice, Poland). *Jpn J Exp Med* 49(1): 9-11; 1979.

The template activity of the DNA polymerase from Gross murine leukemia virus was studied. Endogenous and exogenous heterogenous RNA's were not utilized by the enzyme in spite of the presence of exogenous primers. Moderate enzymatic activity was observed only in the presence of DNA from *Escherichia coli* and activated DNA. With respect to synthetic template-primer complexes, enzyme activity was high against poly(A)-oligo(dT)₁₀, poly(dA)-oligo(dT)₁₀, and poly(dC)-poly(dI). The activity was strongest against the poly(A)-oligo(dT)₂₀ template in which the ratio poly(A)-oligo(dT)₂₀ was 3:1. The enzymatic reaction was dependent on pH in the presence of poly(A)-oligo(dT)₁₀, but not in the presence of poly(C)-oligo(dG)₁₂₋₁₈. (14 refs)

- 79-4575 Assembly of a Temperature-sensitive Mutant of Rauscher Murine Leukemia Virus at the Cell Surface Induced by Low Temperature and by Ligands.** (Eng) Demsey, A. (NCI, Bethesda, MD 20014); Kawka, D.; Galuska, S.; Stackpole, C. W. *Virology* 95(1): 235-240; 1979.

Virus assembly was studied in NIH/3T3 mouse fibroblasts infected with a Rauscher murine leukemia virus temperature-sensitive mutant (*ts25*) defective in assembly of budding particles at 39 C. Virus formation was observed on the surface of the infected cells when the temperature was shifted rapidly to 0 C. Virus buds were not assembled within the first 10 min at 0 C, but they gradually increased in number and degree of development over a 2-hr period. Release of infectious virus could not be demonstrated at 0 C, which suggested that it might be an energy-dependent

process. Significant budding activity was also induced at the nonpermissive temperature by incubating cells with 0.25% glutaraldehyde or with antiserum to the major virus envelope glycoprotein, gp70. Anti-gp70 serum may induce budding by promoting aggregation of gp70-containing molecular assemblies and consequently, association of core components in some transmembrane fashion. Induction of virus assembly with glutaraldehyde might occur as a result of nonspecific cross-linking of membrane proteins. These results suggest that procedures commonly used to minimize ligand-induced redistribution of cell surface molecules, ie, labeling at low temperatures or after mild aldehyde fixation, may not immobilize certain membrane-associated molecules. (31 refs)

- 79-4576 Observations on Antigenic Relatedness between Viruses of the Herpes Simplex 'Neutroseron'.** (Eng) Killington, R. A. (Dept. Microbiology, Sch. Medicine, Univ. Leeds, Leeds, England); Randall, R. E.; Yeo, J.; Honess, R. W.; Halliburton, I. W.; Watson, D. H. *IARC Sci Publ* 24(1): 185-194; 1978.

The antigenic relatedness of three viruses of the herpes simplex type 1 neutroseron -- herpes simplex virus types 1 (HSV-1) and 2 (HSV-2) and bovine mammillitis virus (BMV) -- was examined by immune precipitation and virus neutralization tests. Many virus-specific infected-cell polypeptides were shown to possess antigenic sites shared by both HSV-1 and HSV-2. Cross-neutralization between the viruses was mediated through antibodies to at least two antigenic sites, one shared by HSV-1, HSV-2, and BMV and one shared by HSV-1 and HSV-2 but not BMV. (17 refs)

- 79-4577 BLV-LB Virus-specific Sequences in Cattle with Spontaneous and Experimentally Induced Leukemia.** (Eng) Kukaine, R. A. (August Kirchenstein Inst. Microbiology, Acad. Sciences Latvian SSR, Kleisti 226067 Riga, USSR); Nagayeva, L. I.; Dyachenko, A. G.; Chapenko, S. V.; Ilyinskaya, T. N.; Bratsslavskaya, O. I.; Koudeleva, G. V.; Tsibinogin, V. V.; Yakovleva, L. A.; Lapin, B. A. *Arch Geschwulstforsch* 49(1): 1-5; 1979.

Sequences complementary to bovine leukemia virus (BLV) RNA in cellular genomes of animals with spontaneous and experimentally induced leukemia as well as in cell genomes of normal cattle were compared. Poly(A)-containing RNA's were extracted directly from the culture fluid of BLV-producing continuous and short-term lymphocyte cultures. The poly(A)-RNA's were used as a template to synthesize complementary [³H]DNA (BLV cDNA). BLV cDNA possessed a high degree of homology to poly(A)-RNA isolated directly from the blood plasma of a leukemic animal. In contrast to leukemic cattle, there were no BLV-

related sequences in the WBC of normal animals. The leukemic cells of cattle with spontaneous or experimentally induced leukemia contained BLV sequences in their genomes. (14 refs)

- 79-4578** **Oncogenic Transformation of Non-Permissive Murine Cells by Viable Equine Herpesvirus Type 1 (EHV-1) and EHV-1 DNA.** (Eng) Allen, G. (Univ. Mississippi Medical Center, Jackson, MS); O'Callaghan, D.; Randall, C. *IARC Sci Publ* 24(I): 509-516; 1978.

The oncogenic transformation of nonpermissive murine (BALB/c MEF) cells by viable equine herpesvirus type 1 (EHV-1) and EHV-1 DNA was studied. Primary cultures infected with as much as 100 plaque-forming units per cell of EHV-1 did not exhibit cytopathology or synthesize detectable EHV-1-specific RNA, DNA, or infectious virus. Addition of 1-2 μ g of nonfragmented EHV-1 DNA as a co-precipitate with calcium phosphate to such cultures resulted in the appearance of foci of piled-up, morphologically altered cells after 4-6 wk. Cell lines established from such foci exhibited a greatly increased growth rate, unlimited growth potential, aneuploid karyotype, and growth in soft agar. Inoculation of 10^6 transformed cells into newborn syngeneic mice resulted in the formation of serially transplantable tumors (undifferentiated fibrosarcomas) at an incidence of 100% within 8 wk. Infectious virus could not be rescued from the EHV-1 transformed or tumor-derived cell lines by growth in the presence of 5-iodo-2'-deoxyuridine, by cocultivation with permissive cells with transformed or tumor cell DNA. However, EHV-1 specific membrane antigens were detected in the transformed cells by immunofluorescence with hyperimmune anti-EHV-1 mouse serum, and the presence of a fragment of EHV-1 genome was demonstrated in both the transformed and tumor cells. These results indicate that cells nonpermissive for replication of EHV-1 remain susceptible to neoplastic transformation by the EHV-1 genome. (6 refs)

- 79-4579** **Reverse Transcription of the RNA of Eukaryotes and Viruses. Conditions for Obtaining the Full-sized Product.** (Rus) Kavsan, V. M. (Inst. Molecular Biology and Genetics, Kiev, USSR). *Mol Biol (Mosk)* 13(2): 266-280; 1979.

Current data pertaining to the synthesis of DNA on a RNA template are reviewed. Recent findings indicate that under certain conditions, the RNA-dependent DNA polymerases can synthesize complementary DNA molecules consisting of 5,000 nucleotides. (114 refs)

- 79-4580** **Episomal Viral DNA in Herpesvirus Saimiri-transformed Lymphoid Cell Lines.** (Eng)

Werner, F. J. (Institut für klinische Virologie, Erlangen, W. Germany); Bornkamm, G. W.; Fleckenstein, B.; Mulder, C. *IARC Sci Publ* 24(I): 125-130; 1978.

Structural analyses of the episomal viral genomes from two herpesvirus saimiri (HVS)-transformed tumor cell lines (1670 and 70N2) showed that both types of episomes had a higher mol wt (131.5 and 119.7×10^6 daltons, respectively) than linear virion DNA (approx 100×10^6 daltons). The arrangement of unique (L) and repetitive DNA in line 1670 episomes was studied by partial denaturation mapping. Some of the L sequences present in linear virion DNA were found to be missing and some were found to be duplicated. The episomal L-DNA regions were correlated with the known physical gene maps of linear HVS DNA. (5 refs)

- 79-4581** **Herpesvirus Papio: State of Viral DNA in Baboon Lymphoblastoid Cell Lines.** (Eng) Falk, L. (Dept. Microbiology, Rush-Presbyterian-St. Luke's Medical Center, 1753 W. Congress Parkway, Chicago, IL 60612); Lindahl, T.; Klein, G. *IARC Sci Publ* 24(I): 131-135; 1978.

The state of herpesvirus papio (HVP) in baboon lymphoblastoid cell lines (LCL) established from splenic or circulating lymphocytes of lymphomatous (*Papio hamadryas*) or clinically well (*P. anubis*) baboons, respectively, was investigated by cesium chloride density-gradient centrifugation and DNA-complementary RNA filter hybridization. Five HVP producer cultures and one nonproducer culture were studied. Nonintegrated and integrated HVP DNA was detected in all producer cultures, whereas only integrated HVP DNA could be demonstrated in the nonproducer LCL. Nonintegrated HVP DNA had a density of 1.715 - 1.716 g/cm³ in cesium chloride, a value corresponding to a guanine + cytosine content of 56%-57%. (6 refs)

- 79-4582** **Detection of Herpes Simplex RNA in Human Sensory Ganglia.** (Eng) Galloway, D. A. (Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104); Fenoglio, C.; Shevchuk, M.; McDougall, J. K. *Virology* 95(1): 265-268; 1979.

Examination of several paravertebral ganglia from seven autopsy cases for the presence of herpes simplex virus (HSV) RNA by in situ cytological hybridization using a nick-translated ³H-labeled HSV type 2 DNA probe revealed HSV RNA in all ganglia from two patients. The autoradiographic grains were localized in the neurons (ganglion cells), and only a fraction of the neurons in any one ganglion were synthesizing viral-specific RNA. This indicates that the HSV genome can be active in the latent state. (16 refs)

- 79-4583** **Diagnosis and Therapy of Viral Skin Diseases.** (Ger) Rohde, B. (Dermatologische Abt., Bundeswehrkrankenhaus, Leserstrasse 180, 2000 Hamburg 70, W. Germany). *Z Hautkr* 54(8): 326-327; 1978.

The diagnosis and therapy of skin diseases caused by veru-
ruca, herpes simplex, and herpes zoster viruses are review-
ed. The treatment of skin diseases induced by herpes
simplex and herpes zoster with informational RNA injec-
tions is promising. (2 refs)

- 79-4584** **Mapping of the Herpes Simplex Virus DNA Sequences in Three Herpes Simplex Virus Thymidine Kinase-transformed Cell Lines.** (Eng) Leiden, J. M. (Committee on Virology, Univ. Chicago, Chicago, IL); Frenkel, N.; Polacek, D.; Rapp, F. *IARC Sci Publ* 24(1): 473-488; 1978.

A novel filter hybridization approach was used in order to map the herpes simplex virus (HSV) DNA sequences which are present in three HSV thymidine kinase (TK)-transformed cell lines. The cell line 33A+, produced by infection of 3T3 TK- cells with UV-irradiated HSV-2 (333), was found to contain one contiguous stretch of viral DNA sequences which maps between 0.15 and 0.57 on the HSV-2 genome. The sequences mapping from 0.31 to 0.37 were present in 3- to 4-fold higher abundance than the rest of the viral DNA sequences in this cell line. Cell lines 5A and 8N were produced by transfection of mouse CL1D cells with sheared HSV-1 (1023) DNA. The 5A cell line was found to contain a contiguous set of viral DNA sequences mapping between 0.26 and 0.41 on the HSV-1 genome. The 8N cell line was found to contain three non-contiguous sets of viral DNA sequences, mapping between 0.09 and 0.41, 0.53 and 0.58, and 0.94 and 1.0 on the HSV-1 genome. These results seem to indicate that many different sets of viral DNA sequences can be incorporated into the cell during HSV-mediated biochemical transformation. (27 refs)

- 79-4585** **Enhanced Efficiency of Transfection of HSV DNA in HSV-1 DNA-transformed Hamster Cells.** (Eng) Colbere-Garapin, F. (Virology Dept., Institut Pasteur, Paris, France); Horodniceanu, F. *IARC Sci Publ* 24(1): 523-526; 1978.

The transfection efficiency, ie, the number of plaques formed per microgram of DNA, of the DNA's of herpes simplex virus (HSV) types 1 and 2 and herpesvirus eidolon (antigenically unrelated to HSV) was compared in HSV DNA-transformed cells (EH/A44) and in control hamster cells (EHT). The transfection efficiency of the HSV-1 and HSV-2 DNA's was significantly higher in EH/A44 cells than in EHT cells, which shows that an early step in HSV infection is involved in the partial resistance of HSV DNA-transformed cells to superinfection by various intact herpesviruses. (4 refs)

- 79-4586** **The Regulations of γ (Structural) Polypeptide Synthesis in Herpes Simplex Virus Types 1 and 2 Infected Cells.** (Eng) Wolf, H. (Marjorie B. Kovler Viral Oncology Labs., Univ. Chicago, Chicago, IL); Roizman, B. *IARC Sci Publ* 24(1): 327-336; 1978.

Factors regulating the transition from β to γ (structural) polypeptide synthesis in herpes simplex virus types 1 and 2 (HSV-1 and HSV-2)-infected cells were studied. γ Polypeptide synthesis in the presence of inhibitors of DNA synthesis was multiplicity-dependent. Some γ polypeptides were not detectable in cells infected with 1 plaque-forming unit (PFU) per cell, but they formed a significant fraction of viral polypeptides in cells infected with 25-500 PFU's/cell. The messenger RNA's (mRNA's) specifying these polypeptides had a relatively short half-life as measured by the relative rate of decay of polypeptide synthesis in infected cells following exposure to actinomycin D. The data suggest that the transition from β to γ polypeptide synthesis does not require the synthesis of viral DNA and that the rate of synthesis of viral structural polypeptides is linked to the size of the viral DNA pool as a consequence of a relatively short-lived mRNA. (11 refs)

- 79-4587** **Immunological Characterization of a Common Antigen Present in Herpes Simplex Virus, Bovine Mammillitis Virus and Herpesvirus Simiae (B Virus).** (Eng) Ludwig, H. (Inst. Virology, Justus-Liebig- Univ., Giessen, W. Germany); Pauli, G.; Norrild, B.; Vestergaard, B. F.; Daniel, M. D. *IARC Sci Publ* 24(1): 235-241; 1978.

An antigen present in herpes simplex virus (HSV) types 1 and 2, bovine mammillitis virus (BMV), and herpesvirus simiae (B virus) was characterized immunologically. Immunoelectrophoresis with various homologous and heterologous antisera revealed only one common antigen in all preparations. Crossed immunoelectrophoretic analyses gave similar results: strain-specific antisera reacted only with an HSV type 1 immunoprecipitate that corresponded to antigen 11 (Ag11) previously defined in the HSV system. Polyacrylamide gel electrophoresis indicated that the Ag11 complex differed in composition in the four viruses. However, it included a polypeptide with an apparent mol wt of 126,000 daltons in all preparations. The polypeptide was glycosylated, and it appeared that the common antigenic determinant(s) was carried by this glycoprotein. Evidence that the common antigenic sites were exposed on the surfaces of the viral particles was also obtained by cross-protection experiments. Mice immunized with BMV did not develop fetal encephalitis when inoculated intracerebrally with either type of HSV. (6 refs)

- 79-4588** **Definition of an Antigen Shared by Herpesviruses and Oncornaviruses.** (Eng) Ber-

nhard, M. I. (Sloan-Kettering Inst. Cancer Res., New York, NY); Old, L.J.; Takahashi, T.; Sarkar, N. H.; Lawrence, W. C. *IARC Sci Publ* 24(1): 215-224; 1978.

An antigen shared by herpesviruses and oncornaviruses was investigated. The component serologically identical to that present in oncornaviruses of avian, reptilian, and mammalian origin was precipitated by the following anti-herpesvirus nucleocapsid (nc) sera: equine herpesvirus type 1, herpes simplex virus types 1 and 2, Epstein-Barr virus, Marek's disease virus, cytomegalovirus, and Lucke herpesvirus from frog renal adenocarcinomas. The shared antigen was not detected in preparations of viruses from other classes. Nevertheless, given the data from immunodiffusion tests and immunoelectron microscopy, the possibility that the antigen is host-derived rather than virus coded cannot be excluded. (3 refs)

79-4589 Crossed Immunelectrophoretic Analysis and Viral Neutralizing Activity of Five Monospecific Antisera Against Five Different Herpes Simplex Virus Glycoproteins. (Eng) Vestergaard, B. F. (Dept. Clinical Virology, Inst. Medical Microbiology, Copenhagen, Denmark); Norrild, B. *IARC Sci Publ* 24(1): 225-234; 1978.

Five antisera monospecific against five different herpes simplex virus (HSV) glycoproteins were studied by crossed immunelectrophoretic analysis (CIE) and viral neutralizing assays. CIE of Triton X-100-solubilized HSV-infected cells revealed several major HSV glycoprotein antigens in the precipitating profile. Immunization of rabbits with the corresponding precipitates resulted in the production of monospecific antisera against the antigen parts of the precipitates. The immunologic importance of the five HSV glycoproteins with respect to their representation on the outer virion envelope as targets for neutralizing antibodies was evaluated by comparison of the immunoprecipitating activity and neutralizing potency of the corresponding monospecific antisera. (13 refs)

79-4590 The DNA of Serially Passaged Herpes Simplex Virus: Organization, Origin, and Homology to Viral RNA. (Eng) Locker, H. (Dept. Biology, Univ. Chicago, Chicago, IL); Frenkel, N. *IARC Sci Publ* 24(1): 75-85; 1978.

The structural and functional organization of the DNA extracted from serially passaged herpes simplex virus type 1 (HSV-1) was studied. High-density DNA prepared from this HSV-1 contained three major classes of modified viral DNA molecules. The altered DNA molecules were composed of multiple repetitions of sequences derived from the right-hand side of the S region of the parental plaque-purified viral DNA. The repeat units contained in the three

types of high-density DNA shared most of their DNA sequences but differed with respect to a small region derived from the unique sequences of the S component of HSV-1 DNA. Hybridization of the defective DNA to HSV-infected cell RNA showed that the high-density DNA contained sequences complementary to both early and late viral transcripts. (15 refs)

79-4591 Virus Transcript Mapping Studies in Cells Infected with Temperature-sensitive Mutants of Herpes Simplex Virus Type 1. (Eng) Watson, R. J. (Inst. Virology, Univ. Glasgow, Glasgow, Scotland); Clements, J. B. *IARC Sci Publ* 24(1): 313-326; 1978.

Nuclear and cytoplasmic transcripts, synthesized in cells infected with six DNA-negative temperature-sensitive (*ts*) mutants of herpes simplex virus (HSV)-1 (*ts*B, *ts*D, *ts*E, *ts*K, *ts*S and *ts*T) under nonpermissive conditions, were isolated and hybridized to unlabelled fragments of HSV-1 DNA, generated by restriction endonuclease digestion and immobilized onto nitrocellulose membranes. Those regions of the HSV-1 genome represented by stable transcripts in the mutant-infected cells were then mapped and compared with those regions transcribed in cells infected with the wild-type virus at early and late times post infection (before and after viral DNA replication) and in the presence of DNA- and protein-synthesis inhibitors. Viral transcription in *ts*D, *ts*T and *ts*K-infected cells is restricted, the patterns of hybridization being similar but not identical to that observed with immediate early RNA. Since these three mutants fall into two complementation groups, these experiments suggest that at least two viral products are required for the switch-on of early transcripts. In contrast, transcript mapping with the other early mutants (*ts*B, *ts*E and *ts*S) has shown a much less restricted transcriptional pattern, resembling that with early rather than late RNA. (14 refs)

79-4592 Evidence for a Protein(s) Bound to Herpes Simplex Virus DNA. (Eng) Hyman, R. W. (Dept. Microbiology, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033); Richards, J. C.; Kudler, L. *Biochem Biophys Res Commun* 88(2): 522-528; 1979.

The endonuclease *Xba* I cleavage sites on herpes simplex virus (HSV) DNA were studied before and after deproteinization. The *Xba* I cleavage pattern for highly purified nondeproteinized HSV DNA did not match the published patterns, whereas a match was obtained after the DNA had been subjected to specific deproteinization. The cleavage pattern of incompletely *Xba* I-digested, rigorously deproteinized HSV DNA did not match that of nondeproteinized HSV DNA. The percent of total HSV DNA that remained at the origin of the electrophoretic gel was between 2% and 3% for both deproteinized and rigorously deproteinized

DNA. However, the percent of total nondeproteinized DNA that remained at the origin of the gel varied among different gels. The reason for this variation is not known. The data suggest that there is a protein(s) bound to HSV DNA. (14 refs)

79-4593 Annealing of Alkali-resistant HSV DNA Strands and Isolation of S and L Components.

(Eng) Friedmann, A. (Dept. Genetics, Hebrew Univ. Jerusalem, Jerusalem, Israel); Broit, M.; Becker, Y. *IARC Sci Publ* 24(1): 137-148; 1978.

The isolation of intact L, S, and repeat sequences of herpes simplex virus type 1 (HSV-1) DNA is described. DNA isolated from highly purified virions of HSV-1 (HF-strain) was denatured by centrifugation in alkaline sucrose gradients, and DNA molecules corresponding to intact single-stranded (ss) virion DNA (50×10^6 daltons) were isolated and adjusted to neutral pH. The DNA was annealed under conditions permitting reassociation of intact ss molecules and studied by electron microscopy. Three classes of DNA molecules with double-stranded (ds) sequences were observed: (1) fully ds DNA molecules the size of the intact HSV DNA genome (52 μ m); (2) DNA hybrids with a region of partial double-strandedness ranging from 5 to 12 μ m, plus long single strands; and (3) DNA hybrids with a ds region of 32-40 μ m, plus short single strands. These results suggest that the alkali-resistant ss HSV DNA molecules are composed of several subclasses that permit annealing of either the total genome or the S or L components. The 5- μ m ds region probably constitutes the S component of HSV DNA, and sequences $>5 \mu$ m and $<12 \mu$ m represent annealing of the repeat sequences on either or both sides of the S component. The 32- to 40- μ m ds sequences may represent the L component. Treatment of the annealed, partially ds hybrid DNA molecules with S_1 endonuclease to remove the ss termini and centrifugation in neutral sucrose gradients yielded two distinct peaks. Centrifugation of fractions from the two peaks in cesium chloride density gradients showed that the small DNA component (possibly the S and the repeat sequences) had a higher buoyant density and the longer (possibly the L) DNA component had a lower density than the HSV DNA marker. Annealing of alkali-resistant viral DNA strands therefore provides a means of isolating the L, S and repeat sequence regions of HSV DNA. (11 refs)

79-4594 Regulation of Herpes Simplex Virus Type 1 DNA Synthesis: Temperature-Shift Studies with DNA-Negative Temperature-sensitive Mutants.

(Eng) Parris, D. S. (Div. Basic Sciences, Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA); Schaffer, P. A.; Courtney, R. J. *IARC Sci Publ* 24(1): 299-311; 1978.

The regulation of expression of viral genes involved in the

synthesis of herpes simplex virus HSV type 1 DNA was studied using three DNA- temperature-sensitive (*ts*) mutants (B, C, and D). These mutants were examined for their ability to synthesize viral DNA and polypeptides following temperature shift-down in the presence or absence of the transcription inhibitor actinomycin D. The results demonstrated that the B gene product is required transiently early in infection and apparently controls a transcriptional step required for HSV DNA synthesis. The C gene product is required continuously during infection and also controls a transcriptional step needed for viral DNA synthesis. In contrast, the product of the D gene does not directly control a transcriptional step, is required continuously, appears to be directly involved in HSV DNA synthesis, and is probably the gene for viral DNA polymerase. The results further show that recovery of viral DNA and polypeptide synthesis following temperature shift-down in the absence of inhibitor was greater for the D mutant than for the mutants blocked in viral DNA synthesis at the level of transcription. (8 refs)

79-4595 Herpesvirus Production by Ultraviolet-irradiated Human Skin Cells: A Marker of Repair.

(Eng) Coppey, J. (Fondation Curie-Institut du Radium, Paris, France); Nocentini, S.; Moreno, G. *IARC Sci Publ* 24(1): 407-414; 1978.

The herpes simplex virus (HSV) yield corresponding to the first cycle (18 hr) was measured in various human skin cells infected at various times after UV irradiation. At time 0, HSV production capacity was more resistant to UV irradiation in xeroderma pigmentosum (XP) heterozygous cultures than in normal cultures. The extent of recovery decreased with increased UV dose, and it was lower in XP heterozygotes than in normal cells. In contrast, in strains deficient in unscheduled DNA synthesis, this capacity decreased with increasing time intervals between UV exposure and infection. Recovery of HSV production capacity appears to be a very sensitive probe for detecting differences in the ability of UV-irradiated human skin cell lines to repair damaged DNA. (14 refs)

79-4596 The Thymidine Kinase Gene of Herpes Simplex Virus Type 1: Cell-Free Protein Synthesis and Substrate Specificity Studies.

(Eng) Cremer, K. J. (Dept. Therapeutic Radiology, Yale Univ. Medical Sch., New Haven, CT); Summers, W. P.; Summers, W. C. *IARC Sci Publ* 24(1): 337-345; 1978.

An *in vitro* messenger RNA (mRNA)-dependent protein synthesis system was used to study some independently isolated thymidine kinase-negative (TK⁻) mutants of herpes simplex virus type 1 (HSV-1). Quantitative studies of TK⁻ to TK⁺ mutation frequencies suggested that there was no significant difference in the number of mutants obtained

with or without exogenous mutagen. However, all stocks may have been exposed to 5-bromodeoxyuridine mutagenesis. Of the 30 mutants, 12 appeared to be missense mutants, whereas the rest appeared to be deletion, chain termination, internal start, or some sort of protein or mRNA processing mutants. An in vitro system programmed with polysomes from HSV-infected cells faithfully reproduced the in vivo phenotype of the mutants, but the possibility of posttranslational modification could not be excluded. The mutants produced the same polypeptide products in vitro as those seen in vivo. At least four of the mutants appeared to be chain termination rather than deletion mutants. However, these mutants may have had an altered amino acid sequence creating a new site for protease action, or the mutation may have had its effect at the level of posttranscription RNA processing. (13 refs)

79-4597 Analysis of Herpes Simplex Virus Low Molecular-Weight Native Proteins by Polyacrylamide Gel Electrophoresis. (Eng) O'Hara, M. K. (Dept. Microbiology, Univ. Tennessee, Knoxville, TN); Courtney, R. J. *IARC Sci Publ* 24(1): 195-201; 1978.

The potential application of gel electrophoresis for the isolation and further characterization of herpes simplex virus (HSV)-2 native proteins was demonstrated. At least eight major HSV-2 specific proteins with characteristic isoelectric points could be reproducibly resolved by gel isoelectric focusing. Their isoelectric points ranged from 5.0 to 7.5, and analysis by two-dimensional electrophoresis on sodium dodecyl sulfate gels indicated that their mol wt ranged from 11,000 to 49,000. (5 refs)

79-4598 DNA-mediated Transfer of Herpes Simplex Virus TK Gene to Human TK⁻ Cells: Properties of the Transformed Lines. (Eng) Bacchetti, S. (Dept. Pathology, McMaster Univ., Hamilton, Ontario, Canada); Graham, F. L. *IARC Sci Publ* 24(1): 495-499; 1978.

Human thymidine kinase-negative (TK⁻) cells carrying the herpes simplex virus type 2 TK gene express a TK activity of viral origin and maintain the TK⁺ phenotype when grown in hypoxanthine/aminopterin/thymidine (HAT) medium. Under nonselective or counterselective conditions, however, reversion to a TK⁻ phenotype occurs with a significant frequency characteristic of each transformed line. The TK⁻ phenotype appears to be stable, since no instances of TK⁻ to TK⁺ reversion have been observed. (3 refs)

79-4599 Incorporation of the Herpes Simplex Virus Thymidine-Kinase Gene into a Mammalian Cell Line Using Fragments of the Virus Genome. (Eng)

Darby, G. (Dept. Pathology, Univ. Cambridge, Cambridge, England); Minson, A. C.; Wildy, P. *IARC Sci Publ* 24(1): 489-493; 1978.

The interaction of viral DNA fragments with eukaryotic chromosomes was studied using LTK-cells transformed with sheared herpes simplex virus type 2 (HSV-2) DNA. A number of clonally unrelated lines were established which contained significant proportions of thymidine kinase (TK)-negative cells. The possession of the TK gene appeared to impart a selective advantage to the cells, even in nonselective medium. One clone, line D2₁, produced almost wild-type yields of the temperature-sensitive mutants N102 and N103 at the restrictive temperature. Five clonally unrelated lines were established after infection of LTK⁻ cells with DNA from D2₁ cells. All five clones possessed HSV-specific TK. Virus DNA was only 10-fold more efficient as a transforming agent than D2₁ DNA. Two alternative explanations for the high frequency of transformation with D2₁ DNA are either that the transforming DNA is a fragment of virus DNA flanked by host sequences, or that the virus DNA is carried in a circular form in transformed cells and that this is a far more efficient transforming element than the original linear DNA fragments. The only reasonable hypothesis for this observation appears to be that the viral DNA carried in D2₁ is modified by the cell so that it receives a more favorable reception than normal virus DNA fragments when it entered an LTK⁻ cell. (7 refs)

79-4600 Sites of Integration of Herpes Simplex Virus Type-2 Thymidine Kinase Gene in Human Chromosomes. (Eng) Kit, S. (Div. Biochemical Virology, Baylor Coll. Medicine, Houston, TX 77030); Teitz, Y.; Hazen, M.; Qavi, H. *Int J Cancer* 23(6): 846-853; 1979.

The sites of integration of the herpes simplex type 2 (HSV-2) thymidine kinase (TK) gene in biochemically transformed human [HeLa(BU25)/HSV-2-6 Cl 4] cells were investigated by isozyme analyses. Extracts were prepared from the HeLa(BU25)/HSV-2-6 Cl 4 cells and from human-mouse somatic cell hybrids obtained by fusing the biochemically transformed human cells with TK-deficient mouse [LM(TK⁻)] cells, and they were assayed for 26 isozymes representing markers for 20 human chromosomes (CS's). The isozyme analyses were generally consistent with previous karyotype studies, which revealed that the HSV-2 TK gene was associated with an iso-CS, designated M13, formed from the short arm of the human X CS in human-mouse hybrid lines HL/2 to HL/7, but with human CS 17 containing a translocation on the short arm in hybrid line HL/1 and its TK-positive subclones. The isozyme analyses also indicated that the translocation on the short arm of human CS 17 in hybrid line HL/1 was probably derived from human CS 21. Hybrid line HL/1 and its TK-positive subclones expressed a human-mouse heteropolymeric form of superoxide dismutase, a marker for human CS 21, but

bromodeoxyuridine-resistant, TK-negative subclones of HL/1, and hybrid lines HL/3 to HL/6, which did not contain the modified CS 17, failed to express the human-mouse heteropolymeric form of superoxide dismutase. The human galactokinase isozyme, which is coded by a gene mapping close to the cytosol TK gene on human CS 17, was detected in extracts of TK-positive HeLa S3, TK-deficient HeLa/(BU25), and biochemically transformed HeLa-(BU25)/HSV-2-6 Cl 4 cells, but not in extracts prepared from hybrid line HL/1 and its subclones. These results suggest that the gene for human galactokinase was deleted or inactivated in HL/1 and its subclones, perhaps as a result of the translocation to CS 17. (29 refs)

- 79-4601 Identification of Sequences Coding for Thymidine Kinase in Herpes Simplex Virus and Transformed Cell DNA. (Eng) Silverstein, S. (Dept. Microbiology, Columbia Univ., New York, NY); Wigler, M.; Pellicer, A.; Axel, R. *IARC Sci Publ* 24(I): 501-508; 1978.

To investigate the mechanisms that control the expression of herpes simplex virus (HSV) genes in transformed cells, a 3.4 kilobase (Kb) fragment generated by *Bam*H1 restriction endonuclease and containing the gene coding for thymidine kinase (TK) was isolated. The specific fragment containing the TK gene was identified by transfecting LTK⁻ cells with restriction endonuclease-derived fragments and selecting for the TK⁺ phenotype by growth in medium containing hypoxanthine, aminopterin, and thymidine (HAT). Cleavage with *Hpa*I or *Kpn*I yielded products which when digested with *Bam* yielded the 3.4 Kb fragment containing the gene coding for TK. Unlike the parental LTK⁻ cell line, transformants infected with this fragment cloned with equal efficiency in HAT or non-selective medium. A low but reproducible number of cells lost the ability to express TK and they could not be recloned in HAT. In a cell line transformed using the isolated *Bam* 3.4 Kb doublet there was only one copy of the TK gene per diploid quantity of transformed cell DNA. (10 refs)

- 79-4602 Regulation of the Herpes Simplex Virus Gene for Thymidine Kinase in Clonal Derivatives of Transformed Mouse L-Cells. (Eng) Buttyan, R. (Dept. Microbiology, Univ. Chicago, Chicago, IL); Spear, P. G. *IARC Sci Publ* 24(I): 517-522; 1978.

To identify the factors that regulate viral thymidine kinase (TK) synthesis in clonal derivatives of herpes simplex virus (HSV)-transformed mouse L cells, several phenotypically TK⁻ and TK⁺ clones were isolated from an HSV type 1 (HSV-1) transformed cell line. The ability of the derivatives to form colonies in selective media was consistent with the levels of TK activity detectable in cell extracts. The viral TK gene was retained in the phenotypically TK⁻ clones and the

susceptibility of this gene to regulation by products of superinfecting HSV-1 was unaltered. The clonal variants differed from one another not only in the levels of viral TK synthesized (in the absence of superinfection), but also in their ability to support HSV replication. When productively infected with HSV-1 (TK⁺), cells with high levels of viral TK synthesis produced significantly higher virus yields than did transformed or nontransformed TK-cells. This correlation between HSV TK synthesis and enhanced expression of an input viral genome suggests that the same factor or factors may be responsible for both phenomena. (5 refs)

- 79-4603 Repair of Single-stranded Breaks in Host DNA When Rabbit Kidney Cells Are Infected with Herpes Simplex Virus Type 2. (Eng) Kelman, A. D. (Boston Univ. Sch. Medicine, Boston, MA); Gundberg, C. M.; Sinex, F. M. *IARC Sci Publ* 24(I): 415-421; 1978.

Repair of single-stranded breaks in host DNA was studied in rabbit kidney (RK) cells infected with herpes simplex virus type 2 (HSV-2). ³H-thymidine was incorporated into the DNA of HSV-2-infected RK cells to a greater extent than into the DNA of mock-infected cells. Incorporation occurred maximally up to 6 hr postinfection (PI). At 6-8 hr PI, there was an onset of viral DNA synthesis, and unscheduled DNA synthesis was diminished. Density-shift studies demonstrated that increases in nucleotide incorporation after HSV-2 infection resulted from repair synthesis. Repair synthesis in RK cells during productive infection may be related to the initiation of latency and transformation by herpesviruses. Estradiol in the culture medium decreased repair synthesis by 11% at a concentration of 10⁻⁶ M and completely inhibited it at 10⁻⁵ M. Semiconservative replication was simulated by 6% with 10⁻⁶ M estradiol and by 20% with 10⁻⁵ M. Thus, in this system, 10⁻⁵ M estradiol facilitates error-prone postreplication repair. In women, infection with HSV-2 may produce damage to cervical DNA. Hormone action may inhibit the cellular repair processes and stimulate DNA synthesis with error-prone postreplicative repair. The resulting mutations might promote HSV-2 transformation and accelerate the progression from normal tissue to dysplasia to cancer in situ to invasive cancer; the first step in this progression would be reversible. (7 refs)

- 79-4604 Pronephric Tumour Cell Lines from Herpesvirus-transformed Cells. (Eng) Tweedell, K. S. (Dept. Biology, Univ. Notre Dame, Notre Dame, IN). *IARC Sci Publ* 24(II): 609-616; 1978.

Several pronephric tumor cell lines were established from larval and metamorphosing *Rana pipiens* after the in vivo transformation of stage 17 (tailbud) embryos by a herpesvirus isolated from an adenocarcinoma of an adult frog. The pronephric tumor cell lines contained approx

85% polygonal epithelial cells and some fibroblast-like cells. The epithelial cells were heteroploid with a major chromosome mode of 39 (range, 34-43). The cell types were aneuploid rather than polyploid. Two marker chromosomes, a minute and a translocation, were present in high incidence. The cells showed a lack of contact inhibition, and they could be grown in rotary and overlay cultures. Eye implants of pronephric tumor nodules from rotary cultures continued to proliferate in yearling frogs of the same species, often invading the lens or iris. Lucke herpesvirus antigens were present in the tumor cell lines. (7 refs)

79-4605 Alterations in Biological Properties of Different Lines of Cytomegalovirus-transformed Human Embryo Lung Cells Following In Vitro Cultivation. (Eng) Geder, L. (Dept. Microbiology and Specialized Cancer Res. Center, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA); Laychock, A. M.; Gorodecki, J.; Rapp, F. *IARC Sci Publ* 24(II): 591-601; 1978.

Diverse alterations in biological properties of cytomegalovirus (CMV)-transformed cell lines were observed during prolonged in vitro cultivation. In the CMV-Mj-HEL-2 parent line there was a gradual decrease in the number of cells expressing CMV-related antigens; at the same time, an increase in oncogenicity was observed. One tumor line, designated CMV-Mj-HEL-2,T-1, retained the original ratio of cells expressing CMV-related antigens for over 100 in vitro passages. The cells lost their original moderate oncogenicity during this period. A later increase in the ratio of cells without CMV antigenic markers was accompanied by the return of moderate tumorigenicity and karyotypic changes. Both cell lines were studied to determine sensitivity to superinfection with herpesviruses, induction of immune response in nude mice, and release of infectious virus. (12 refs)

79-4606 Neutralization Kinetic Studies with Genital Cytomegalovirus Isolates, an Antigenically Variable Group. (Eng) Weller, T. H. (Dept. Tropical Public Health, Harvard Sch. Public Health, Boston, MA); Waner, J. L.; Hopkins, D. R.; Allred, E. N. *IARC Sci Publ* 24(I): 177-184; 1978.

Neutralization kinetics experiments were performed on genital and nongenital strains of human cytomegalovirus (CMV) to determine whether there are CMV groupings analogous to those of herpes simplex virus types 1 and 2. Antisera were prepared in rabbits against four low-passage genital isolates and against two established strains of human CMV. With these sera plus seven strains of virus, 42 virus-antiserum combinations were examined and NK values (a proportional rate at which a serum neutralized a heterologous virus) were derived. No evidence accrued in-

dicating that the genital isolates constituted an antigenically distinct group. The findings support the view that the human CMV's are antigenically heterogeneous, with different strains reflecting an antigenic mosaic, the elements of which are present in varying amounts. (20 refs)

79-4607 Analysis of Viral Gene Fractions by Means of Early Temperature-sensitive Mutants of Human Cytomegalovirus. (Eng) Ihara, S. (Dept. Molecular Biology, Tokai Univ. Sch. Medicine, Tokai, Japan); Hirai, K.; Watanabe, Y. *IARC Sci Publ* 24(I): 365-371; 1978.

Early temperature-sensitive (ts) mutants of human cytomegalovirus (CMV) were used to analyze viral gene functions. Five of 15 mutants failed to produce detectable amounts of viral DNA in infected human embryo liver cells at the nonpermissive temperature (39 C); these mutants were classified as DNA⁻ and the other 10 were classified as DNA⁺. DNA⁻ mutant ts 256 had a defect in the induction of ammonium sulfate-stimulated DNA polymerase, suggesting that this polymerase is a virus-mediated entity and responsible for the replication of CMV DNA. All DNA⁻ mutants were able to induce virus-specific antigens in infected-cell nuclei at 34 or 39 C. None except ts 256 induced noticeable morphological changes in infected-cell nuclei or any structure related to virions at 39 C; ts 256 induced empty capsids in a large proportion of infected cells. Cellular DNA synthesis was stimulated by all DNA⁻ mutants at 34 and 39 C. The DNA⁻ mutants were divided into two complementation groups: Group A included the DNA polymerase-positive mutants, and Group B included the DNA polymerase-negative member. All the DNA⁻ mutants complemented well with the DNA⁺ mutants tested. (14 refs)

79-4608 Cell DNA Induction by Human Cytomegalovirus. (Eng) St. Jeor, S. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA); Hernandez, L.; Tocci, M. *IARC Sci Publ* 24(I): 373-379; 1978.

Human embryonic lung cell (HEL) DNA induction by human cytomegalovirus (CMV) was studied. The results indicated a temporal relationship between cell and virus DNA synthesis. In cells pretreated with 5-iodo-2'-deoxyuridine, only a fraction of the cell genome was induced by the virus to replicate. Cell DNA induced to replicate by CMV contained primarily unique rather than repetitive DNA sequences. In contrast, 15% of uninfected cell DNA appeared to reassociate at a *Cot* of 10, with the majority of the DNA reassociating at a slower rate. The unique cell DNA sequences appeared to be the primary species in the virus-induced system. (6 refs)

- 79-4609 Induction of Cellular DNA Synthesis by Defective Human Cytomegalovirus.** (Eng) Demarchi, J. M. (Dept. Microbiology, Vanderbilt Univ. Sch. Medicine, Nashville, TN); Kaplan, A. S. *IARC Sci Publ* 24(II): 603-601; 1978.

The role of human cytomegalovirus (CMV) in the stimulation of cellular DNA in permissive cultures of human embryonic lung (HEL) cells was studied. Infection of serum-starved cells with CMV virions exposed to UV rays for 30 sec stimulated cellular DNA synthesis ninefold. Irradiation of the virions for 1-5 min reduced their ability to stimulate DNA synthesis. However, cultures infected with virus irradiated for 5 min contained 3.5-fold more cells synthesizing cellular DNA than uninfected cultures. Three different stocks derived from individual plaques (which presumably contained few defective particles) and passaged only twice at low multiplicity were poor inducers of cellular DNA synthesis. Stocks of virions obtained after serial undiluted passage and enriched for defective particles were highly effective in stimulating cellular DNA synthesis. Thus, defective CMV particles play a major role in stimulating cellular DNA synthesis in permissive cultures. (5 refs)

- 79-4610 Early Virus-specific Proteins Synthesized in Human Cytomegalovirus Infected Cells.** (Eng) Stinski, M. F. (Dept. Microbiology, Univ. Iowa Coll. Medicine, Iowa City, IA). *IARC Sci Publ* 24(I): 353-363; 1978.

The synthesis of 15 early infected cell-specific polypeptides (ICSP) by human fibroblast cells infected with the Towne strain of human cytomegalovirus (CMV) is described. Twenty-two late ICSP, synthesized after viral DNA synthesis, were also identified. The apparent mol wts of all but four of the early ICSP differ from those of late ICSP. Late, but not early, ICSP were inhibited by treatment with phosphonoacetic acid. Early CMV ICSP were shown to represent the translation products of messenger RNA (mRNA) that was transcribed prior to or in the absence of viral DNA synthesis. Most early CMV ICSP synthesized in permissive cells were also synthesized in nonpermissive cells, suggesting that the early peptides represent viral genome products rather than host-induced proteins. The synthesis of early CMV viral mRNA and proteins was completed in the infected cell within approx 14 hr of infection. DNA synthesis was not initiated until 20 hr postinfection. (11 refs)

- 79-4611 Characterization of Human Cytomegalovirus DNA: Infectivity and Molecular Weight.** (Eng) Geelen, J. L. (Laboratorium voor de Gezondheidsleer, Univ. Amsterdam, Amsterdam, Netherlands); Walig, C.; Wertheim, P.; Van Der Noordaa, J. *IARC Sci Publ* 24(I): 97-103; 1978.

Data on the infectivity and mol wt of human cytomegalovirus (CMV) DNA are presented. The viral DNA was isolated from purified virions and further purified by sucrose density gradient centrifugation. The viral DNA molecules were studied by electron microscopy and found to be linear and to have a length of 76.22 μ m, corresponding to a mol wt 147.13×10^6 . The DNA was infectious when tested in human embryonic lung cells using the diethylaminoethyl-dextran and calcium phosphate techniques. Its density in cesium chloride gradients was 1.717 g/cm³. (12 refs)

- 79-4612 Structural Organization of Human Cytomegalovirus DNA.** (Eng) Kilpatrick, B. A. (Dept. Bacteriology and Immunology, Univ. North Carolina Sch. Medicine, Chapel Hill, NC); Huang, E. S. *IARC Sci Publ* 24(I): 105-112; 1978.

The general arrangement of human cytomegalovirus (HCMV) DNA sequences was studied. HCMV DNA preparations in which purity was characterized by sedimentation, buoyant density, and restriction enzyme cleavage contain two mol wt size classes of HCMV DNA molecules ($150-155 \times 10^6$ and 100×10^6 daltons) in addition to some shear products. Partial denaturation mapping reveals that both size classes contain the same sequences and that molecules of $150-155 \times 10^6$ daltons contain the apparent full complement of sequences which are variously contained in the smaller molecules. The complete genome seems to be divisible into two regions of about 78%-82% and 18%-22%, which undergo complete inversions with respect to one another. Enriched or purified preparations of molecules of $150-155 \times 10^6$ daltons generate cleavage products identical to those from total HCMV DNA preparations. (6 refs)

- 79-4613 Isolation of the Epstein-Barr Virus Nuclear Antigen from Chromatin Preparations.** (Eng) Pikler, G. M. (Developmental Therapeutics, M.D. Anderson Hosp. and Tumor Inst., Houston, TX); Pearson, G. R.; Spelsberg, T. C. *IARC Sci Publ* 24(I): 243-247; 1978.

Epstein-Barr virus (EBV) nuclear antigen (EBNA) isolated from the chromatin of EBV-positive NC37 and Raji lymphoblastoid cells was characterized. The antigenic activity of EBNA from both cell lines was not destroyed by a variety of denaturing conditions and solvents; thus, it appeared to be based on the primary structure of a protein. About 60%-70% of the initial antigenic activity was extracted after treatment with 0.5 M sodium chloride; this activity represented a weak-binding class of chromatin-EBNA interaction. The remaining activity, representing a high affinity class, could be removed only under conditions that stripped all of the protein from the DNA. Preparative isoelectric focusing demonstrated a pI of 4.6 for both EB-

NA and complement-fixing soluble antigen. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis of the proteins focusing at pH 4.6 revealed three major and three minor bands. (8 refs)

- 79-4614 A Novel Surface Antigen on Lymphoid Cells Transformed by Epstein-Barr Virus.** (Eng) Hinuma, Y. (Dept. Microbiology, Kumamoto Univ. Medical Sch., Kumamoto, Japan); Sakamoto, K. *IARC Sci Publ* 24(1): 249-251; 1978.

A new antigen (SA) that is distinct from known membrane antigens (MA's) was demonstrated on the surface of Epstein-Barr Virus (EBV)-carrying lymphoid cell lines. The antigen was detected by an indirect membrane immunofluorescence test with serum from rabbits immunized with Raji cells; the antiserum had been extensively absorbed with normal human blood and tonsil cells. The SA was not detected on normal human umbilical cord and adult peripheral blood lymphocytes or EBV-negative cell lines. The incidences of the SA and EBV-determined MA on certain EBV-carrying cell lines were not compatible. Antibody against SA or MA was differentially abolished by absorption with the SA-positive but MA-negative cell line or the MA-positive but SA-negative cell line, respectively. The results of cross-absorption tests of antisera against either Raji cells or P3HR-1 cells suggest that SA is not a single but a complex antigen. (2 refs)

- 79-4615 A Possible Function of Epstein-Barr Virus-determined Nuclear Antigen (EBNA): Stimulation of Chromatin Template Activity In Vitro.** (Eng) Kamata, T. (Dept. Molecular Biology, Tokai Univ. Sch. Medicine, Isehara, 259-11, Japan); Tanaka, S.; Aikawa, S.; Hinuma, Y.; Watanabe, Y. *Virology* 95(1): 222-226; 1979.

The involvement of the Epstein-Barr virus-determined nuclear antigen (EBNA) in the regulation of chromatin template activity for transcription was studied in vitro using EBNA partially purified from Raji cells. The EBNA was added to chromatin from quiescent human embryo lung cells (HEL), and template activity was determined by measuring the ethidium bromide (EB)-binding capacity of the chromatin or the transcription with exogenous RNA polymerase. A factor(s) responsible for observed increases in the dye-binding capacity of the HEL chromatin was detectable only in EBNA-positive cell nuclei. The factor(s) corresponded to EBNA in molecular size. The addition of EBNA to chromatin also resulted in a small but definite increase in template activity, whereas other Raji cell fractions had no significant effect. The stimulation of chromatin template activity was abolished by the addition of anti-EBNA-positive IgG, but not by the addition of negative IgG. Although the EBNA preparations showed con-

siderable impurity, the results suggest the involvement of EBNA in the regulation of chromatin template activity. (18 refs)

- 79-4616 Simultaneous Presence of EBNA-positive and Colony-forming Cells in Peripheral Blood of Patients with Infectious Mononucleosis.** (Eng) Katsuki, T. (Dept. Microbiology, Kumamoto Univ. Medical Sch., Kumamoto 860, Japan); Hinuma, Y.; Saito, T.; Yamamoto, J.; Hirashima, Y.; Sudoh, H.; Deguchi, M.; Motokawa, M. *Int J Cancer* 23(6): 746-750; 1979.

Studies were conducted to determine whether infectious mononucleosis (IM) patients whose peripheral blood contained Epstein-Barr virus (EBV)-associated nuclear antigen (EBNA)-positive colony-forming cells also contained EBNA-positive cells that could be detected by a cell smear procedure. EBNA-positive lymphoblast cells were detected in 0.1%-0.9% of the T-cell-depleted lymphocytes obtained from peripheral blood samples of five IM patients. The same blood specimens from 4/5 patients contained cells that formed EBNA-positive colonies in soft agar containing EBV antibodies. The ratio of the colony formers to EBNA-positive cells was higher in blood samples taken early in the disease than in those obtained in later stages of the disease. The results strongly suggest that EBV-transformed cells are present in the peripheral circulation of IM patients and that such cells can directly give rise to immortalized cell lines in vitro. (20 refs)

- 79-4617 Epstein-Barr Virus Nuclear Antigen (EBNA)-Positive Cells in a Lymph-Node of a Child with Severe Primary EBV Infection.** (Eng) Lenoir, G. (International Agency Res. on Cancer, Lyon, France); De-The, G.; Virelizier, J. L.; Griscelli, C. *IARC Sci Publ* 24(II): 733-738; 1978.

The presence of Epstein-Barr virus (EBV) nuclear antigen (EBNA)-positive cells in a lymph node of a 6-yr-old girl with a severe primary EBV infection is reported. When first seen, the child, who did not have typical clinical symptoms of infectious mononucleosis, showed very high EBV serologic reactivities: A high IgG EBV viral capsid antigen (VCA) titer was associated with anti-VCA IgM antibodies; the EBV early antigen (EA) titer was very high. In contrast, the EBNA titer was 40 and no complement-fixing soluble antigen (CF-S) was detectable. The Paul-Bunnell-Davidsohn test for heterophil antibodies was positive only at a dilution of 1:8. Three mo later, there were no heterophil antibodies, the VCA and EA titers were still very high; the anti-VCA IgM titer dropped considerably; and the EBNA titer had increased to 80. Cell smears from an axillary lymph node biopsy showed approx 1% EBNA-positive cells. The finding of persisting EBNA-positive lymphocytes in the absence of neoplasia strongly supports

the hypothesis that the development of Burkitt's lymphoma occurs in two steps at the cellular level: one involving the transformation of B-lymphocytes by EBV and leading to an EBNA-carrier state, and the other characterized by a cytogenetic change involving translocation of chromosome 14. (11 refs)

- 79-4618** Establishment of Somatic Hybrid Cell Clones from Rat Embryonic Fibroblast Cells with Burkitt's Lymphoblastoid Cells Using Polyethylene Glycol. (Eng) Darai, G. (Institut für medizinische Virologie, Universität Heidelberg, Heidelberg, W. Germany); Doerr, H. W.; Matz, B.; Flugel, R. M.; Zentgraf, H.; Munk, K. *IARC Sci Publ* 24(1): 571-576; 1978.

The polyethylene glycol technique was used to transfer the Epstein-Barr virus (EBV)-carrying chromosome of human Burkitt's lymphoma (BL) cells to the cells of other mammals. One BL-rat hybrid cell clone (BR-H2) retained its ability to grow both in monolayers and as floating cells. The chromosome counts in this clone ranged from 38 to 92 with a modal number of 55. Areas of monolayer cultures that were in the process of producing floating cells were consistently positive for EBV nuclear antigen. EBV-specific antigen (VCA) was detectable in at least 1/200 BR-H2 cells. Nucleocapsidlike structures comparable in size with EBV were observed in the cytoplasm and complete EBV particles were found in the nuclei of at least 1/200 cells. Supernatants of BR-H2 cells were positive for reverse transcriptase, indicating that type-C RNA viruses were activated in this clone. Recently established hybrid cell clones from BL cells and embryo fibroblasts of the primitive prosimian tupaia (tree shrew) had properties similar to those of the BR-H2 cell clone. (7 refs)

- 79-4619** Identification and Partial Purification of Two EBV-associated DNA Polymerases. (Eng) Goodman, S. R. (Sidney Farber Cancer Inst., Boston, MA); Prezyna, C.; Clough, W. *IARC Sci Publ* 24(1): 395-405; 1978.

Two Epstein-Barr virus (EBV)-associated DNA polymerases were partially purified and identified. One of the enzymes was intracellular and the other virion-associated. Both enzymes were in the 8S-10S size range of the intracellular enzyme activity reported to be present in EBV-infected P3HR-1 producer cells but not in Raji or Nunn nonproducer lines. The EBV-induced polymerase differed from α , β , and virion-associated polymerases in being less sensitive to salt inhibition, having a more basic pH optimum in Tris buffer (pH 9.5), and copying "activated DNA" more efficiently. The EBV virion-associated polymerase was distinguishable from the other three enzymes in that it could not use artificial initiated deoxy- and ribohomopolymers as template. The virion-associated

polymerase was highly sensitive to salt inhibition and, unlike the EBV-induced polymerase, was partially inhibited (33%) by 1 mM N-ethylmaleimide. Neither of the EBV-associated enzymes could copy the ribohomopolymers dT₁₀poly(rA) or dG₁₂₋₁₈poly(rC) efficiently compared with activated DNA. The EBV-induced, virion-associated, and β polymerases were unaffected by antibody prepared in rabbits against HeLa cell α polymerase. The EBV-induced polymerase was inhibited by high levels of phosphonoacetic acid (23% inhibition at 100 μ g/ml), but it was less sensitive than the host α polymerase. (13 refs)

- 79-4620** Studies on the Association of the Epstein-Barr Virus Genome with Chromosomes in Human (Burkitt)/Mouse Hybrid Cells. (Eng) Glaser, R. (Dept. Microbiology, Pennsylvania State Univ. Coll. Medicine, Hershey, PA); Croce, C.; Nonoyama, M. *IARC Sci Publ* 24(1): 565-570; 1978.

Somatic cell hybrids of mouse fibroblasts and Burkitt's lymphoma cells were used to further clarify the association between Epstein-Barr virus (EBV) DNA and human chromosomes. A hybrid cell, designated CL1D/HR-1, was cloned in soft agar, and each of 10 clones was assayed for the spontaneous expression of EBV-associated nuclear antigen (EBNA), early antigen (EA), and virus capsid antigen (VCA). Six of 10 clones were EBNA-positive but negative for EA and VCA, even after treatment with iododeoxyuridine. One clone, designated M44, contained approx 90% EBNA-positive cells and 0.3-0.5 EBV genome equivalent per cell. Thirty subclones of clone M44 were analyzed for EBV DNA, EBNA, and human chromosomes; four of these subclones (3 EBNA-positive and 1 EBNA-negative) were studied in detail. None of the four subclones contained any intact human chromosomes. Isozyme analysis indicated that all four subclones, regardless of the status of the EBV genome, synthesized nucleoside phosphorylase, an enzyme that has been linked to human chromosome 14. (5 refs)

- 79-4621** Studies on Epstein-Barr Virus DNA Polymerase Activities in Various Human Lymphoblastoid Cell Lines. (Eng) Ooka, T. (Département de Biologie Générale et Appliquée, Université Claude Bernard, Lyon-I, Villeurbanne, France); Daillie, J.; Costa, O.; Lenoir, G. *IARC Sci Publ* 24(1): 389-393; 1978.

The presence of Epstein-Barr virus (EBV)-directed DNA polymerase activity was studied in three cell lines (BJA-B, Raji, and P3HR-1) derived from Burkitt's lymphoma (BL) and R3HR-2- and in early antigen (EA)-containing cells prepared from Raji cells superinfected with P3HR-1 virus and from P3HR-1 cells following chemical (iododeoxyuridine) induction. About 90% total enzyme activity was

inhibited in all cell lines in the presence of potassium chloride and sodium chloride, and no significant enzyme activities were detected in BJA-B, Raji, or P3HR-1 cell extracts in the presence of 150 mM ammonium sulfate. However, superinfected Raji cells showed about 50% of the total DNA polymerase activity at pH 8.5 and about 29% of the total activity at pH 7 in the presence of 150 mM ammonium sulfate. About 5% of the total activity of chemically induced P3HR-1 cells was evident in the presence of ammonium sulfate. Cellular DNA polymerase activities were almost completely inhibited by ammonium sulfate, but the virus-induced enzyme was partly resistant. The data indicate that EBV-induced DNA polymerase can be found only in highly EA-positive cells. (13 refs)

79-4622 Transcription of Epstein-Barr Virus Genomes in Human Lymphoblastoid Cells and in Somatic-Cell Hybrids of Burkitt's Lymphoma. (Eng) Nonoyama, M. (Life Sciences Biomedical Res. Inst., St. Petersburg, FL); Tanaka, A.; Silver, S.; Glaser, R. *IARC Sci Publ* 24(I): 559-563; 1978.

A study was made of the expression of latent Epstein-Barr virus genomes in D98 human lymphoblastoid cells and in somatic cell hybrids of Burkitt's lymphoma cells. Iododeoxyuridine (IUDR) treatment induced the formation of early antigen (EA), virus capsid antigen, and virus DNA replication in D98/Raji and D98/HR-1 cells, whereas only EA was induced in Raji cells. HR-1 clone No. 9, derived from virus-producing HR-1 cells by treatment with cycloheximide, did not respond to IUDR treatment. The pattern of transcription of virus genomes in these cell lines without IUDR treatment was uniform, with 20%-25% of the virus DNA being transcribed. IUDR treatment enhanced the transcription of virus DNA to 50% in D98/Raji, D98/HR-1, and Raji cells, but no enhancement of virus genome transcription was observed in HR-1 clone No. 9. The amount of virus RNA in the cells calculated from DNA-RNA hybridization kinetics was found to be proportional to the number of virus genomes per cell, indicating that every copy of virus DNA in these cells is actively transcribed. (5 refs)

79-4623 Inhibition of Epstein-Barr Virus Transformation: Evidence for a Block Between EBNA Production and Cell Proliferation. (Eng) Pope, J. H. (Oncology Unit, Queensland Inst. Medical Res., Herston, Queensland, Australia); Moss, D. J. *IARC Sci Publ* 24(II): 617-622; 1978.

Macrophages and fibroblasts were used as control feeder layers to study cell relationships in Epstein-Barr virus (EBV) transformation of lymphocytes from humans with previous EBV infection. Inhibition of transformation on adult fibroblasts was observed with nonadherent lymphocytes from several donors with prior EBV infection but not with lymphocytes from cord blood. Inhibition of transformation was dependent on prior EBV infection of the donor of the lymphocytes but not on previous infection of the donor of the fibroblasts. Inhibition was detectable only in the presence of adult fibroblasts. Addition of adult fibroblasts to nonadherent lymphocytes up to 2-4 days after EBV infection also resulted in inhibition of transformation. These results indicate that EBV transformation involves two recognizable phases, with an immunological block between EBV nuclear antigen production and cell proliferation. (7 refs)

79-4624 Intracranial Heterotransplantation of Human Hematopoietic Cells in Nude Mice. (Eng) Schaadt, M. (Abteilung für Haematologie/Onkologie, D 3000 Hanover, W. Germany); Kirchner, H.; Fonatsch, C.; Diehl, V. *Int J Cancer* 23(6): 751-761; 1979.

To evaluate whether tumor formation of human lymphoid cells transplanted into the brains of nude (BALB/c nu/nu) mice is restricted to malignant cells, tests were conducted with a wide variety of established cell lines and several primary biopsy specimens. Not only lymphoma and leukemia cell lines, but also lymphoblastoid cell lines lacking markers of malignancy, were tumorigenic in the brains of the nude mice. These findings indicate that tumorigenicity following intracranial heterotransplantation in nude mice cannot be used as proof of the malignant nature of established cell lines. Heterotransplantation of primary cell specimens yielded only a few tumor takes. When primary cells were infected with exogenous Epstein-Barr virus prior to transplantation, tumorigenicity was increased significantly. Cytogenetic evaluation of tumors growing after intracranial transplantation of human hematopoietic cells showed, in some cases, a selection of cytogenetically aberrant cell clones. (45 refs)

79-4625 In Vitro Lymphocyte Transformation by Epstein-Barr Virus (EBV)-like Viruses Isolated from Old-World Non-human Primates. (Eng) Rabin, H. (Viral Oncology Program, NCI Frederick Cancer Res. Center, Frederick, MD); Neubauer, R. H.; Hopkins, R. F.; Rasheed, S. *IARC Sci Publ* 24(I): 553-557; 1978.

Lymphoid cell lines were established and Epstein-Barr virus (EBV)-like viruses isolated from baboons and an orangutan. The cell lines have the properties of B or undifferentiated lymphocytes, and they have antigens and DNA related to those of EBV. The baboon virus has a broad in vitro transformation host range among lymphocytes of Old World simian species, but the orangutan isolate has a narrower host range. Baboon and orangutan viruses as well as EBV show transforming activity for gibbon lymphocytes. Baboon virus is infectious for rhesus monkeys and baboons, but it has not induced neoplastic disease in these

species. The isolation of these simian EBV-like viruses indicates that EBV is a member of a family of primate lymphotropic herpesviruses that are related to each other antigenically and by DNA homology and that share biological activity, including the ability to transform cells in vitro. (10 refs)

- 79-4626 Suppression of In Vitro Epstein-Barr Virus Infection: A New Role for Adult Human T-Lymphocytes.** (Eng) Thorley-Lawson, D. A. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA); Chess, L.; Strominger, J. L. *IARC Sci Publ* 24(II): 623-626; 1978.

B lymphocytes from fetal cord blood and from the peripheral blood of Epstein-Barr virus (EBV) seropositive and seronegative adult donors were infected with EBV, and the rate of DNA synthesis was determined. There were no significant differences in the number of cells infected or the rate at which the infected cells proliferated. In subsequent experiments, correlations between the rate of DNA synthesis and the rate of appearance of EBV-transformed cells were studied. Unfractionated adult lymphocytes transformed less efficiently than those from fetal cord blood. However, separated adult B cells grew out as fast (13-14 days) and with the same efficiency (100% in 2 wk) as did B cells from fetal cord blood. Mixtures of 20% fetal B lymphocytes and 80% autologous Ig-negative lymphocytes were strongly inhibited in their ability to grow out, compared with pure adult B lymphocytes. The outgrowth of infected adult B cells was suppressed by the addition of >60% Ig-negative lymphocytes, which suggested that the suppression was the result of a specific interaction between adult Ig-negative lymphocytes with the infected B cells. The suppressor cells were adult T lymphocytes; fetal T lymphocytes were not suppressive. (no refs)

- 79-4627 Differential Inducibility of Epstein-Barr Virus in Cloned, Non-producer Raji Cells.** (Eng) Bister, K. (Dept. Molecular Biology, Univ. California, Berkeley, CA 94720); Yamamoto, N.; zur Hausen, H. *Int J Cancer* 23(6): 818-825; 1979.

In order to test the possibility that cultures derived from single cells might differ in their inducibility of Epstein-Barr virus (EBV)-associated early antigen (EA) synthesis, cells of the human lymphoblastoid nonproducer line Raji were cloned in soft agar, and individual colonies were analyzed for EA inducibility. The induction of EA by the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) varied among the 24 different cell clones. Clones with very high and very low inducibility of the resident EBV genome were further analyzed. Constant differences in the inducibility of EA were observed after activation of the clones by tumor promoters, 5-iododeoxyuridine, or antibodies to

human IgM. Induction of EA synthesis by superinfection of the cells with EBV from the P3HR-1 line, however, did not vary among the clones tested. No differences in the expression of the EBV-associated nuclear antigen were noted in cells of clones with high or low susceptibility to EA induction. DNA reassociation kinetics demonstrated that Raji cells with high susceptibility to EA induction contained a significantly higher number of EBV genome equivalents per cell than cells with low susceptibility. Treatment of Raji cells with TPA did not change the ratio of EBV-specific DNA to cellular DNA. (46 refs)

- 79-4628 Search for Human Tumour Viruses by Transfection: Uptake of Melanoma and Epstein-Barr Virus DNA by Human Cells.** (Eng) Brown, S. G. (Queensland Inst. Medical Res., Bramston Terrace, Herston, Queensland 4006, Australia); Parsons, P. G.; Pope, J. H. *Aust J Exp Biol Med Sci* 57(part 1): 1-7; 1979.

Transfection of chicken embryo fibroblasts (CEF) and human cells with melanoma cell DNA or Epstein-Barr virus (EBV) to obtain high yields of virus was studied. Transfection consistently occurred when DNA from the Rous sarcoma virus-transformed rat cell line XC was inoculated onto CEF cultures, but application of XC DNA to human fibroblasts gave negative results. No evidence of transfection was found when the DNA from three malignant melanoma cell lines (MM96, 200, and 253) was inoculated onto human fibroblasts or cord lymphocytes. Negative results were also obtained when DNA from the EBV-infected Raji and QIMR-WIL cell lines was inoculated onto human cord lymphocyte or amnion cells. Calcium treatment enhanced the uptake of Raji DNA by cord mononuclear cells. When five human fibroblast lines were treated with calcium-precipitated DNA from two melanoma lines, the amount of labeled DNA at the cell surface (6%-12%) was much lower than that observed with Raji DNA and cord mononuclear cells (47%). The level of intracellular labeled DNA was similar with the melanoma and Raji DNA samples (8.8%-14.3%). There appeared to be no major difference between the nuclear uptake in a system showing transfection (XC DNA in CEF) and that in systems that did not. No EBV, melanoma retrovirus, or other potential tumor virus was recovered. (23 refs)

- 79-4629 Epstein-Barr Virus-induced Membrane Antigens: Immunochemical Characterization of Triton X-100 Solubilized Viral Membrane Antigens from EBV-superinfected Raji Cells.** (Eng) Qualtiere, L. F. (Dept. Microbiology, Mayo Clinic/Foundation and Mayo Medical Sch., Rochester, MN 55901); Pearson, G. R. *Int J Cancer* 23(6): 808-817; 1979.

To qualitatively identify the membrane antigen (MA) complex induced by Epstein-Barr virus (EBV) infection of lymphoblastoid cells, superinfected Raji cells were surface-

labeled with ^{125}I by the lactoperoxidase method and solubilized with Triton X-100. The ^{125}I -labeled membrane proteins were then precipitated by sera containing high antibody titers to MA. Analysis of these immune precipitates by sodium dodecyl sulfate-polyacrylamide gel electrophoresis identified four major EBV-specific membrane proteins with mol wts of 280,000 daltons (280K), 250K, 170K, and 90K. Sera from patients with Burkitt's lymphoma (BL), nasopharyngeal carcinoma (NPC), and infectious mononucleosis (IM) and from EBV-infected disease-free individuals showed different patterns of reactivity to these antigens, with some sera only recognizing three or less of the antigens. The four major proteins were not related to EBV-induced viral capsid antigen (VCA) or to the virus-associated early antigen (EA) complexes, as defined by immunofluorescence. Metabolic labeling of EBV-infected Raji cells with [^{14}C]glucosamine, followed by Triton X-100 solubilization and radioimmune precipitation, identified the 280K, 250K, and 90K components as glycoproteins. The lactoperoxidase-labeled 170K component was not significantly glycosylated and, therefore, could not be categorized as a glycoprotein on the basis of this study. In addition, a glycoprotein with a mol wt of 130K identified by this approach also appeared to be specified by EBV. The results of these investigations indicate that the EBV-induced MA complex is composed of four major glycoproteins and one nonglycosylated high-mol-wt protein. (24 refs)

79-4630 Light and Electron Microscopic Investigations of Nasopharyngeal Carcinomas with Regard to the Viral Etiology of These Tumors. (Eng) Arnold, W. (Pathologisches Institut der Universität Dusseldorf, Moorenstrasse 5, D-4000 Dusseldorf, W. Germany); Huth, F. *J Cancer Res Clin Oncol* 94(1): 87-109; 1979.

Five nasopharyngeal carcinomas (4 Regaud-type lymphoepithelial carcinomas and 1 squamous cell carcinoma) were examined light and electron microscopically. Because of the increased antibody titers against Epstein-Barr virus antigens in all five patients, all cytoplasmic and nuclear inclusions that could be interpreted as indicative of virus contact were examined. The following structures were found: (1) particles and microtubules that corresponded in diameter, shape, and location to corona viruses; (2) particles surrounded by a double membrane and resembling in form and diameter oncornaviruses; (3) tubuloreticular, coil-shaped cytoplasmic inclusions interpreted as being an unspecific reaction of the host cell to viral attack; (4) spherical nuclear bodies, which are frequently observed in tumors and in viral infections; (5) intranuclear particles that corresponded in diameter, structure, and distribution to herpes-type viruses, such as those that have been described in cell cultures of Burkitt's lymphoma and nasopharyngeal carcinoma. These findings plus the appearance of increased numbers of ribosomes and of a particular chromatin distribution within the tumor cell nuclei

indicate that the particles are morphological indications of a viral etiology of the five tumors. (76 refs)

79-4631 Viruses, Virus-like and Virus-related Structures in Nasopharyngeal Carcinoma. (Ger) Arnold, W. (Hals-, Nasen- und Ohrenklinik, Universität Dusseldorf, Moorenstrasse 5, 4000 Dusseldorf, W. Germany); Huth, F. *Arch Otorhinolaryngol* 222(4): 295-317; 1979.

Five cases of histologically confirmed nasopharyngeal carcinoma were studied by light and electron microscopy. The Epstein-Barr virus (EBV) antigen titers were typically elevated in all cases (1:2,048 or higher). Corona viruses of different shapes, regular capsid-like particles with a diameter of 60 nanometers (nm), tubuloreticular aggregates, and pathological alterations of the members of the endoplasmic reticulum were found in the cytoplasm. The tumor cell nuclei often showed particles within the nucleoplasm with an av diameter of 100 nm and containing a central core; the appearance of the particles was similar to that of herpesvirus as shown in the literature concerning EBV. Atypical mitoses were common, as were free nucleoplasm-like condensates within the cytoplasm. Multiple doubling of the membranes of the endoplasmic reticulum were characteristic of the tumor cell cytoplasm. Myelin figures were often located within the enlarged tubules of the endoplasmic reticulum and in the vicinity of the mitochondria and the nuclei. The particles found within the nucleoplasm are not believed to be related to nuclear pores. (69 refs)

79-4632 Circulating Immune Complexes in Nasopharyngeal Carcinoma Patients and Their Household Contacts. (Eng) Lamelin, J. P. (International Agency Res. Cancer, INSERM U 80, Lyon, France); Vincent, C.; Charnay, B.; Revillard, J. P. *Protides Biol Fluid Proc Colloq* 26: 353-358; 1978.

The levels of complement C1q binding activity (C1qBA) and anti-Epstein-Barr virus (EBV) capsid antigen (VCA), anti-EBV early antigen (EA), and anti-EBV nuclear antigen (EBNA) antibody titers were determined in sera from 29 patients with nasopharyngeal carcinoma (NPC), 133 household contacts, and 66 unrelated matched healthy controls. The mean value of C1qBA was higher among NPC patients than among household contacts ($p = 0.05$), and more household contacts than unrelated controls were positive for C1qBA ($p < 0.001$). The IgG anti-VCA geometric mean titer (GMT) of household contacts was lower than that of the probands ($p < 0.001$) and higher than that of unrelated controls ($p < 0.01$). Similarly, the anti-EBNA GMT of household contacts was lower than that of the probands ($p < 0.01$) and higher than that of unrelated controls ($p < 0.01$). The prevalence of anti-EA

was similar among household contacts and unrelated controls, but it was significantly higher among NPC patients ($p < 0.001$). Spouses and first-degree relatives of NPC patients did not differ significantly in any of these parameters. Among NPC patients and household contacts, the level of C1qBA was significantly correlated with the titer of IgG anti-VCA antibodies ($p = 0.026$, $p < 0.01$, respectively). There were no other significant correlations. (17 refs)

79-4633 Effects of Arabinosyl-Cytosine on Thymidine Triphosphate Pools and Polyoma DNA Replication. (Eng) Hellgren, D. (Dept. Biochemistry, Medical Nobel Inst., Karolinska Inst., S-104 01 Stockholm, Sweden); Nilsson, S.; Reichard, P. *Biochem Biophys Res Commun* 88(1): 16-22; 1979.

At 5-100 nanomolar, cytosine arabinoside inhibited 3H-thymidine incorporation into the polyoma DNA of infected mouse fibroblasts without affecting the labeling of the 3H-deoxythymidine triphosphate (dTTP) pool. Inhibition of DNA synthesis was shown to affect chain elongation and not the initiation of new cycles of replication. The new simple method used to determine the specific activity of dTTP should be applicable to other deoxynucleoside triphosphates. (10 refs)

79-4634 Evidence for the Expression of TSTA in BKV-transformed Cells: Cross-reaction with SV40 TSTA. (Eng) Seehafer, J. (Dept. Biochemistry, Univ. Alberta, Edmonton, Alberta T6G 2H7, Canada); Downer, D. N.; Gibney, D. J.; Colter, J. S. *Virology* 95(1): 241-243; 1979.

BALB/c mice that had received two ip injections of BK virus-transformed mouse, rat, and hamster cells (killed by exposure to γ -irradiation) were shown to be resistant to subsequent challenge with cells of a syngeneic simian 40 (SV40) virus-transformed line, mKSA-ASC. The degree of resistance acquired by the mice differed sharply depending on the line used for immunization, with resistance indices ranging from 0 to >6000. A low but significant level of resistance was induced by the injection of BKV, but none was induced by the injection of polyoma virus-transformed cells or untransformed mouse, rat, or hamster cells. The data show that BKV-transformed cells contain a tumor-specific transplantation antigen (TSTA) that is closely related to SV40 TSTA. (19 refs)

79-4635 Structure of Late Adenovirus 2 Heterogeneous Nuclear RNA. (Eng) Berget, S. M. (Center for Cancer Res., Massachusetts Inst. Technology, Cambridge,

MA 02139); Sharp, P. A. *J Mol Biol* 129(4): 547-565; 1979.

Late adenovirus 2 (Ad 2) messenger RNA's (mRNA's) are processed from a single transcriptional unit some 20,000 nucleotides in length. All of these cytoplasmic RNA's have multiple leaders spliced to their 5' termini; the most common form is a tripartite leader set. Sequences complementary to the 5' proximal leader lie adjacent to position 17, the site for initiation on this transcriptional unit. The structure of steady-state nuclear RNA in late Ad 2-infected HeLa cells was analyzed by electron microscopy of hybrids between nuclear RNA and restriction endonuclease cleavage fragments and by digestion of similar hybrids with single-strand-specific nucleases and resolution of the protected DNA strands by electrophoresis in agarose gels. While all cytoplasmic mRNA's have three or more leader segments spliced together following excision of appropriate intervening sequences, long nuclear RNA contains some or all of these intervening sequences. Long, completely colinear nuclear RNA's have 5' termini at position 17 on the genome. All other nuclear RNA forms with one or two intervening segments excised have a structure consistent with the proposition that the 5' proximal splice must occur before the second internal segment of intervening sequences can be excised. Long nuclear viral RNA's typically have unique 3' termini, which map in the same position as the 3' termini of cytoplasmic mRNA's. In general, the structure of steady-state nuclear late Ad 2 RNA is consistent with these molecules being intermediates in the intramolecular processing of mRNA from a much longer initial transcript. (40 refs)

79-4636 The Major Late Adenovirus Type-2 Transcription Unit: Termination is Downstream from the Late Poly(A) Site. (Eng) Fraser, N. W. (Rockefeller Univ., New York, NY 10021); Nevins, J. R.; Ziff, E.; Darnell, J. E. *J Mol Biol* 129(4): 643-656; 1979.

Late in adenovirus type-2 (Ad 2) infection of HeLa cells, the majority of viral RNA synthesis occurs from a transcription unit that extends between 16% and 100% on the physical map of the Ad 2 genome. Poly(A) is added to RNA at five distinct sites within the transcription unit. As many as 13 different messenger RNA's (mRNA's) can be processed from the primary RNA transcripts of this region of the genome. Examination of nuclear RNA in several different experiments (RNA fingerprint studies and hybridization of pulse-labeled nascent RNA and labeled nuclear RNA synthesized after UV irradiation to an ordered set of restriction endonuclease-generated DNA fragments) indicated that the mRNA 3' terminus most distant from the promoter does not correspond to the RNA polymerase termination site for the transcription unit. Transcription continued approx 2500 to 3000 residues beyond the 3' terminus of the most promoter distal mRNA (localized between coordinates 89.7 and 91.9) and into the region 98.2-

100 on the Ad 2 physical map. The occurrence of RNA termination sites distal to the poly(A) site may be a general feature of transcriptional unit design in animal cells. (39 refs)

- 79-4637 Derivation and Characteristics of Seven Lines of Rat Embryo Cells Transformed by Adenovirus Type 5 and Its DNA.** (Rus) Zalmanzon, E. S. (Inst. Molecular Biology, Moscow, USSR); Frolova, E. I.; Richter, B.; Mikhailova, L. N.; Turetskaia, R. L.; Savina, A. A.; Bobrova, N. B. *Mol Biol (Mosk)* 13(2): 292-308; 1979.

A series of cell lines transformed by adenovirus type 5 (Ad5) were characterized with respect to their content of Ad DNA sequences and their biological properties. Random-bred rat embryo cells were transformed by Ad5, and August rat embryo cells were transformed by virus DNA. A total of seven transformed cell lines were obtained. All seven lines did not produce mature virus, and their ability to produce virus particles did not change after repeated inoculation. All the lines were sensitive to reinfection with Ad5. These findings indicate that the presence of the virus genome is due not to chronic infection but to virus-induced transformation. None of the lines induced tumors, even in immunosuppressed newborn rats. However, they all formed colonies in soft agar. Analysis of the reassociation rate of the fragments produced by cleavage with restriction endonuclease *HpaI* with ³²P-labeled Ad DNA fragments showed that the DNA from the transformed lines contains at least 6% of the left "oncogenic" end of Ad DNA and that the number of copies (per diploid complement) ranges from 2.7 to 7.7. Three lines contained approx 16% of the right end of virus DNA, with the number of copies ranging from 1.6 to 4.5. (36 refs)

- 79-4638 Incomplete Transformation of Rat Cells by a Small Fragment of Adenovirus 12 DNA.** (Eng) Shiroki, K. (Inst. Medical Science, Univ. Tokyo, Shirokanedai, Minato-ku, Tokyo 108, Japan); Shimojo, H.; Sawada, Y.; Uemizu, Y.; Fujinaga, K. *Virology* 95(1): 127-136; 1979.

Rat cells (3Y1) were transformed by the *BpaI*-H fragment (4.5%, the left end) of adenovirus 12 (Ad 12) DNA, cloned, and seven transformed cell lines (HY1-HY7 cells) were established. Most of the viral DNA sequences of the *BpaI*-H fragment were present in HY1 and HY2 cells. The properties of HY1, HY2, and HY7 cells were compared with those of GY1 cells, a rat cell line transformed by the *HindIII*-G fragment of Ad12 DNA; CY1 cells, a rat cell line transformed by the *EcoRI*-C fragment; and WY3 cells, a rat cell line transformed by whole Ad 12 DNA. The morphology and the growth of HY1, HY5, and HY7 cells in Eagle's minimal essential medium with 10% fetal calf

serum resembled that of CY1 cells, demonstrating the transformed phenotype. However, the following properties of HY1, HY5, and HY7 cells were intermediate between transformed and untransformed cells: (1) growth in Eagle's medium with 2% fetal calf serum; (2) colony formation in soft agar culture; (3) cellular DNA synthesis in Methocel medium; and (4) tumor growth after transplantation into rats. Ad 12 tumor antigens were not detected in HY cells by immunofluorescence, and viral antigen could not be detected by a complement-fixation test. These results indicate that HY cells do not express the complete transformed phenotype. It is possible that the *BpaI*-H fragment contains the genetic information for the initiation of transformation but lacks the information necessary for the maintenance of transformation. (19 refs)

- 79-4639 Parameters Affecting the Stability of SV40 Virions During the Extraction of Nucleoprotein Complexes.** (Eng) Seidman, M. (Dept. Biochemical Sciences, Princeton Univ., Princeton, NJ 08540); Garber, E.; Levine, A. J. *Virology* 95(1): 256-259; 1979.

Monolayer cultures of BSC-1 cells infected with simian virus 40 (SV40) were used to characterize the parameters affecting the stability of SV40 virions during the extraction of viral nucleoprotein complexes. The major parameters were identified as the ionic strength of the extraction solution, which should be between 50 and 100 mM KCl; chelators such as EDTA, which must be eliminated from the extraction solution but may be used in subsequent analyses; and the concentration of nuclei, which should be kept below 10⁷/ml of extraction buffer to minimize virion disruption by cellular factors in the nuclear extract. The instability of SV40 virions in crude nuclear extracts in vitro may be analogous to the virus uncoating reactions that permit virus infection to begin in vivo. (9 refs)

- 79-4640 Fused Cells Are Suited for Microinjection.** (Eng) Graessmann, A. (Institut für Molekularbiologie, Biochemie der Freien Universität Berlin, Arnimallee 22, D-1000 Berlin 33, W. Germany); Graessmann, M.; Mueller, C. *Biochem Biophys Res Commun* 88(2): 428-432; 1979.

Volumes transferred per cell by microinjection were enhanced by a factor of 10⁴-10⁵ when multinucleated HeLa cells fused by polyethylene glycol 1000 were used as recipients instead of single HeLa cells. Fused HeLa cells were viable and supported simian virus 40 gene expression upon microinjection of the viral DNA. (14 refs)

- 79-4641 Removal of T4 Endonuclease V-sensitive Sites from SV40 DNA after Exposure to Ultraviolet**

Light. (Eng) Williams, J. I. (Dept. Pathology, Stanford Univ. Medical Center, Stanford, CA 94305); Cleaver, J. E. *Biochim Biophys Acta* 562(3): 429-437; 1979.

T4 endonuclease V-sensitive sites in simian virus 40 (SV40) Form I DNA (supercoiled) were determined at various times after UV irradiation of cultured monkey CV-1 cells infected with SV40 (5-10 plaque-forming units/cell) for 60-85 hr. Agarose tube gel electrophoresis of the irradiated viral DNA showed that endonuclease-sensitive sites were induced at a rate of 0.049 sites/SV40 genome per joules (J)/m², or 1.4 sites/1.10⁸ daltons of DNA per J/m². This value was similar to the yield of endonuclease-sensitive sites and pyrimidine dimers in uninfected host CV-1 cell DNA. Removal of endonuclease-sensitive sites was dose-dependent and nonlinear for at least 24 hr after irradiation. These results suggest that SV40 DNA is subject to the excision-repair mechanisms of the host cell and that the excision of pyrimidine dimers is one of the biochemical events underlying host cell reactivation. (41 refs)

79-4642 Isolation and Characterization of L-Methionine-resistant Mutants of SV40-transformed Balb 3T3 (SVT2) Affecting L-Methionine Transport. (Eng) Heiser, W. (Salk Inst., La Jolla, CA); Englesberg, E. *Somatic Cell Genet* 5(3): 345-361; 1979.

Single- and multistep procedures for isolating L-methionine-resistant (met-r) mutants of simian virus 40 (SV40)-transformed BALB 3T3 mouse cells (SVT2) are described. In addition, since differences in sensitivity to amino acid inhibition of growth may reflect differences in ability to transport certain amino acids, the ability of SVT2 and BALB 3T3 cells to transport met was compared. The SVT2 met-r mutants were stable and arose in the absence of the selective agent at a mutation rate of 3.5×10^{-5} . Treatment with ethyl methanesulfonate raised the frequency of met-r mutants 32- to 47-fold. SVT2 cells transported met at an initial rate that was 4.5 times that of the 3T3 cells. The met-r mutants had a decreased initial rate of met transport. Some mutants showed both an increase in Km and a decrease in Vmax, but others showed only an increase in Km or a decrease in Vmax. Kinetics studies permitted the identification of two met transport systems for 3T3 cells and only one for SVT2 cells and the met-r mutants. The Vmax for SVT2 cells was three times larger than that for the high-affinity system of 3T3 cells, but the Km's were the same. The change in initial rate of transport of a few specific amino acids besides met suggests that effects on transport observed with both transformed and mutant cells are not due solely to a general membrane change. A comparison of the amino acid spectrum of inhibition of independently transformed SV40 cell lines demonstrated that each transformant is unique. (23 refs)

79-4643 Nucleocytoplasmic Interactions in Experimental Binucleates Formed from Normal and Transformed Components. (Eng) Muggleton-Harris, A. L. (Dept. Life Sciences, Worcester Polytechnic Inst., Worcester, MA 01609); Palumbo, M. *Somatic Cell Genet* 5(3): 397-407; 1979.

The nuclear transfer and fusion of individual viable nuclei into late phase normal and transformed human fibroblasts are described. Experimental binucleates were formed by fusing nuclei from normal fibroblasts (line WI-38), simian virus 40-transformed fibroblasts, and HeLa carcinoma cells into individual diploid fibroblasts that had undergone their total life-span (population doublings) and had been maintained in culture for 10-12 wk (late phase III) prior to the nuclear transfer. Many of the cells receiving additional nuclei replicated and formed clones of cells with a limited life-span; the morphology of these cells remained fibroblastlike. (21 refs)

79-4644 Structure of Simian Virus 40 Recombinants That Contain Both Host and Viral DNA Sequences. I. The Structure of Variant CVP8/1/P2 (*EcoRI* res). (Eng) Wakamiya, T. (Lab. Biochemistry and Molecular Biology, NCI, NIH, Bethesda, MD 20205); McCutchan, T.; Rosenberg, M.; Singer, M. *J Biol Chem* 254(9): 3584-3591; 1979.

The primary structure of the defective simian virus 40 (SV40) genome, CVP8/1/P2 (*EcoRI* res), was studied. This virus contains SV40 DNA sequences, DNA segments homologous to monkey α component, and DNA segments derived from less highly reiterated sequences in the genome of the African green monkey. The basic repeat unit of CVP8/1P2 (*EcoRI* res) is a DNA segment 1,210 base pairs in length. There are 427 contiguous base pairs of wild-type SV40 sequence, including the region identified with the origin of replication. Of these 427 base pairs, 393 appear as they do in the wild-type genome and 34 have been inverted. Residues 648 through 803 of the basic repeat unit are homologous to 156/172 base pairs in the highly reiterated α component repeat unit of African green monkey DNA. There were differences at two positions, residues 662 and 676. Between 25% and 33% of the basic repeat units in CVP8/1P2 (*EcoRI* res) DNA contain, in addition to the 1,210 base pairs, an extra segment of DNA. The extra segment corresponds to a duplication of base pairs 345 through 567 of the basic repeat unit, inserted between residues 567 and 568. The data demonstrate that the various recombination events that resulted in the formation of this defective variant did not depend on extensive homology between recombining segments. (36 refs)

79-4645 Structure of Simian Virus 40 Recombinants That Contain Both Host and Viral DNA Se-

quences. II. The Structure of Variant 1103 and Its Comparison to Variant CVP8/1/Pw (*EcoRI* res). (Eng) McCutchan, T. (Labs. Molecular Biology and Biochemistry, NCI, NIH, Bethesda, MD 20205); Singer, M.; Rosenberg, M. *J Biol Chem* 254(9): 3592-3597; 1979.

To characterize sites of recombination between simian virus 40 (SV40) and monkey DNA, the primary sequence of a large portion of the SV40 variant 1103 was determined. This virus contains DNA sequences derived from the wild-type SV40 genome and from the permissive monkey cell in which the virus was propagated. The monkey sequences included in the defective genome were homologous to both highly repeated monkey DNA (α component) and sequences that are infrequently repeated in the monkey genome. The regions of the 1103 genome where DNA sequences were determined included the segments of the variant that surrounded joints connecting SV40 and monkey sequences; the segment that contained the joint between monkey sequences of high and low reiteration frequency; and the DNA segment of the variant that was homologous to monkey α component DNA. Comparison of the data obtained from the sequence analysis of the SV40 variants 1103 and CVP1/1/p2 (*EcoRI* res) revealed similarities between the two that may be involved in eukaryotic recombination and defective variant formation. (28 refs)

79-4646 Characterization of a 54K Dalton Cellular SV40 Tumor Antigen Present in SV40-transformed Cells and Uninfected Embryonal Carcinoma Cells. (Eng) Linzer, D. I. (Dept. Biochemical Sciences, Princeton Univ., Princeton, NJ 08540); Levine, A. J. *Cell* 17(1): 43-52; 1979.

An intermediate mol wt [54,000-dalton (54K)] simian virus 40 tumor (T) antigen of murine cells was characterized. SV40 infection or transformation of murine cells stimulated the production of a 54K protein that was specifically immunoprecipitated, along with SV40 large T and small t antigens, with sera from mice or hamsters bearing SV40-induced tumors. The same SV40 anti-T sera immunoprecipitated a 54K protein from two different, uninfected murine embryonal carcinoma cell lines. These 54K proteins from SV40-transformed mouse cells and the uninfected embryonal carcinoma cells had identical partial peptide (PP) maps that were different from the PP map of SV40 large T antigen. An Ad2 + ND4-transformed hamster cell line also expressed a 54K protein that was specifically immunoprecipitated by SV40 T sera. The PP maps of the mouse and hamster 54K protein were different, showing the host cell species specificity of these proteins. The 54K hamster protein was also unrelated to the Ad2 + ND4 SV40 T antigen. Analogous proteins immunoprecipitated by SV40 T sera, ranging in mol wt from 44K to 60K, were detected in human and monkey SV40-infected or transformed cells. A wide variety of sera from hamsters

and mice bearing SV40-induced tumors immunoprecipitated the 54K protein of SV40-transformed cells and murine embryonal carcinoma cells. Antibody produced by somatic cell hybrids between a B cell and a myeloma cell (hybridoma) against SV40 large T antigen also immunoprecipitated the 54K protein in virus-infected and -transformed cells, but it did not do so in the embryonal carcinoma cell lines. It is concluded that SV40 infection or transformation of mouse cells stimulates the synthesis or enhances the stability of a 54K protein. This protein appears to be associated with SV40 T antigen in SV40-infected and -transformed cells, and it is coimmunoprecipitated by hybridoma sera to SV40 large T antigen. The 54K protein either shares antigenic determinants with SV40 T antigen or is itself immunogenic when associated with SV40 large T antigen. The protein varies with the host cell species, and analogous proteins were observed in hamster, monkey, and human cells. The role of this protein in transformation is unclear. (46 refs)

79-4647 Characterization of the Replicative Structures of the DNA of a Herpesvirus (Pseudorabies). (Eng) Ben-Porat, T. (Dept. Microbiology, Vanderbilt Univ. Sch. Medicine, Nashville, TN); Jean, J. H.; Blankenship, M. L.; Tokazewski, S. *IARC Sci Publ* 24(1): 63-73; 1978.

The sedimentation characteristics of pseudorabies virus (PRV) DNA synthesized at various times after infection of RK cells were correlated with the structures of intracellular viral DNA observed by electron microscopy. During the early stages of PRV infection, newly synthesized DNA was associated with molecules sedimenting with an S value up to twofold greater than that of mature viral DNA. These molecules represented unit-size linear or circular molecules, as well as small concatemers in the process of replication. Initiation of replication occurred at a site situated 20 μ m from one of the ends as well as at or near the end of the molecules. At later times, newly synthesized DNA was associated with large, "tangled" concatemers containing single-stranded segments of DNA. The data indicate that at least some of the single-stranded DNA may be produced during the extraction procedure. The large, tangled concatemers consist of linear arrays of viral DNA molecules. (21 refs)

79-4648 SV40-Transformation of Skin Fibroblasts from a Cystinotic Patient: Establishment of an Immortal Cell Clone that Retains the Original Metabolic Defects (Meeting Abstract). (Ger) Flugel, R. M. (Institut für Virusforschung, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg 1, W. Germany); Harms, E.; Darai, G. *Hoppe Seylers Z Physiol Chem* 360(3): 259; 1979 (no refs)

- 79-4649 On the Presence of Reverse Transcriptase in Myelo- and Lymphoproliferative Disorders.**
(Eng) van Muijen, G. N. (Dept. Pathology, Univ. Medical Center, Wassenaarseweg 62, P.O. Box 9603, 2300 RC Leiden, Netherlands); te Velde, J.; den Ottolander, G. J.; Brand, A.; Koopman-Broekhuizen, N.; Schaberg, A.; Warnaar, S. O. *Cancer* 43(5): 1682-1688; 1979.

The distribution and amounts of reverse transcriptase in different hematological malignancies and in normal controls were determined. Buffy coats from 31 patients with a diagnosis of leukemia and from 16 normal donors were tested for the presence of virallike reverse transcriptase. Eighty-five percent of the fresh leukemic buffy coats were positive. Also tested were 16 spleens from patients with hematological disorders and 5 spleens from patients without a history of hematological malignancy. The five normal spleens were negative. Also negative were four spleens from patients with hairy cell leukemia. Of the remaining 12 spleens, 7 were positive. Reverse transcriptase assays can be used to distinguish leukemic from normal buffy coats. (25 refs)

- 79-4650 Spontaneous Production of a C-Type RNA Virus in a Cell Line Derived from Rat Glioma.**
(Eng) Igarashi, K. (Biological Res. Labs., Central Res. Div., Takeda Chemical Industries, Ltd., Osaka, Japan); Sasada, R.; Niiyama, Y.; Kozai, Y.; Sugino, Y. *Microbiol Immunol* 23(1): 1-15; 1979.

The spontaneous production of a rat C-type RNA virus (ACV) in a cultured cell line (AC) established from an ethylnitrosourea-induced Sprague-Dawley rat glioma was studied. The ACV had a morphology typical of that of a C-type RNA virus, a buoyant density of 1.15 g/ml in a sucrose density gradient, RNA-directed DNA polymerase activity, a viral core with a density of 1.28-1.30 g/ml, 70S RNA with a dimer structure, and a structural protein composed mainly of four polypeptides. Analysis of DNA-DNA hybridization kinetics revealed that DNA sequences homologous to DNA transcripts of ACV RNA were pre-

sent in the rat cells. The RNA-directed DNA polymerase partially cross-reacted with antiserum to the polymerase of Rauscher murine leukemia virus. These data suggest that ACV is an endogenous C-type RNA virus of rat origin. (43 refs)

- 79-4651 Isolation and Characterization of an Endogenous Type C Virus of Rhesus Monkeys.**
(Eng) Rabin, H. (Biological Carcinogenesis Program, Frederick Cancer Res. Center, Frederick, MD 21701); Benton, C. V.; Tainsky, M. A.; Rice, N. R.; Gilden, R. V. *Science* 204(4395): 841-842; 1979.

A C-type retrovirus (MMC-1) was isolated from a continuous cell line established from a spontaneous esophageal carcinoma of a rhesus monkey (*Macaca mulata*) by prolonged cocultivation with canine cells. A DNA transcript of the viral RNA hybridized to a high level, and kinetic analysis indicated the presence of multiple copies of the viral genome in rhesus monkey DNA, showing that MMC-1 virus is endogenous in this species. Based on its antigen profile, buoyant density, number of viral copies in host cells, and cross-hybridization levels, MMC-1 virus is closely related to an endogenous C-type virus previously isolated from stumptailed macaques (*Macaca arctoides*). (9 refs)

See also

- * (Rev.): 79-4233, 79-4270, 79-4275, 79-4276, 79-4277, 79-4278, 79-4279, 79-4280, 79-4281, 79-4282, 79-4283, 79-4288, 79-4317.
* (Chem.): 79-4465, 79-4477.
* (Immun.): 79-4652, 79-4653, 79-4657, 79-4664, 79-4666, 79-4668, 79-4676.
* (Path.): 79-4700.
* (Epid.-Biom.): 79-4739, 79-4742, 79-4757.

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- 79-4652 Tubulin and Actin Immunofluorescence as an Aid in Detecting Cell Transformation In Vitro.** (Eng) Brinkley, B. R. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 77030); Fuller, G. M. *Tex Rep Biol Med* 37: 26-44; 1978.

A brief assessment of cell culture bioassays for screening chemical carcinogens is provided, along with results of the use of tubulin and actin immunofluorescence as an aid in detecting cell transformation in vitro. Cells transformed in vitro by chemical or viral agents usually display a variety of distinct properties, including alterations in shape, cell-surface changes, loss of density-dependent growth control, loss of anchorage-dependent growth, and reduced serum requirements. In addition, increased mobility of cell-surface receptors and redistribution of the receptors within the plane of the cell membrane are a consistent property of many transformed cells. Recent studies have identified specific alterations in cytoskeletal elements, including cytoplasmic microtubules and microfilaments, that may account for several of the previously mentioned tumor cell properties. With the use of monospecific antibodies made against 6S bovine brain tubulin as immunofluorescent probes, an extensive cytoplasmic microtubule complex (CMTC) has been identified in a variety of nontransformed cells in vitro. The CMTC consists of a delicate network of fine fluorescent filaments that radiate out from a central focus within the cytoplasm toward the cell periphery. These filaments, believed to be individual microtubules, terminate near the plasma membrane. When most non-transformed cells are stained by antiactin immunofluorescence, an extensive array of parallel "stress fibers" or actin cables can be seen that span the length of the cell. Upon transformation by chemicals or viruses, actin cables disappear or become greatly diminished. Although the biological basis and extent of altered cytoskeletal patterns in transformed cells are not fully understood, tubulin and actin immunofluorescence may serve as useful aids in detecting transformants in many cell populations. However, the procedure should be used in conjunction with other standard transformation assays. (31 refs)

- 79-4653 Inhibition and Promotion of Growth of B77-Virus-induced Rat Tumor with KCl-solubilized Tumor Cell Components.** (Eng) Simkovic, D. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia); Chorvath, B.; Duraj, J.; Hlubinova, K. *Neoplasma* 25(6): 647-651; 1978.

The effect of different doses of KCl-solubilized components of avian sarcoma virus B77-induced rat tumor cells

(LWF B77) on the growth of syngeneic tumors was examined. Rats were immunized with a single dose of KCl-solubilized tumor cell material (TCM: 1 mg protein sc) 10 days prior to challenge with 10^7 viable LWF B77 cells. Other groups of rats were inoculated with five weekly doses of solubilized TCM (1 mg sc) and challenged with 5×10^6 LWF B77 cells 10 days after the last inoculation. A single dose of solubilized TCM inhibited the growth of syngeneic tumor cells. Repeated inoculations of TCM, however, significantly stimulated the growth of the tumor cells. These results indicate that the inhibition or promotion of the growth of syngeneic tumors in rats preimmunized with KCl-solubilized TCM is dose-dependent. Overloading an organism with neoantigens, whether tumor-associated or embryonic, may suppress the immune surveillance mechanisms and thus facilitate tumor growth. (9 refs)

- 79-4654 Antigen-specific Suppressive Factor Produced by a Transplantable I-J Bearing T-cell Hybridoma.** (Eng) Taniguchi, M. (Lab. Immunology, Sch. Medicine, Chiba Univ., Chiba, Japan); Saito, T.; Tada, T. *Nature* 278(5704): 555-558; 1979.

An I-J-bearing (I-J subregion of the mouse major histocompatibility complex) hybrid cell line (9F181a) was derived by fusion of the AKR mouse thymoma cell line BW5147 with the suppressor T cells of C57BL/6 mice. After intracutaneous inoculation, 9F181a cells grew as a solid tumor in (C57BL/6 x C3H) F_1 mice but not in the parental strains. A large quantity of ascites was produced when the cells were transplanted ip into the F_1 mice. The ascites contained I-J determinants and a strong antigen-specific suppressor activity, as did the 9F181a cells. The suppressor molecules secreted and extracted from 9F181a cells had an antigen-binding site and I-J-coded determinants. The mol wt of the hybridoma-derived suppressor molecule was between 42,000 and 68,000 daltons. The ability of the materials extracted from 9F181a cells to suppress the response mounted by spleen cells of the parental haplotypes and those of the F_1 hybrid indicated that I-Jb but not I-Jk is phenotypically expressed on 9F181a cells. (12 refs)

- 79-4655 Immunodepressive Effect of Polyamines.** (Rus) Umanskii, Iu. A. (Inst. Problem Oncology, Kiev, USSR); Iakimenko, L. V.; Berdinskikh, N. K.; Khomenko, A. K. *Dokl Akad Nauk SSSR* 245(5): 1272-1274; 1979.

The immunodepressive effect of polyamines was in-

vestigated in BALB/c mice who were given either a single injection of spermidine phosphate (SP: 50, 100 or 500 μ g) or repeated injections of SP (10 50- μ g doses or 10 50- μ g doses followed by 10 100- μ g doses). On the day after the last injection, mice were inoculated ip with test antigen (sheep RBC). On day 4 after immunization, the number of antibody-forming cells (AFC) in the spleen was determined by local hemolysis reaction. A single injection of 50 or 100 μ g SP decreased the number of AFC by 17% and 30%, respectively. Repeated administration of SP decreased the number of AFC by 41.5% and 45%, respectively. In view of the total doses administered with the repeated injections, these decreases were not that significant. Repeated administration of SP was associated with 11.25- and 15-fold increases in aminooxidase activity, which probably prevented a more drastic immunodepressive effect of SP on the mice. (8 refs)

79-4656 Teratocarcinomas Rarely Develop from Embryos Transplanted Into Athymic Mice. (Eng)

Solter, D. (Wistar Inst. Anatomy and Biology, 36th and Spruce St., Philadelphia, PA 19104); Damjanov, I. *Nature* 278(5704): 554-555; 1979.

The outgrowth of 7-day-old embryos derived from teratocarcinoma-permissive (C3H/HeJ) and teratocarcinoma-nonpermissive (C57BL/6J) mice was studied following transplantation of the embryos under the kidney capsules of normal syngeneic mice, athymic nude mice, and thymectomized irradiated, and bone marrow-reconstituted (TIR) mice. The proportion of teratocarcinomas was significantly higher in C3H/HeJ mice than in nude and TIR recipients, and all embryo-derived tumors in nude and TIR recipients were considerably smaller than those in C3H/HeJ mice. The results show that the removal or absence of the T-cell immune system in recipients of embryonic grafts apparently inhibits teratocarcinogenesis. Two hypotheses could explain these observations: an interaction between the transplanted embryo and the T-cell immune system might be necessary for teratocarcinogenesis; or the development of teratocarcinomas may be inhibited by natural killer cells and/or macrophages. The presence of lymphocytic infiltrates in the embryo-derived tumors in the nude and TIR mice supports the latter hypothesis. (7 refs)

79-4657 Nonspecific Killing of Tumor Cells by SV40 Tumor Antigen-Antibody Complex-induced

Spleen Cells. (Eng) Prather, S. O. (Sch. Life Sciences, Univ. Nebraska-Lincoln, 319A Manter Hall, Lincoln, NE 68588); Shillito, E.; Lausch, R. N. *J Reticuloendothel Soc* 25(3): 283-292; 1979.

An attempt was made to determine why PARA-7 [a simian virus 40 (SV40)-induced LSH hamster fibrosarcoma] an-

tigen (Ag)-excess immune complexes caused cytotoxicity in the presence of nonsensitized, nonadherent spleen cells from normal LSH and GS hamsters. PARA-7 tumor Ag neutralized the antibody-dependent cellular cytotoxicity (ADCC) activity of SV40 antiserum, as detected in a visual microcytotoxicity test. However, immune complexes prepared at elevated Ag concentrations induced cytotoxicity. This killing, max at an Ag concentration of 10 μ g/ml, was nonspecific and could not be attributed to Ag toxicity, complement activation, or the release of a soluble mediator. Nonspecific killing of target cells did not occur when the test was performed in the presence of trypan blue or if the effector cells were first passed through a nylon wool column. The addition of cells recovered from the column restored cytotoxicity. Specific ADCC was not affected by trypan blue or nylon wool filtration. The data suggest that the observed nonspecific cytotoxicity was due to immune complex activation of macrophages. (22 refs)

79-4658 Macrophage-dependent, NK-Cell-independent "Natural" Surveillance of Tumours in

Syngeneic Mice. (Eng) Chow, D. A. (Dept. Pediatrics and Immunology, Manitoba Inst. Cell Biology, Univ. Manitoba, Winnipeg, Manitoba R3E 0V9, Canada); Greene, M. I.; Greenberg, A. H. *Int J Cancer* 23(6): 788-797; 1979.

The "natural" T-independent rejection of syngeneic tumors was studied in DBA/2, CBA, AKR, and A/J inbred mice with the use of the spontaneous L5178Y lymphoma of DBA/2 mice and the P-815-X2 methylcholanthrene-induced mastocytoma of DBA/2 mice. No detectable DBA/2 natural killer (NK) cell activity was demonstrated against the syngeneic tumor lines studied, and these tumors were insensitive to NK cells from high-activity CBA mice. Increases in tumor frequency were consistently observed following ip administration of silica 24 hr before and simultaneously with sc tumor inoculation. Twice-weekly ip injections of fumed silica (100 μ g) significantly decreased the life spans of old and young AKR mice, most of which died with evidence of thymic tumor. The max increase in tumor frequency was observed following silica treatment that began 3 days before tumor inoculation. Reticuloendothelial stimulants such as *Mycobacterium butyricum* and proteose peptone decreased the tumor frequency following small tumor inocula, indicating that the effector mechanism could be stimulated. Soluble tumor antigen enhanced the tumor frequency in normal and immunodeficient mice, suggesting that the specific receptor molecule of the surveillance mechanism was not thymus-dependent. (53 refs)

79-4659 Immunogenic Properties of Some Spontaneous Gastrointestinal Tract Tumors in Mice. (Rus) Avdeev, G. I. (No affiliation given); Svet-

Moldavskaia, I. A.; Khatuntseva, N. I.; Turetskaia, R. L.; Kovaleva, L. P. *Probl Onkol (Sofia)* 6: 69-73; 1978.

The immunogenic properties of a transplantable murine adenocarcinoma of the large intestine were investigated. BALB/c mice were inoculated sc with 5×10^6 tumor cells; 6-7 days later, the tumors were dissected (control mice underwent sham dissections). On day 8 after immunization, the mice were inoculated sc with 1×10^5 to 6×10^6 tumor cells. In another experiment, mice were immunized with irradiated (15,000 rads) tumor cells (5×10^6 cells, sc); 8 days later, the mice were inoculated iv with $10-20 \times 10^4$ tumor cells. On day 20 after transplantation, the mice were sacrificed and the number of pulmonary metastases was determined. The results obtained in the first experiments were rather ambiguous: in 11/15 cases, tumor wt in the immunized mice was nonsignificantly smaller than that in animals who underwent sham surgery, but in 4/15 cases, tumor wt in the immunized mice was greater than that in control mice. In the second experiment, the number of pulmonary metastases in immunized mice was significantly lower than that in control mice, which indicates that these transplantable adenocarcinomas of the large intestine may possess weak antigenic properties. (no refs)

79-4660 Effect of Murine Antiserum Against Isologous Aggregated Immunoglobulins on Transplantation and Antitumor Immunity. (Rus) Kaulen, D. R. (N. F. Gamaleia Inst. Epidemiology and Microbiology, Moscow, USSR); Snegireva, A. E.; Khorobrykh, V. V.; Shevliagin, V. Ia.; Prigoda, O. S. *Biull Eksp Biol Med* 87(5): 438-441; 1979.

The effect of murine antisera against isologous aggregated immunoglobulins on transplantation immunity was studied in mice. CBA mice (H-2k) were given skin grafts from C57BL/6 mice (H-2b). During days 7-13 after transplantation, the recipients were inoculated ip with 0.1 ml of the antiserum (control mice were inoculated with normal murine serum: NMS). Rejection of the allotransplant in the controls occurred 10.9 days after transplantation, compared with 15.8 days in mice treated with the antiserum. These findings were indicative of the immunodepressive effect of the antiserum. Administration of the antiserum to BALB/c mice infected with Moloney sarcoma virus reduced the latent period of tumor development (14.6 days, compared with 19.0 days in mice treated with NMS) and increased the mortality of tumor-bearing animals (57.1%, compared with 0% in controls). In contrast, administration of the antiserum to BALB/c mice with Rauscher leukemia increased the life-span of the tumor-bearing animals (97.6 days, compared with 48.1 days in mice with NMS) and reduced their mortality rate (only 30% died within 40-60 days, compared to 100% of the mice treated with NMS). (8 refs)

79-4661 Induction of Rat Sarcomas in Rats Treated

with Antithymocyte Sera after Transplantation of Human Cancer Cells. (Eng) Huebner, R. J. (Lab. Cellular and Molecular Biology, NCI, NIH, Bethesda, MD 20014); Fish, D. C.; Djurickovic, D.; Trimmer, R. W.; Bare, A. L.; Bare, R. M.; Smith, G. T. *Proc Natl Acad Sci USA* 76(4): 1793-1794; 1979.

Transplantation of human cancer cells (RD rhabdomyosarcoma cells) that had had high (158) tissue culture passages into antithymocyte-treated F344 newborn rats induced rat sarcomas in the rats within 2 or 3 subcultures, whereas transplantation of human cancer cells (melanoma Hs294T, a lung carcinoma, and N-methyl-N'-nitro-N-nitrosoguanidine transformed TE85 cells) with low (5-33) passages in vitro did not cause overt induction of rat sarcomas until after 5-10 subtransplantations. Because oncornavirus activity was not detected in either the rat or human tumors, it is suggested that transforming sequences located on the human tumor cells may have been transferred to supporting rat reticulum cells in close contact with the human cancer cells. (5 refs)

79-4662 Genetics of F₁ Hybrid Susceptibility to Myeloma Grafts. (Eng) Pease, L. R. (Mammalian Genetics Center, Univ. Michigan, Ann Arbor, MI 48109); Foster, M. *Transplantation* 27(4): 270-272; 1979.

F₁ hybrids derived from reciprocal crosses of C57BL/10 and BALB/c (B10CF₁ and CB10F₁) mice are less susceptible to myeloma grafts than were the BALB/c parental hosts. An attempt was made to document which genetic contributions from the C57BL/10 resistant parents influence hybrid susceptibility by comparing the frequency of myeloma graft takes in F₁ hybrids derived from congenic strains containing the C57BL/10 background genome. Similar studies utilizing congenic strains of the A and DBA/2 backgrounds are also reported. Various inbred mouse strains and their descendant congenic lines were crossed with BALB/c mice to produce appropriate F₁ hybrids. Genetic inferences were drawn from a comparison of the frequencies of tumor graft takes among congenic combinations. Hybrids containing the C57BL/10 genetic background were less susceptible to myeloma transplants than were hybrids of other genotypes. Both H-2-associated and non-H-2-associated genetic factors played significant roles in determining host susceptibility to the transplants. The D end of the major histocompatibility complex did not play a predominant role in determining hybrid susceptibility in C57BL-derived F₁ hybrids. (8 refs)

79-4663 Decreased Hybrid Susceptibility to Murine Myeloma Grafts. (Eng) Pease, L. R. (Mammalian Genetics Center, Univ. Michigan, Ann Arbor, MI 48109); Foster, M. *Transplantation* 27(4): 265-269; 1979.

The behavior of BALB/c myeloma RPC-20 transplants in (C57BL/10 x BALB/c) F_1 hybrids and the parental BALB/c strain was investigated. The F_1 hybrids developed fewer tumors after a longer latent period than the parental strain (27% takes at 40 days, vs 27% takes at 13 days and 67% takes at 40 days in the parents). Also, the dose that resulted in a 50% tumor frequency was significantly higher in the hybrids than in the BALB/c mice. The hybrids were also significantly less susceptible to transplants of myelomas W3207 and MOPC-603. Male F_1 hybrids were significantly less susceptible than female hybrids, and both BALB/c and hybrid hosts were more susceptible following x-irradiation or treatment with cyclophosphamide. No evidence of a memory response was observed when survivors of one tumor challenge were rechallenged, and rabbit anti-BALB/c thymus serum had no significant effect on hybrid susceptibility to the myeloma grafts. The latter observations do not support an immune hypothesis. The parent-to-hybrid grafting scheme appears to be capable of detecting genetically determined host-mediated control of tumor grafts. (8 refs)

79-4664 Lymphoma Development in AKR:CBA/H-T6Crc Chimaeras Derived by Neonatal Injection of Spleen Cells. (Eng) Tuffrey, M. (Clinical Res. Centre, Harrow, England); Crewe, P.; Barnes, R. D. *Eur J Cancer* 15(4): 387-393; 1979.

Reciprocal neonatal injections of spleen cells into CBA/HT6Crc and AKR mice rendered these animals highly tolerant, as judged by the persistence of reciprocal skin grafts. Chimerism was demonstrated cytogenetically in several cases, but there was no AKR "takeover" as seen in previous studies with early embryo aggregation chimeras. Of the 22 CBA chimeras tested, AKR cells were found in only 5 animals and only in their spleens. Lymphoma incidence in AKR mice made tolerant to CBA was similar to that in normal AKR. In contrast to normal CBA mice who do not develop lymphomas, the CBA made tolerant to AKR died with lymphomas. However, the latent period was delayed and was similar to that in (CBA x AKR) F_1 hybrids. Murine leukemia virus (MuLV) p30 levels in the tolerant CBA were also elevated and comparable to those seen in AKR. Reciprocal thymic grafting, which, in some groups of mice, was accompanied by further injections of lymphoid cells from the donor strain, appeared to have no effect on lymphoma incidence. The CBA lymphomas were, therefore, attributed to MuLV present in the original spleen cell injections. These results differ from those of an early study in which no lymphomas developed in low leukemic C3H mice made tolerant of AKR cells during the neonatal period. (22 refs)

79-4665 Chemotactic Factor for Tumor Cells Derived from the C5a Fragment of Complement Com-

ponent C5. (Eng) Orr, W. (Dept. Pathology, Univ. Connecticut Health Center, Farmington, CT 06032); Phan, S. H.; Varani, J.; Ward, P. A.; Kreutzer, D. L.; Webster, R. O.; Henson, P. M. *Proc Natl Acad Sci USA* 76(4): 1986-1989; 1979.

The fifth component of complement (C5) serves as an important source of mediators that have locomotory (chemotactic) activity for WBC and tumor cells. C5a, a 11,200-dalton fragment derived from the NH_2 -terminal portion of the α chain of C5, is the major chemotactic peptide for WBC. Cleavage of C5a with trypsin generated a derivative peptide that was chemotactic for tumor cells (Walker carcinosarcoma). This fragment had an estimated mol wt of 6,000, as assessed by gel filtration, and it did not require the $COOH$ -terminal arginine of C5a, because equivalent amounts of chemotactic activity for tumor cells could be generated from des-Arg-C5a by digestion with trypsin. The C5a-derived chemotactic peptide for tumor cells demonstrated peak activity at approx 1 picomolar. These studies emphasize the key role of the C5a region of the C5 molecule in the generation of peptides that affect locomotory responses of cells. (15 refs)

79-4666 Hypocomplementemia Associated with Naturally Occurring Lymphosarcoma in Pet Cats. (Eng) Kobilinsky, L. (Sloan-Kettering Inst. Cancer Res., 1275 York Ave., New York, NY 10021); Hardy, W. D.; Day, N. K. *J Immunol* 122(6): 2139-2142; 1979.

The effect of disease on the serum complement system was studied in 80 pet cats. The cats were classified by indirect immunofluorescence and histologic diagnosis into four categories: normal, feline leukemia virus (FeLV)-infected; normal, noninfected; lymphosarcoma-FeLV infected; lymphosarcoma, no FeLV present. It was found that all viremic cats with lymphosarcoma were hypocomplementemic and that activation of the complement system had occurred via the classical pathway. Sera of cats with lymphosarcoma in the absence of FeLV had varying levels of total hemolytic complement (TCH_{50}) ranging from normal to hypocomplementemic. Approx 50% of the cats that were viremic but histologically and clinically free of disease had TCH_{50} levels within the normal range, and the remainder exhibited varying degrees of hypocomplementemia. A significant percentage of healthy cats that are persistently infected with FeLV and are hypocomplementemic may have an increased susceptibility to the development of lymphosarcoma. (27 refs)

79-4667 Interaction of Glycosyl Immunogens with Immunocyte Receptor Sites in the Synthesis of Anti-glycosyl Isoantibodies. (Eng) Diebold, G. J. (Dept. Biochemistry and Biophysics, Pennsylvania State Univ., University Park, PA 16802); Pazur, J. H.; Bundle, D. R. *ACS Symp Ser* (85): 80-90; 1979.

The nature of the anti-glycosyl antibodies (ab) induced in rabbits by immunogens with different carbohydrate immunodeterminant groups was studied. Among the bacterial antigens (ag) used in this research were: a diheteroglycan of glucose and galactose from the cell wall of *Streptococcus faecalis*; and two synthetic carbohydrate-protein conjugates, galactosyl bovine serum albumin (Gal-BSA) and lactosyl bovine serum (Lac-BSA). All of these ag's possessed the same types of terminal carbohydrate moieties (galactosyl or lactosyl units) which were the immunodeterminant groups. The methods used for the preparation of the ag's and their molecular structures are discussed. The anti-gal and anti-lac ab obtained from the anti-*S. faecalis* serum were of the IgG immunoglobulin type and all their components were glycoproteins. The individual proteins (isoantibodies) of these ab's were homogeneous. The three antisera and anti-glycosyl ab's isolated from the antisera were tested for cross-reactivity with the three immunogens and with BSA. The differences in the responses to the three immunogens suggest a role for cell surface topography and macromolecular conformation of the immunogen in directing the synthesis of ab. (36 refs)

- 79-4668 **The Genetic and Antigenic Basis for the IgA Antibody Response to Epstein-Barr Viral Capsid Antigen.** (Eng) Ng, M. H. (Dept. Microbiology, Univ. Hong Kong, Hong Kong); Ho, H. C.; Kwan, H. C. *IARC Sci Publ* 20: 449-458; 1978.

To study the relationship between the IgA antibody response to Epstein-Barr viral capsid antigen (VCA) and nasopharyngeal carcinoma (NPC), levels of IgA antibody to VCA (IgA anti-VCA) were determined in the sera of 126 NPC patients before radiation therapy, 10 patients at varying periods after therapy, 47 relapse-free survivors at up to 12 yr following therapy, and 57 healthy subjects and in the plasma of 133 apparently healthy family members of the NPC patients. IgA anti-VCA was detected in the sera of 96.03% of the NPC patients combined and in none of the sera of the healthy controls. The family members constituted an intermediate association group, with 21.05% of their sera being positive. These results suggest that the IgA antibody response to VCA might be genetically determined. Analysis of the distribution of individuals with IgA anti-VCA among siblings from sibships of different sizes showed that the number of individuals with IgA anti-VCA correlated with that expected, as if the IgA antibody response were determined by an autosomal recessive gene. The frequency of IgA anti-VCA was slightly higher in NPC patients with regional disease than in those with local disease or in survivors who had had regional disease before therapy. IgA anti-VCA persisted in all 10 NPC patients tested following therapy. A substantial rise in IgA antibody titer was observed concomitantly with the appearance of clinical NPC in one of the family members of an NPC patient. This assay system appears to be highly sensitive for

NPC, and it may indicate the existence of occult and subclinical residual tumors. (19 refs)

- 79-4669 **Association of IgA Multiple Myeloma with Pre-existing Disease.** (Eng) Schafer, A. I. (Dept. Medicine, Univ. Chicago Hosps. and Clinics, 950 E. 59th St., Chicago, IL 60637); Miller, J. B. *Br J Haematol* 41(1): 19-24; 1979.

A retrospective analysis was made of 153 patients with multiple myeloma (84 men, 69 women aged 35-86 yr, mean 61 yr) to evaluate the possible significance of antecedent disease. There was no significant preexisting disorder in 37% of the patients. The most common prior illnesses were peptic ulcer disease and gallbladder disease. Of the 12 patients who had prior biliary tract disease and for whom immunoelectrophoretic studies were available, 8 had IgA paraproteins. This figure is statistically higher ($p < 0.01$) than the 14.1% prevalence of IgA paraproteins in myeloma patients without biliary disease. Of 20 individuals with IgA myeloma, only 2 had no significant antecedent disease. The rest had primarily chronic biliary, peptic ulcer, or other gastrointestinal or respiratory tract inflammatory diseases. It is concluded that prior inflammatory gastrointestinal, pulmonary, and, particularly, biliary disease may be implicated in the pathogenesis of the IgA subset of multiple myeloma. (15 refs)

- 79-4670 **Immunoglobulins and Hyposensitization for Allergy.** (Eng) Keeling, M. M. (Clinical Lab. Service, Veterans Admin. Medical Center, 800 Zorn Ave., Louisville, KY 40213); Broghamer, W. L.; Miller, T.; White, A. F.; Esselmann, M. T. *Ann Allergy* 42(5): 319-322; 1979.

The serum immunoglobulins (Ig) of 313 patients who had been hyposensitized for allergy were compared with those of 138 nonallergic and 189 allergic individuals not subjected to this therapy in an attempt to determine if there is a relationship between protracted stimulation of the immune system and the induction of malignant immunocytopathies. The mean serum concentration of IgG, IgA, and IgM was 1023 mg/deciliter (dl), 182 mg/dl, and 160 mg/dl, respectively, for the hyposensitized allergic patients, compared with 1131, 196, and 174 mg/dl, respectively, for the control group. Values for the allergic non-treated patients fell in between the extremes. The differences in the mean IgG levels between the hyposensitized allergic patients and the control group were statistically significant. The depression of the serum IgG concentration in allergic hyposensitized persons was related to the length of the desensitization program and to whether it was continuous or interrupted. Interrupted hyposensitization produced serum IgG concentrations approaching values seen in the non-desensitized and nonallergic subjects. Although

there were some significant changes in the Ig's of those receiving hyposensitization, this study indicates that no malignancies of the immune system could be attributed to this therapy. (13 refs)

- 79-4671 A Novel Interaction Involving a Polypeptide Chain (P33) in Covalent Linkage with IgM on the Surface of a Burkitt Lymphoma Cell Line (Daudi).** (Eng) Singer, P. A. (Max-Planck-Institut für Biologie, Corrensstrasse 42, D-7400 Tübingen 1, W. Germany); Williamson, A. R. *Eur J Immunol* 9(3): 224-231; 1979.

The surface IgM of Daudi Burkitt's lymphoma cells was investigated by several techniques, including radioiodination, immunoprecipitation, gel electrophoresis, and isoelectric focusing. The surface IgM molecule was found to consist of μ chains and κ chains covalently linked, by disulfide bonding, to a novel polypeptide chain with an approx mol wt of 33,000 daltons. This novel polypeptide chain (P33) was similar to the heavy chain of $\text{J}\alpha$ antigen with respect to glycoprotein nature, apparent mol wt, isoelectric behavior, and cysteine content. The linkage of P33 to surface immunoglobulin has not been observed with any other human lymphoma or lymphoblastoid cell lines. (27 refs)

- 79-4672 Nuclear Transcripts of Mouse Heavy Chain Immunoglobulin Genes Contain Only the Expressed Class of C-Region Sequences.** (Eng) Marcu, K. B. (Dept. Biology, State Univ. New York Stony Brook, Stony Brook, NY); Schibler, U.; Perry, R. P. *Science* 204(4397): 1087-1088; 1979.

The ontogenic switch in expression of constant region (CH) genes during B-lymphocyte differentiation was investigated by examining the nuclear transcripts in cells producing immunoglobulins of one particular class for the presence of sequences specifying other classes. Nuclear precursors of heavy (H)-chain messenger RNA's (mRNA's) were detected by their specific hybridization to cloned complementary DNA sequences. The myeloma cell line MPC-11, which produces γ_2b chains, and plasmacytoma tumors J558 and PC3741, which produce α and μ H chains, were used as the source of nuclear RNA and cytoplasmic mRNA. When nuclear RNA from MPC-11 cells was fractionated on denaturing gels and hybridized with a labeled γ_2b probe, hybridization was detected by autoradiography. When the same nuclear RNA was incubated with the α or μ probe, no hybridization was detected. Thus, the nuclear precursors of the γ_2b -mRNA, which contain sequences coding for the γ_2b C-region, do not contain sequences coding for the α or μ C-region. Similar analyses indicated that the nuclear transcripts of α -mRNA contained only $C\alpha$ coding sequences and nuclear RNA from the μ -chain-producing tumors contained only $C\mu$ coding sequences. These results do not support the existence of

multiple C-region sequences in the nuclear precursors of either γ_2b -, α -, or μ -mRNA's. This indicates that the switch from one class of H chain to another during B-cell ontogeny does not occur by altered processing of a complex gene transcript. (13 refs)

- 79-4673 Comparative Evaluation of Macrophage Stimulation and Depression on Host Defense Against Neoplasia.** (Eng) Gilbert, K. M. (Tulane Univ., New Orleans, LA). *Diss Abstr Int [B]* 39(11): 5313-5314; 1979 (no refs)

- 79-4674 Detection of a Transformation-related Antigen in Chemically Induced Sarcomas and Other Transformed Cells of the Mouse.** (Eng) DeLeo, A. B. (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021); Jay, G.; Appella, E.; Dubois, G. C.; Law, L. W.; Old, L. J. *Proc Natl Acad Sci USA* 76(5): 2420-2424; 1979.

Antisera prepared against a BALB/c methylcholanthrene (MC)-induced sarcoma (Meth A) in syngeneic or compatible F₁ mice recognized a protein with an apparent mol wt of 53,000 in extracts of [³⁵S]methionine-labeled transformed BALB/c cells. This component, designated p53, was not detected in normal adult mouse fibroblasts, lymphoid cells, or hematopoietic cells or in mouse embryo cells or 3T3 cells. An extensive variety of antisera, including alloantisera and heterologous antisera directed against structural antigens of murine leukemia viruses, was tested for reactivity with p53; other than Meth A antisera, only comparably prepared antisera against another BALB/c MC-induced sarcoma, CMS4, had anti-p53 activity. All transformed mouse cells tested were found to express p53; they included chemically induced sarcoma cells, leukemia cells, spontaneously transformed fibroblasts, and cells transformed by simian virus 40 and murine sarcoma virus. The presence of p53 in tumors of no known viral etiology indicates coding by resident cellular genes; this does not exclude endogenous viruses as the source of coding sequences or the possibility that transforming viruses code directly for p53. (31 refs)

- 79-4675 HLA Frequencies in Cancer: A Third Study.** (Eng) Perdue, S. T. (Dept. Surgery, Sch. Medicine, Univ. California, Los Angeles, CA 90024); Terasaki, P. I.; Mickey, M. R. *IARC Sci Publ* 20: 263-269; 1978.

The frequencies of 25 HLA antigens in 526 Caucasian cancer patients [with acute lymphocytic leukemia (ALL), acute myeloid leukemia (AML), breast cancer, Hodgkin's disease, lung cancer, lymphoma, or ovarian cancer] were

compared with those in 629 healthy controls. Patients and controls were selected from Caucasian persons HLA-typed in one laboratory between September 1975 and February 1977. Haplotypes were compared for 711 patients (with ALL, AML, lymphoma, or breast, lung, or ovarian cancer) and 549 controls typed between September 1974 and December 1976. Frequency deviations were found in those with ALL, AML, breast cancer, lymphoma, and ovarian cancer, but only the increase in A29 in AML patients was statistically significant. Other associations were the elevation of AW24 in ALL and AML patients and of B27 in ALL patients. Significant haplotype differences were an increase of A3-B8 and an absence of A1-BW17 in ALL patients and an increase of ALL-B5 and A2-BW40 and an absence of A2-B5 in AML patients. This study confirms the finding that there are no strong associations between histocompatibility HLA-A and -B locus antigens and the types of cancers studied; however, some consistent results indicate that there may be some linkage between HLA and cancer. (9 refs)

79-4676 Antibody Blockade of Lysis by T Lymphocyte Effectors Generated Against Syngeneic SV40 Transformed Cells. (Eng) Gooding, L. R. (Dept. Microbiology and Immunology, Duke Univ. Medical Center, Durham, NC 27710). *J Immunol* 122(6): 2328-2336; 1979.

Antisera to the major histocompatibility antigens of the mouse (H-2), to H-2-associated molecules, and to simian virus 40 (SV40) associated surface antigens were used to probe the role of these molecules in T-lymphocyte recognition of syngeneic virus-transformed mouse cells. Alloantisera to H-2 antigens were highly effective in inhibiting lysis. Anti-H2 antibody blockage of lysis was haplotype-specific, even on transformed F₁ target cells. Inhibition occurred only when antibody was bound to the target cell. Antibody binding to the effector did not inhibit lysis. Inhibition required that antibody be bound to the H-2 molecule itself; antibody to molecules associated with H-2, such as β_2 -microglobulin, had no effect. Attempts to block lysis by using antisera to known virus-coded molecules were uniformly unsuccessful. These results are discussed in light of current controversy concerning the nature of the SV40-specific transplantation antigen. (50 refs)

79-4677 A Study of HLA Antigens and Haplotypes in a Population of Caucasians with Acute Non-lymphocytic Leukemia. (Eng) Daly, P. A. (Univ. Maryland Hosp., 22 S. Greene St., Baltimore, MD 21201); Simon, R.; Schiffer, C. A.; Aisner, J.; Terasaki, P. I.; Wiernik, P. H. *Leuk Res* 3(2): 75-82; 1979.

The histocompatibility locus antigen (HLA) types of 136 Caucasian patients with acute nonlymphocytic leukemia

(ANLL) were compared with those of a control group of 1,008 subjects who were matched racially, regionally, and temporally. Haplotypes were determined from family typing data for 101 of these patients. There was no increased prevalence of any A- or B-locus antigen in the ANLL patients compared with the controls. The distribution of antigens in the patients did not differ from that in their siblings, and the haplotype distribution did not differ from that in the controls. Long-term survival (>2 yr) was not associated with any particular HLA antigen or haplotype. The number of homozygotes in the study group was not increased. Therefore, in this group of patients, no association could be found between HLA antigens and the development or subsequent course of ANLL. (23 refs)

79-4678 Familial Clustering of Plasma Carcinoembryonic Antigen (CEA) in the Cancer Family Syndrome. (Eng) Lynch, H. T. (Dept. Preventive Medicine/Public Health, Creighton Univ. Sch. Medicine, Omaha, NE); Guirgis, H. A.; Harris, R. E.; Frichot, B. C.; Lynch, J.; Vandevoorde, J.; Lynch, P. *Scand J Immunol* 8 (Suppl. 8): 465-470; 1978.

Plasma carcinoembryonic antigen (CEA) levels were assayed in 246 relatives and 96 spouses of 6 kindreds manifesting the Cancer Family Syndrome and in 206 normal individuals (controls). CEA levels were highest in the cancer patients and their spouses (Class 1), with a linear decline in antigen levels occurring through unaffected first-degree relatives (Class 2) and unaffected more-distant relatives (Class 3). The difference in CEA levels between high-risk individuals (Classes 1 and 2) and low-risk individuals (Class 3 and controls) was significant in both smokers and nonsmokers. Five immunochemical markers (CEA, α -fetoprotein, and IgA, IgM, and IgG) were measured in 15 high-risk relatives from a pedigree with familial malignant melanoma. Eleven of the 15 had two or more abnormal values. The probability of selecting such a sample from the general population is $< 1 \times 10^{-10}$. This indicates that there is an association between the results of these tests, melanoma, and high risk for the latter. A model to explain the presumptive connubial CEA effect proposes that the high-risk relative is a carrier of the putative cancer gene, which causes derepression of the oncogene. This process directly or indirectly elevates CEA levels. The secondary effects of this process may induce a communicable factor capable of eliciting specific information, such as CEA production in the spouses. The CEA elevation in the spouses of high-risk relatives may or may not be related etiologically to cancer predisposition. A duration effect relevant to prolonged exposure to the alleged communicable agent may be of importance. (18 refs)

79-4679 Tumour Antigens and Alloantigens. I. Relation of a Rat Tumour-specific Antigen with

Normal Alloantigens of the Host Strain. (Eng) Bowen, J. G. (Cancer Res. Campaign Labs., Nottingham Univ., University Park, Nottingham NG7 2RD, England); Baldwin, R. W. *Int J Cancer* 23(6): 826-832; 1979.

The relationship of rat hepatoma D23 tumor-specific antigen with normal alloantigens of the host strain (WAB/Not) was studied. The D23 antigen was recognizable by alloantisera raised in KX/Not rats (which differ from WAB rats at the major histocompatibility locus) against antigens present on WAB spleen, lymph node, and liver, but not on RBC. This was an immunologically specific phenomenon. The cross-reactivity of hepatoma D23-specific antigen with normal antigens recognized by KX rats also extended to the immunologically distinct hepatomas D30 and D192a. (32 refs)

79-4680 Tumour Antigens and Alloantigens. II. Lack of Association of Rat Hepatoma-D23-specific Antigen with β_2 Microglobulin. (Eng) Bowen, J. G. (Cancer Res. Campaign Labs., Nottingham Univ., University Park, Nottingham NG7 2RD, England); Baldwin, R. W. *Int J Cancer* 23(6): 833-839; 1979.

The association, if any, between rat hepatoma D23-specific antigen and β_2 microglobulin (β_2 M) was studied using turkey anti-rat β_2 M antiserum. Membrane immunofluorescence reactions and complement-dependent antibody lysis indicated that pretreatment of hepatoma D23 cells with anti- β_2 M antiserum had no effect on the reactivity of syngeneic anti-hepatoma D23 antiserum. Furthermore, immune precipitates formed from soluble tumor extracts containing hepatoma D23-specific antigen with turkey anti-rat β_2 M failed to generate a tumor-specific antibody response in syngeneic rats, although a cross-reactive antiserum was produced following immunization of allogeneic rats with the immune precipitate. The data suggest that the hepatoma D23-specific antigen is a modified normal component of the cell membrane or a derepressed component not normally exposed on normal adult cells. (36 refs)

79-4681 Serologically Detectable Human Melanoma-associated Antigens Are Not Genetically Linked to HLA-A and B Antigens. (Eng) Curry, R. A. (Dept. Molecular Immunology, Scripps Clinic and Res. Foundation, 10666 N. Torrey Pines Road, La Jolla, CA 92037); Quaranta, V.; Pellegrino, M. A.; Ferrone, S. *J Immunol* 122(6): 2630-2632; 1979.

Expression of serologically detectable melanoma-associated antigens (MAA) and histocompatibility antigens (HLA-A and -B) antigens by hybrid clones derived from the fusion of cultured human melanoma cells with murine fibroblasts was studied. HLA-A and -B antigens

and MAA segregated independently on the hybrid cells, and clones expressing MAA but not HLA-A or -B antigens lacked human chromosome 6. The expression of HLA antigens on the hybrid cells was lower than that on the parental human cells. However, HLA antigens and MAA were both serologically active and immunogenic on the hybrids, as shown by the fact that they elicited xenoantibodies reactive with the heavy chain of the HLA-A,B antigenic complex and with MAA following injection into rabbits. The data indicate that serologically detectable MAA segregate independently from HLA antigens in the interspecific somatic cell hybrids, indicating that these two sets of antigens are coded by genes that are not linked. (12 refs)

79-4682 Accessory Cell Requirements for the Generation of Cytolytic T Lymphocytes. (Eng) Lutz, C. T. (Committee Immunology, Dept. Pathology, Univ. Chicago, Chicago, IL 60637); Fitch, F. W. *J Immunol* 122(6): 2598-2604; 1979.

A system that facilitates the study of cellular interactions leading to the generation of cytolytic T lymphocytes (CTL) was studied. In this system, cortisone-resistant (CR) thymocytes constituted an enriched source of responding T cells, including CTL precursors. The CR thymocytes did not respond to allogeneic tumor cells and showed no special susceptibility to tumor cell suppressive factors or for the generation of active suppressor cells in CR thymocyte-allogeneic tumor cell cultures. Cortisone treatment was not responsible for the differential response of CR thymocytes to allogeneic tumor cells or spleen cells. The CR thymocytes lacked non-T accessory cells, but peripheral T cells were shown to require non-T accessory cells for CTL generation. The accessory cells required by glass wool and nylon wool nonadherent spleen cells exhibited many properties of macrophages. (34 refs)

79-4683 Phenotypic and Physical Characteristics of the Lymphoid Cells Involved in the Immunity to Syngeneic UV-induced Tumors. (Eng) Daynes, R. A. (Dept. Pathology, Univ. Utah, Salt Lake City, UT 84132); Schmitt, M. K.; Roberts, L. K.; Spellman, C. W. *J Immunol* 122(6): 2458-2464; 1979.

Studies using female C3Hf/HeN mice were performed to determine whether the cells involved in the regulation of anti-UV tumor immunity are phenotypically distinct and possess different physical properties from the cells involved in immunity to other tumors. Suppressor T cells (Ts) were shown to be positive for immune response-associated antigens (Ia) and extremely radiosensitive, whereas the cytotoxic T cells were Ia negative and radioresistant. The in vivo administration of anti-Ia antiserum to mice after tumor challenge caused a marked reduction in tumor growth rates. Furthermore, exposure of tumor-bearing

mice to low-dose gamma radiation resulted in the cessation of tumor growth and, in many cases, complete tumor rejection. The data strongly suggest that UV-induced Ts cells play an extremely important role in the tumor susceptible state induced by chronic UV exposure. (25 refs)

79-4684 In Situ Lymphoid Cells of Mouse Mammary Tumors. IV. Comparison of Functional Activity of Lymphoid Cells Separated from Mammary Tumors to That of Spleen and Lymph Node Cells of Tumor-sensitized Mice. (Eng) Ruppert, B. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 70030); Blazar, B.; Medina, D.; Heppner, G. *J Immunol* 122(6): 2180-2183; 1979.

The ability of in situ lymphoid cells from C4 or D2 mammary adenocarcinomas to influence tumor growth was compared with that of lymphoid cells from the spleens (SC) and lymph nodes (LNC) of tumor-bearing mice (BALB/c-Crgl females) and mice that had undergone surgical tumor removal. At a ratio of 100 tumor lymphoid cells to 1 tumor cell, tumor cell survival was markedly stimulated compared with the effect observed with normal LNC. More pronounced results were seen at a ratio of 1,000:1. SC and LNC from tumor-bearing mice inhibited the growth of C4 cells in vitro on the day of surgical excision. At 4-7 days after surgery, the target tumor cells showed an increased survival. By 10 days, the cellular immune reactivity changed from a stimulatory to an inhibitory pattern, with max inhibition of tumor cell survival being seen on day 13 after surgery. There was a decrease in inhibition through day 17, and by day 24 there were no differences in survival between tumor cells treated with sensitized or control lymphoid cells. The identity of the lymphoid cell(s) responsible for the stimulation of tumor cell growth in vitro is not known. (12 refs)

79-4685 Cyclical Changes in Susceptibility of a Myeloma Tumor (LPC-1) to Immune Destruction. III. Periodic Production of a Cell Surface Glycoprotein and Changes in Reactivity with Cytotoxic T Cells and Anti-H-2d Sera. (Eng) Celis, E. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Chang, T. W.; Eisen, H. N. *J Immunol* 122(6): 2245-2250; 1979.

Previous studies showed that when LPC-1 myeloma cells were grown as ascites tumor cells in syngeneic (BALB/c) mice, the cells harvested after 2-4 days of growth ("early" cells) were susceptible to lysis by cytotoxic T lymphocytes (CTL) and highly reactive with anti-H-2d antisera. Cells harvested after 12-14 days of growth ("late" cells) were resistant to lysis by CTL and poorly reactive with anti-H-2d antisera. This study shows that exposure to trypsin, chymotrypsin, or subtilisin (but not to thrombin or

Staphylococcus A protease) promptly converts LPC-1 cells with the late phenotype into cells with the early phenotype. Comparison of radiolabeled cell-surface proteins by gel electrophoresis indicated that the late cells possess a prominent trypsin-sensitive, high-mol-wt (160,000 daltons) surface glycoprotein that is present in smaller amounts on the early LPC-1 cells. This glycoprotein (gp160) was not detectable in four other BALB/c tumors that do not undergo the early-late transition of LPC-1. That gp160 was produced by LPC-1 cells, rather than adsorbed by these cells as they grow in vivo, was evident from the presence of an indistinguishable metabolically labeled glycoprotein on cultured LPC-1 cells. (18 refs)

79-4686 Lymphocyte Sensitization in Sheep Inoculated with Extracts of Spontaneously Occurring Malignant Lymphomas. (Eng) Johnstone, A. C. (Dept. Veterinary Pathology and Public Health, Massey Univ., Palmerston North, New Zealand); Moriarty, K. M.; Manktelow, B. W. *Aust J Exp Biol Med Sci* 57(part 1): 87-93; 1979.

An indirect macrophage migration inhibition assay was used to determine the presence of lymphocytes sensitized to tumor antigens in extracts derived from ovine lymphomas in sheep that had been inoculated with disrupted tumor cell material. The assay showed that the blood lymphocytes of 15/27 sheep inoculated in utero or at birth with disrupted cells from ovine malignant lymphomas responded in culture to antigens derived from three other lymphomas. A close association was seen between animals whose lymphocytes were antigen-sensitive and those that developed lymphocytosis. This association and the specificity of reaction between the various tumor antigens employed in the tests was interpreted as further evidence of a viral etiology for ovine malignant lymphoma. (21 refs)

79-4687 Simultaneous Occurrence of Immunoglobulin-dependent and Immunoglobulin-independent Mechanisms in Natural Cytotoxicity of Human Lymphocytes. (Eng) Pape, G. R. (Medical Clinic II, Grosshadern, Univ. Munich, 8000 Munich, W. Germany); Troye, M.; Axelsson, B.; Perlmann, P. *J Immunol* 122(6): 2251-2260; 1979.

A ^{51}Cr -release assay in which fragments of rabbit or goat antibodies to human IgG (a-Ig), aggregated IgG, or protein A from *Staphylococcus aureus* (SpA) were added to the incubation mixture was used to elucidate the mechanisms of lymphocyte-mediated natural cytotoxicity in humans. All fragments inhibited natural cytotoxicity to a variety of target tumor cells, including those from the K562 myeloid cell line. When lymphocytes or target cells were pretreated with inhibitors [Fab and F(ab')₂ preparations], no inhibition was obtained. Depletion of B cells from the lymph

phocyte preparations did not consistently decrease their cytotoxicity, which was as strongly inhibited by the a-Ig fragments (as in the presence of B cells). Depletion of lymphocytes with Fc receptors (FcR⁺) abolished the natural cytotoxicity, whereas depletion of FcR⁺ target cells (K562) had no effect. Addition of aggregated IgG caused a significant but incomplete inhibition of natural cytotoxicity. SpA in a wide dose range either increased or did not affect natural cytotoxicity and, at best, only weakly inhibited antibody-dependent cellular cytotoxicity in the presence of anti-target cell antibodies. The results suggest that a significant part of the natural cytotoxicity studied reflects killer cell reactions and that part of it is immunoglobulin-independent. (62 refs)

79-4688 Cutaneous T-Cell Lymphoma: Neoplasm of T Cells with Helper Activity. (Eng) Berger, C. L. (Dept. Dermatology, Columbia Univ., 630 W. 168th St., New York, NY 10032); Warburton, D.; Raafat, J.; LoGerfo, P.; Edelson, R. L. *Blood* 53(4): 642-651; 1979.

The helper activities [ability to promote immunoglobulin (Ig) synthesis by normal human B lymphocytes] of neoplastic T lymphocytes from three patients with aleukemic cutaneous T-cell lymphoma were studied. Homogeneous populations of neoplastic T cells were isolated from involved lymph nodes, and in two of three instances were shown to be of monoclonal origin by karyotypic analysis. The neoplastic cells of two patients frequently had receptors for sheep RBC and the third component of complement. Addition of control T-cell fractions to control B-cell fractions increased the number of Ig-producing cells from 8 to 34, whereas addition of T cells from the three patients increased the number of Ig-producing cells to a mean of 125 (range, 80-217), demonstrating exceptional helper activity. The peripheral blood T-cell-enriched fraction from a patient with Sezary syndrome increased the number of Ig-producing cells to 82. Lymphocytes from one lymphoma patient showed a proliferative response to phytohemagglutinin (PHA), but those of the other two patients did not respond to PHA, pokeweed mitogen (PM), or concanavalin A (Con A). The peripheral blood mononuclear WBC of the patient with Sezary syndrome responded to PM, but not to PHA and Con A. The data support a relationship between Sezary syndrome and aleukemic cutaneous T-cell lymphoma and suggest that malignant helper T cells have a distinct affinity for the skin. (22 refs)

79-4689 Studies on Experimental Pulmonary Granulomas. I. Detection of Lymphokines in Granulomatous Lesions. (Eng) Masih, N. (Dept. Pathology, Univ. Connecticut Health Center, Farmington,

CT 06032); Majeska, J.; Toshida, Y. *Am J Pathol* 95(2): 391-406; 1979.

Several experimental models of pulmonary granuloma formation were investigated. Granulomas were induced by injecting (iv) BCG or complete Freund's adjuvant (CFA) into Hartley guinea pigs immunized with BCG or CFA and by injecting (iv) agarose beads coated with dinitrophenylated bovine serum albumin (DNP-BSA) into animals immunized with DNP-BSA. Aqueous extracts of tissue obtained from the lungs at various stages of granuloma formation were examined for macrophage migration-inhibition factor (MIF) activity. The levels of this activity were high during the early stages of the granulomatous reaction. In contrast, MIF activity could be detected only rarely in granulomatous spleens and it was not found in granulomatous livers. Chemotactic factor activity and mitogenic factor activity were only sporadically detectable. The MIF activity was associated with fractions showing chemical heterogeneity. One fraction was physicochemically indistinguishable from conventional lymphocyte-derived MIF (mol wt 67,000), and the other was a substance with a high mol wt (>100,000). These results demonstrate the presence of biologically active mediators (lymphokines) in immune granulomas. These lymphokines may be related to early events involved in the induction or enhancement of granulomatous reactions. (21 refs)

79-4690 Hb F Production in Endogenous Colonies of Polycythemia Vera. (Eng) Papayannopoulou, T. (Div. Hematology, Dept. Medicine, Univ. Washington, Seattle, WA 98195); Buckley, J.; Nakamoto, B.; Kurachi, S.; Nute, P. E.; Stamatoyannopoulos, G. *Blood* 53(3): 446-454; 1979.

Fetal Hb production by circulating progenitors from 11 patients with polycythemia vera (PV) was studied. Erythroid colonies were formed in the absence of erythropoietin (EP) in cell cultures from seven subjects, the plating efficiencies ranging from 16 to 802 colonies/10⁵ inoculated mononuclear cells and individual colony sizes ranging from 8 to 400 cells. Most of the colonies appeared scattered. Addition of EP increased the number and size of burst-forming unit-erythroid (BFU-E)-derived colonies. The EP response curve of the circulating PV precursors differed strikingly from that of normal controls. Among the hemoglobinized endogenous colonies, frequencies of colonies positive for Hb F or Hb F + Hb A ranged from 27% to 53%. Supporting evidence for elevated Hb F production was obtained by studies of globin synthesis. Synthesis of γ chains in endogenous colonies accounted for 13%-42% of the total non- α chains. When γ -chain production at increasing levels of exogenously added EP was measured, a decrease in the proportion of γ chains was noted in 3/4 experiments. The data provide evidence that a single pluripotent stem cell can have committed progeny that differ in their production of Hb F. (15 refs)

79-4691 Monocyte Production in Hodgkin's Disease and Non-Hodgkin's Lymphoma. (Eng) Meuret, G. (Medizinische Klinik, St. Elisabethen-Krankenhaus, D-7980 Ravensburg, W. Germany); Schmitt, E.; Tseleni, S.; Widmer, M. *Blut* 37(4): 193-200; 1978.

To gain further insight into tumor-induced variations of monocyte kinetics, monocytopoiesis was investigated in patients with untreated Hodgkin's disease, Hodgkin's disease in long-term complete remission, and untreated non-Hodgkin's lymphoma of the lymphosarcoma and reticulosarcoma types. Untreated Hodgkin's disease was found to be associated with a rise in medullary monocyte production that returned to normal during long-term complete remissions. In contrast, monocyte production was increased in only 5/14 patients with lymphosarcoma and reticulosarcoma, normal in 3, and reduced in 6. In neither of these lymphomas was any relation between monocyte production and stage or histology of the disease detectable. (26 refs)

79-4692 Growth Characteristics of a Human Small Cell Carcinoma of the Lung in Athymic, Nude Mice (Meeting Abstract). (Eng) Meck, R. A. (State of Florida Comprehensive Cancer Center, Univ. Miami Sch. Medicine, Miami, FL 33136); Tejada, F.; Leung, I.; Zubrod, C. G. *Proc Am Assoc Cancer Res* 20: 254; 1979 (no refs)

See also

- *(Rev.): 79-4233, 79-4238, 79-4279, 79-4282, 79-4284, 79-4285, 79-4286, 79-4287, 79-4288, 79-4312.
- *(Chem.): 79-4327, 79-4359, 79-4361, 79-4376, 79-4391, 79-4400, 79-4402, 79-4411, 79-4454, 79-4466.
- *(Phys.): 79-4532, 79-4537.
- *(Viral): 79-4570, 79-4587, 79-4588, 79-4589, 79-4617, 79-4623, 79-4624, 79-4626, 79-4632, 79-4634.
- *(Path.): 79-4698, 79-4712.
- *(Epid.-Biom.): 79-4755, 79-4776.

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79-4693 Mechanism of Cancer Invasion (Meeting Abstract). (Eng) DiStefano, J. F. (Veterans Admin. Hosp., Northport, NY); Zucker, S.; Lysik, R. M. *Clin Res* 26(4): 633A; 1978 (no refs)

79-4694 Trisomy 13 in Bone Marrow Cells in Acute Myelocytic Leukemia and Myelofibrosis. (Eng) Hsu, L. Y. (Dept. Pediatrics, New York Sch. Medicine, 550 First Ave., New York, NY 10016); Greenberg, M. L.; Kohan, S.; Wittman, R. *Clin Genet* 15(4): 327-331; 1979.

Trisomy 13 was identified in three patients with hematologic malignancies involving hematopoietic stem cells. Direct preparation of bone marrow chromosomes for karyotypic analysis was performed for 570 patients whose diseases were thought to involve the pluripotent stem cell and its progeny. Trisomies were noted in 25 of these patients. Three of the 25 patients had trisomy 13: a 75-yr-old man with acute myelocytic leukemia, a 64-yr-old woman with agnogenic myelofibrosis and myeloid metaplasia, and a 68-yr-old woman with Philadelphia chromosome-negative chronic myelogenous leukemia. In the first and third patients, 100% of the cells had trisomy 13; in the second patient, 10% of the cells had trisomy 13. The probability of finding three such patients in this case material was calculated to be 0.05-0.08, suggesting that trisomy 13 may be another nonrandom chromosomal aberration associated with malignancies of hematopoietic pluripotent stem cells. (18 refs)

79-4695 Hematologic and Cytologic Characterization of 8/21 Translocation Acute Granulocytic Leukemia. (Eng) Trujillo, J. M. (Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bertner Drive, Houston, TX 77030); Cork, A.; Ahearn, M. J.; Youness, E. L.; McCredie, K. B. *Blood* 53(4): 695-706; 1979.

Chromosomes studies were made of bone marrow and peripheral blood samples from 32 acute granulocytic leukemia (AGL) patients. In addition, Giemsa-banding studies were made of samples from 15/32 patients. The 32 patients exhibited similar chromosome alterations that involved specifically chromosomes 8 and 21. The Giemsa-banding studies clearly identified a 8/21 translocation. The Y chromosome was missing in 8/19 men, whereas one of the two X chromosomes was absent in 2/13 women. Dur-

ing complete remission (16 patients), the 8/21 translocation clone was absent and diploid cells were present. During relapse, the 8/21 translocation clone always reappeared. Common morphologic features of all 32 samples included Auer rods, peroxidase-positive granules, and some degree of differentiation. Ultrastructurally, the leukemic cells consistently demonstrated a more differentiated cytoplasm than a nucleus. A high frequency of nuclear blebs was present in the immature myeloid cells. These 32 patients responded to therapy better than other adult AGL patients. The 8/21 translocation patients represent approx 7.3% of all AGL's and 16.6% of all aneuploid AGL's. The fact that all these patients present common cytologic and clinical features supports categorization of this entity as a definite AGL subgroup. (44 refs)

79-4696 Emergence of a Cell Line with Extreme Hypodiploidy in Blast Crisis of Chronic Myelocytic Leukemia. (Eng) Como, R. M. (Dept. Medical Genetics, UCLA Sch. Medicine, Los Angeles, CA 90024); Graze, P. R. *Blood* 53(4): 707-711; 1979.

A patient with Philadelphia chromosome (Ph')-positive chronic myelocytic leukemia (CML) was found to have an extensive chromosome rearrangement in addition to hypodiploidy. Bone marrow and peripheral blood samples were studied by standard techniques and by trypsin-Giemsa chromosome banding. At blast crisis, a modal chromosome number of 35 was found. The modal karyotype was 35, XY, -3, -4, -5, -7, -9, -11, -12, -13, -15, -16, -17, -19, -20, -22, +t(9;22)(q34;q11), +Mar₁, +Mar₂, +Mar₃. Giemsa banding demonstrated identical marker chromosomes in each of five cells examined. Mar₁ appeared to be a chromosome 12 with the deleted short arms replaced by the short arms and satellites of an acrocentric chromosome. Mar₂ had the appearance of chromosome 19, with a partial deletion of the long arm and the addition of two distinct bands. Mar₃ also had a distinct banding pattern, but the derivative chromosome(s) was not identified. The cells exhibited the morphologic characteristics of myeloid and lymphoid blast types. The patient lacked chromosomes 7, 16, and 17. All leukemic cells still carried the Ph' chromosome. At present, associations between specific chromosome changes and neoplastic growth cannot be made. The karyotypic variability of CML may reflect a particular susceptibility to abnormal mitosis. Extensive chromosome loss may be compatible with the development of a selective growth advantage of neoplastic cells in blast crisis over the growth of competing hematopoietic cells. (21 refs)

79-4697 Chromosome 8 Abnormalities as Components of Neoplastic and Hematologic Disorders. (Eng) Riccardi, V. M. (Kleberg Genetics Center, Baylor Coll. Medicine, Texas Medical Center, Houston, TX 77030); Forgason, J. *Clin Genet* 15(4): 317-326; 1979.

A review of the literature revealed the occurrence of 277 cases of chromosome 8 abnormality as of July 1977; these abnormalities comprised congenital aneuploidy (74 cases), congenital rearrangements (38), acquired aneuploidy (130), and acquired rearrangements (35). There were 170 cases of neoplastic and hematologic disorders associated with congenital aneuploidy, two with congenital rearrangements, and the rest with acquired aberrations. Among the trisomy 8 patients, 67/105 had chronic or acute myelogenous leukemia. Solid tumors represented in this series include meningiomas, nephroblastoma, and colorectal tumors. Two ovarian adenocarcinomas showed mosaic nullisomy 8. The results indicate that there is a definite, although nonspecific, correlation between congenital or acquired chromosome 8 abnormalities and the development of certain types of neoplasia. However, no chromosome abnormality is either specific, sufficient, or necessary for the full natural evolution of a neoplasm. It is suggested that a chromosome abnormality ordinarily develops during or after malignant transformation, with the net effect of enhancing the further evolution and development of the neoplasm. (98 refs)

79-4698 Malignant Progression of Angioimmunoblastic Lymphadenopathy. (Eng) Bamberg, M. (Dept. Radiotherapy, West German Tumor Center, Univ. Essen, Hufelandstrasse 55, D-4300 Essen 1, W. Germany); Donhuijsen, K.; Hoher, P. G.; Holfeld, H.; Hossfeld, D. K. *J Cancer Res Clin Oncol* 93(3): 255-263; 1979.

The progression of angioimmunoblastic lymphadenopathy with dysproteinemia (AILD) to malignant lymphoma within 1 yr after diagnosis is reported. The patient, a 79-yr-old woman, presented initially with enlarged bilateral cervical lymph nodes and splenomegaly. Three biopsy specimens from cervical and inguinal lymph nodes and one tonsil, obtained at intervals of several months, showed an increasing destruction of the tissue architecture and the development of histological characteristics for a lymphoid neoplasm. At autopsy, this neoplasm was confirmed as a malignant non-Hodgkin's lymphoma. The demonstration of a chromosomally abnormal clone of lymph node cells and the laboratory findings agreed with the histological changes and the sequential clinical deterioration. Initially, a symptom-free interval of 8 mo was achieved with prednisone therapy. However, this treatment failed after the malignant transformation had become evident. (29 refs)

79-4699 A 14q+ Chromosome in a B-Cell Acute Lymphocytic Leukemia and in a Leukemic Non-

endemic Burkitt Lymphoma. (Eng) Slater, R. M. (Lab. Human Genetics, Univ. Amsterdam, Amsterdam, Netherlands); Philip, P.; Badsberg, E.; Behrendt, H.; Hansen, N. E.; van Heerde, P. *Int J Cancer* 23(5): 639-647; 1979.

The results of cytogenetic studies of cells from a Burkitt's type B-cell acute lymphocytic leukemia (ALL) and a nonendemic Burkitt's lymphoma presenting as ALL are reported. In both patients, a marker chromosome 14q+ was found that was morphologically identical to those reported for endemic and non-endemic Burkitt's lymphoma and a few other B-cell malignancies. However, the origin of the translocated segment could not be identified in either case. In addition, both patients had a 13q+ chromosome with a breakpoint in the 13q3 region but involving different material of unknown origin. Other marker chromosomes in the B-cell ALL included rearrangements of chromosome arms 1q and 6q. Serial studies showed that cells with a partial duplication of the long arm of chromosome 1, in addition to the 14q+ chromosome, were important in the karyotypic evolution of the malignant cell population. In this patient, the 14q+ chromosome had a brightly fluorescing satellite region, indicating the probable monoclonal development of the leukemic cell population. From this and other reports, it appears that the B-cell type of ALL is characterized by a 14q+ chromosome. Because of the pathological and cytogenetic similarities between certain types of B-cell ALL and Burkitt's lymphoma, they may be different manifestations of the same disorder. (48 refs)

79-4700 Burkitt's Lymphoma--"An Autopsy Report with Review of the Literature". (Eng) Sharma, S. C. (Dept. Pathology, All India Inst. Medical Sciences, New Delhi 110 016, India); Choudhry, V. P.; Verma, K. *Indian Pediatr* 16(1): 71-75; 1979.

The occurrence of Burkitt's lymphoma in a 10-yr-old Indian boy is described. On hospitalization, a clinical diagnosis of osteomyelitis of the left tibia with generalized septicemia and myocarditis with congestive heart failure was made. Hypochromic microcytic anemia and myeloid hyperplasia were demonstrated, and an x-ray film of the left tibia showed lytic lesions in the upper two-thirds of the diaphysis. The patient died 1 mo later. At autopsy, the heart, pericardium, pancreas, and both kidneys were almost completely replaced by tumor, and microscopic foci of tumor were seen in the spleen, both adrenals, thyroids, stomach, ileum, testes, lungs, and CNS. The left tibia also showed extensive tumor infiltration. A diagnosis of Burkitt's lymphoma with extensive visceral involvement was made based on the histologic and cytochemical characteristics. The jaw bone was not involved in this case or in 8/24 previous cases from India. (28 refs)

79-4701 A Family with Multiple Endocrine Adenomatosis (MEA Type I) and Some Additional Peculiarities. α -1-Antitrypsin Deficiency (Twice), Polyp of Small Intestine with Heterotopic Gastric Mucosa (Once), Medullary Thyroid Carcinoma (Once). (Ger) Eberle, F. (Medizinische Universitätsklinik, Mannkopfstrasse 1, D-3550 Marburg/Lahn, W. Germany); Assmus, C.; Martini, G. A. *Klin Wochenschr* 57(10): 499-509; 1979.

Three generations (37 members) of a family with multiple endocrine adenomatosis type I (MEA-I, Wermer's syndrome) were investigated clinically, biochemically, and histologically. Twenty members had diseases or findings that were characteristic of MEA-I. One patient had a chromophobic pituitary adenoma, adenomas in the parathyroid glands as well as in the islets of Langerhans, a nodular colloid goiter, and bilateral nodular hyperplasia of the adrenal cortex. This is the complete picture of MEA-I. In addition, he had a medullary thyroid carcinoma. Of the 14 members who presented with symptoms and signs of primary hyperparathyroidism (pHPT), 5 had kidney stones. The diagnosis of pHPT was verified histologically in three patients, from resected parathyroid glands. Increased serum Ca and decreased P levels were the most important signs in detecting asymptomatic carriers of the gene. Six patients had stomach and/or duodenal ulcers: five exhibited a proved or probable pHPT, and only one also had a gastrinoma and adenomatosis of the islets of Langerhans. Three patients had pancreatitis, of whom two also had pHPT. A nontoxic goiter was present in two patients, a hyperthyroid goiter in one; the plasma cortisol level was elevated in two members. Similarly, two patients presented with lipomas in the sc adipose tissue. The discovery of an α -antitrypsin deficiency (ZZ phenotype) in at least two patients with pHPT suggests that there is a genetic connection between the defective Z allele and MEA-I. As far as is known, this is the first time that a bleeding polyp in the proximal jejunum with heterotopic gastric mucosa (fundic and pyloric glands) has been described in this syndrome. This is considered to be a developmental aberration of the entoderm. The medullary thyroid carcinoma in one patient shows that the line between MEA-I and MEA-II might not always be as strict as originally supposed. (60 refs)

79-4702 Adenomatous and Carcinomatous Changes Within Hyperplastic Colonic Epithelium. (Eng) Cooper, H. S. (Dept. Surgical Pathology, Thomas Jefferson Univ. Hosp., Rm. 7608, 11th and Walnut St., Philadelphia, PA 19107); Patchefsky, A. S.; Marks, G. *Dis Colon Rectum* 22(3): 152-156; 1979.

The case of a 67 yr-old white woman with numerous hyperplastic colonic polyps in which adenomatous changes occurred and adenocarcinoma developed is reported. Colonoscopic examination and biopsy revealed a papillary car-

cinoma, and the patient underwent resection of the ascending and transverse colon. The resection specimen showed a small lesion with numerous hyperplastic polyps adjacent to and continuous with it. Microscopic examination revealed a papillary adenocarcinoma arising within a field of hyperplastic colonic epithelium. In the adjacent hyperplastic epithelium, there were also numerous small islands of adenomatous epithelium. (10 refs)

79-4703 Partial Triplication and Deletion of 13q: Study of a Family Presenting with Bilateral Retinoblastomas. (Eng) Riccardi, V. M. (Kleberg Genetics Center, Baylor Coll. Medicine, Texas Medical Center, Houston, TX 77030); Hittner, H. M.; Francke, U.; Pippin, S.; Holmquist, G. P.; Kretzer, F. L.; Ferrell, R. *Clin Genet* 15(4): 332-345; 1979.

The pathogenetic influences of selective deletion and triplication of chromosome 13 derived from a familial 12;13 insertional translocation were compared in two siblings. In the proband (a boy who died at 111 days of age), a heritable chromosomal basis for his bilateral retinoblastomas was established [46,XY,del(13)(pter--q12.5::q22.1--qter)mat]. The tumor from the enucleated left eye had some histologic and ultrastructural features similar to those described in retinoblastomas from patients without chromosome anomalies. This tumor also demonstrated only incipient photoreceptorlike differentiation and limited calcification. Other features indicated a widespread retinal developmental defect, presumably due to the del(13q). In the proband's sister, in whom chromosome studies were performed at age 18 mo, the relatively modest effects of triplication of the midportions of 13q were demonstrated [46,XX,ins(12;13)(12pter--12p11.2::13q22.1--13q12.5::12p11.2-12qter)mat]. She was originally thought to be normal, but extensive evaluation indicated mild to moderate developmental delay. The mother of the children and her mother were balanced carriers of a 12;13 insertional translocation. There were no detectable changes in the late-labeling pattern, either within the translocated segment or on either side of the break points in the ins(12) and del(13) chromosomes. A new term and concept is presented to describe a mechanism that may account for expression for a chromosome rearrangement. The term is the haplicon (a single representation), and the concept is that of a contiguous array of genes that, when represented singly when double representation is usual, accounts for a more or less specific syndrome or complex of features. (61 refs)

79-4704 Familial Cardiac Myxoma: Emphasis on Unusual Clinical Manifestations. (Eng) Powers, J. C. (Div. Cardiology, Dept. Medicine, Genesee Hosp., 224 Alexander St., Rochester, NY 14607); Falkoff, M.; Heinle, R. A.; Nanda, N. C.; Ong, L. S.; Weiner, R.

S.; Barold, S. S. *J Thorac Cardiovasc Surg* 77(5): 782-788; 1979.

Case histories describing the familial occurrence of right-sided cardiac myxoma in a father and daughter are presented. The daughter (19 yr old) presented with weakness, headache, general malaise, and intermittent fever. The initial diagnosis was valvular subacute bacterial endocarditis. She was treated with antibiotics and rapidly improved. However, 5 mo later, a repeat echocardiogram showed a tumor in the outflow tract of the right ventricle. The tumor was resected and identified as a myxoma. The father (48 yr old) presented within 8 mo of his daughter with fatigue, malaise, sore throat, and severe pleuritic chest pain associated with upper abdominal pain. Echocardiograms revealed a large mass in the right atrium and mitral valve prolapse. A large fibrogelatinous mass attached to an atrial septal defect was removed from the right atrium, and a second tumor was excised from the inferior vena cava. Both tumors were identified as myxomas. There are four previous reports of familial myxoma, which is probably transmitted genetically. The temporal proximity of symptoms in the father and daughter raises the possibility of environmental etiological factors, such as viral infection. (29 refs)

79-4705 **Convolved Secretory Material in Thyroid Follicular Epithelial Tumors.** (Eng) Marcus, P. B. (Dept. Pathology, Baylor Univ. Medical Center, 3500 Gaston Ave., Dallas, TX 75246); Martin, J. H.; Lieberman, Z. H. *Am J Surg Path* 3(3): 279-281; 1979.

A 53-yr-old man presented with progressive pain in the left groin. Biopsy revealed a metastatic adenocarcinoma in the superior part of the left pubic ramus, but the primary tumor site was not identified. Despite radiotherapy, bone destruction progressed and a left hindquarter amputation was performed. The tumor was identified as being of the thyroid follicular epithelial type. A technetium thyroid scan revealed a cold nodule in the upper pole of the left lobe of the thyroid gland, and a thyroidectomy confirmed the presence of a primary thyroid carcinoma with features similar to those of the metastatic bone tumor. Mitochondrion-rich oncocytic-type tumor cells formed microvillus-lined lumina in the skeletal metastases, and convoluted secretory material was observed between cells in some areas and in membrane-bound spaces in others. The convoluted material appeared better defined in the metastatic than in the primary tumor samples. The material may have value as a marker for thyroid follicular epithelium in the typing of tumors by electron microscopy. (6 refs)

79-4706 **Bowen Carcinoma Following Many Years' Use of a "Radium Cushion".** (Ger) Grassel,

R. (Dermatologische Klinik und Poliklinik der Universität, Hartmanstrasse 14, D-8520 Erlangen, W. Germany); Hornstein, O. P. *Munch Med Wochenschr* 121(22): 753-756; 1979.

Precancerous changes of the abdominal skin, misinterpreted as eczema, were found in a 73-yr-old woman who had used a radium cushion for at least 10 yr. These cushions had been available commercially until 1960. Skin carcinoma was finally diagnosed 7 yr after the onset of the first-skin lesions. (24 refs)

79-4707 **Carcinoma Arising in Eccrine Spiradenoma.** (Eng) Evans, H. L. (Mayo Clinic, Rochester, MN 55901); Su, W. P.; Smith, J. L.; Winkelmann, R. K. *Cancer* 43(5): 1881-1884; 1979.

Two cases of eccrine spiradenoma (ES) in which carcinomatous transformation occurred are reported. A long-standing nodule on the interscapular area of a 26-yr-old white woman was excised after it became enlarged and tender. Microscopically, the lesion contained two distinct components: typical lobulated benign ES and invasive undifferentiated carcinoma. In one lobule, a direct transition of ES to carcinoma was observed. There was no evidence of squamous differentiation or gland formation in the malignant portion of the tumor. The patient showed no evidence of local recurrence or distant metastasis. A long-standing (29 yr) nodule on the knee of a 67-yr-old white woman was excised after it changed to a reddish purple color. It was diagnosed as adenocarcinoma arising in ES. The area of this tumor was later reexcised, along with an enlarged inguinal lymph node. The primary lesion showed distinct areas of ES and carcinoma and one area of transition from ES to carcinoma. Gland formation was present in some carcinomatous nests. The lymph node contained solid sheets of anaplastic carcinoma cells. The patient has remained clinically free of tumor. (20 refs)

79-4708 **Malignant Fibrosarcomatous Mesothelioma and Benign Pleural Fibroma (Localized Fibrous Mesothelioma) in Tissue Culture. A Comparison of the In Vitro Pattern of Growth in Relation to the Cell of Origin.** (Eng) Alvarez-Fernandez, E. (Servicio de Anatomia Patologica, Ciudad Sanitaria Provincial, Dr. Esquerdo 46, Madrid 30, Spain); Diez-Nau, M. D. *Cancer* 43(5): 1658-1663; 1979.

The case reports of two patients (26-yr-old man, 52-yr-old woman) with fibrous mesothelioma are presented. The first patient had a malignant tumor containing bundles of spindle-shaped cells with a dense reticulin network and nests of epithelial-like cells. The second had a benign tumor made up of spindle-shaped cells arranged in bundles with abundant reticulin and collagen fibers. Tissue culture in the

first case revealed plaques similar to those formed by epithelial tumors. The second case had a fibroblastic pattern with single isolated spindle-shaped cells. These findings confirm the mesothelial nature of fibrosarcomatous mesothelioma and support the view that the so-called localized fibrous mesotheliomas could be fibroblastic neoplasms derived from submesothelial connective tissue. (15 refs)

- 79-4709 Metastasis-induced Acute Pancreatitis in Small Cell Bronchogenic Carcinoma.** (Eng) Yeung, K. Y. (6525 Belcrest Rd., Suite 460, Hyattsville, MD 20782); Haidak, D. J.; Brown, J. A.; Anderson, D. *Arch Intern Med* 139(5): 552-554; 1979.

The occurrence of clinical acute pancreatitis in 3 of a consecutive series of 40 patients with oat cell lung cancer is reported. The patients were two 53-yr-old men and a 37-yr-old woman. Metastasis-induced acute pancreatitis (MIAP) was initially suspected when the patients showed symptoms of acute abdominal pain associated with nausea and vomiting, signs of epigastric tenderness, palpable masses in the epigastric and left upper quadrant areas, and elevated serum and urine amylase levels. The diagnosis was confirmed by the rapid and complete resolution of all evidence of pancreatitis following initiation of multidrug antineoplastic therapy. Signs of resolution of the pancreatitis coincided with signs of objective tumor regression elsewhere. It is suggested that MIAP is not a rare clinical entity in patients with small cell lung cancer and that it should be considered in any patient with known small cell bronchogenic carcinoma in whom an acute abdomen develops. Also, small cell lung cancer should be added to the differential diagnosis of acute pancreatitis. Intensive polychemotherapy is indicated in MIAP since this treatment offers the greatest likelihood of rapid palliation and prolongation of survival. (20 refs)

- 79-4710 Bronchiolo-alveolar Carcinoma with Nodal Metastases. An Ultrastructural Study.** (Eng) Morningstar, W. A. (Veterans Adm. Hosp., Cleveland, OH); Hassan, M. O. *Am J Surg Path* 3(3): 273-278; 1979.

The ultrastructural features of a metastatic bronchiolo-alveolar carcinoma in a 58-yr-old man were studied. The primary tumor and lymph node metastases showed identical features. The tumor cells were cuboidal to columnar and were arranged in a glandular pattern separated from the adjacent tissue by a poorly formed basement membrane. The nuclei were large and irregular, and prominent nucleoli, microvilli, desmosomes, swollen mitochondria, and rough endoplasmic reticulum were observed. The most prominent feature was the presence of dense bodies showing a characteristic lamellar pattern. In most cells these bodies were abundant and appeared to replace most of the

cell organelles. No secretory granules were seen. The cells showed features characteristic of type II granular pneumocytes, suggesting that at least some variants of bronchiolo-alveolar carcinoma arise from type II alveolar pneumocytes. (20 refs)

- 79-4711 A Variant of Intestinal Metaplasia Associated with Gastric Carcinoma. A Premalignant Lesion (Meeting Abstract)?** (Eng) Jass, J. R. (Dept. Histopathology, Westminster Medical Sch., London, England); Filipe, M. I. *Br J Surg* 66(5): 361; 1979 (2 refs)

- 79-4712 Gastric Adenocarcinoma due to Ataxia-Telangiectasia (Louis-Bar Syndrome).** (Eng) Frai, M. A. (Walton Hosp., Rice Lane, Liverpool 9, England). *J Med Genet* 16(2): 160-161; 1979.

Gastric adenocarcinoma associated with ataxia-telangiectasia (AT) occurred in a 26-yr-old man who had had AT since childhood. He presented with wt loss, anorexia, and dyspepsia. Laparotomy disclosed extensive tumor infiltration of the greater omentum. It is proposed that AT be recognized as predisposing to gastric adenocarcinoma. (8 refs)

- 79-4713 Familial Form of Carcinoma of the Colon** (Fre) Gautier-Benoit, C. (Service de Chirurgie Generale, Centre Hospitalier de Lens, F62302 Lens, France); Deregnaucourt, G.; Prat, A. *Gastroenterol Clin Biol* 3(2): 131-134; 1979.

Cancer of the cecum was diagnosed in 3 siblings (2 brothers and 1 sister, aged 42, 43, and 46 yr). The tumors were identified as differentiated adenocarcinoma, mucinous carcinoma, and differentiated, partially mucinous adenocarcinoma, respectively. Intestinal polyps were absent in all 3 cases. A genealogical examination revealed cancer of the sigmoid colon in the siblings' father and in his two brothers, as well as in their paternal grandfather. Familial carcinoma of the colon may be related to immunological disorders. (9 refs)

- 79-4714 Cytogenetic Investigation of Murine Tumors Originating from Fetal Intestine.** (Rus) Manolov, G. (No affiliation given); Urumov, I.; Argirova, R.; Petkova, P. *Probl Onkol (Sofia)* 6: 74-77; 1978.

The results of cytogenetic studies of a transplantable adenocarcinoma of the large intestine are presented. The tumor was obtained from a sc transplanted fetal large intestine of BALB/c mice. Analysis of the distribution of

marker chromosomes showed that chromosomes M1, M2, and M3 were dominant. Chromosome M1 consisted of segments of chromosome 11 (centromere region) and chromosome 9 (distal region). Chromosome M2 occurred after the deletion of chromosome 16, and chromosome M3 occurred after inversion of the distal region of chromosome 19. (no refs)

79-4715 Extragenital Adenomatoid Tumor. Evidence for the Mesothelial Theory of Origin. (Eng) Craig, J. R. (Los Angeles County-Univ. Southern California Medical Center, Los Angeles, CA); Hart, W. R. *Cancer* 43(5): 1678-1681; 1979.

The development of a histologically typical adenomatoid tumor in the small intestinal mesentery of a 39-yr-old chronic alcoholic man is reported. The nodule, which was diagnosed as a metastatic adenocarcinoma, was found during exploratory surgery following a gunshot wound to the abdomen. Additional studies showed no evidence of any other neoplasm, and a percutaneous needle biopsy of the liver 3 yr later showed no evidence of neoplasm. The microscopic features of the resected nodule were atypical of an adenomatoid tumor as characteristically encountered in the genital tract. This case provides corroborative evidence for the mesothelial origin of adenomatoid tumors. These distinctive tumors should be referred to as benign adenomatoid mesotheliomas regardless of their site of origin. (18 refs)

79-4716 Failure to Demonstrate Specificity of the Morphological and Histochemical Changes in Mucosa Adjacent to Colonic Carcinoma (Transitional Mucosa). (Eng) Isaacson, P. (Dept. Pathology, Southampton General Hosp., Tremona Road, Southampton SO9 4XY, England); Attwood, P. R. *J Clin Pathol* 32(3): 214-218; 1979.

A high iron diamine/alcan blue stain was used to examine 80 sections of colorectal adenocarcinoma with adjacent benign mucosa, 22 sections of normal rectosigmoid mucosa, and the tumor-mucosal junctions of 3 anal melanomas, 5 anal squamous cell carcinomas, and 1 malignant lymphoma of the colon. Normal rectosigmoid mucosa consisted predominantly of sulfomucins that stained brown/black; a few sialomucin-containing cells that stained blue were found near the surface. In comparison, the mucosa adjacent to the colorectal adenocarcinomas was thicker with deeper crypts and taller goblet cells; sialomucins predominated. Generally, the mucosa adjacent to the other tumors and that taken from specimens of solitary ulcer syndrome and colostomies showed similar histologic and morphologic changes. Crypt depth and goblet cell height were increased in transitional mucosa and in the other conditions studied. The results suggest that the

morphologic appearances and histochemical characteristics of transitional mucosa do not reflect a specific premalignant change but are secondary to the presence of colonic adenocarcinoma. (11 refs)

79-4717 Establishment of a New Cell Line (NBT-2) Derived from a Human Urinary Bladder Carcinoma and Its Characteristics. (Jpn) Yamamoto, T. (Dept. Urology, Niigata Univ. Sch. Medicine, Niigata, Japan). *Jpn J Urol* 70(3): 351-357; 1979.

A continuous cell line (NBT-2) was established from a urinary bladder carcinoma in a 64-yr-old man. The tissue for the cell cultures was taken during a partial cystectomy. A papillary growth (1.0 x 1.5 x 1.5 cm) was found near the left ureteral orifice, and there were basal invasions into the bladder wall. Although the papillary growth appeared to be a Stage III transitional cell carcinoma, the intramuscular invasions were found to be more undifferentiated (Stage IV). The cultured cells have been maintained for 28 mo and passaged 120 times. The NBT-2 cells are concluded to be identical to those of the urinary bladder carcinoma on the basis of the following: (1) the epitheliallike cells grew more rapidly than the fibroblastlike cells, and at the final transition stage in the primary cultures, there was a complete monolayer of epitheliallike cells; (2) the ultrastructural characteristics of the NBT-2 cells resembled those of the Stage IV urinary bladder carcinoma cells; (3) chromosomal analysis of the early cultured cells (fourth passage) showed the presence of two hypertriploid stemlines (72 and 74); (4) histological examination of heterotransplants in Syrian golden hamster cheek pouches revealed that the nature of the cells was very similar to that of the original Stage IV undifferentiated transitional cell carcinoma cells. (25 refs)

79-4718 A Unique Ultrastructural Variant of Wilms' Tumor. Its Possible Histogenetic Implications. (Eng) Kurtz, S. M. (Lab. Div., Veterans Admin. Hosp., 100 Bee St., Charleston, SC 29403). *Am J Surg Pathol* 3(3): 257-264; 1979.

A rare ultrastructural variant of Wilms' tumor in a 3-month-old black infant is described. The child was one of twins born to a 50-yr-old woman. The tumor was removed surgically and has shown no evidence of recurrence. Toluidine-blue staining showed the presence of densely staining granules in the cytoplasm of virtually all tumor epithelial cells. They were not present in the rare, well-differentiated tubules, or in the stromal cells. Interspersed among the granules were many clear, small vacuolar structures. In all other respects the appearance of the tissue conformed to that of Wilms' tumor. Electron microscopy showed that the dense bodies were limited by a single membrane and that they contained heterogeneous masses of coarse, dense amorphous material, finely granular

substance, and fragments of membranous structures. Morphologically, these bodies resembled autophagosomes of the type that are seen in abundance only in granular cell myoblastoma. The clear vacuoles, which were limited by a double membrane, appeared to be altered mitochondria. The occurrence of the autophagosomes in this case supports a neurectodermal origin for Wilms' tumor. (14 refs)

- 79-4719 Multiple Adenocarcinomas and Premalignant Changes in "Backwash" Ileitis.** (Eng) Schlipfert, W. (2202 Lloyd Center, Portland, OR 97232); Mitros, F.; Schulze, K. *Am J Med* 66(5): 879-882; 1979.

The development of multiple carcinomas in the colon and ileum of a 27-yr-old woman with long-standing ulcerative colitis and "backwash" ileitis is reported. Three slightly elevated plaques in the terminal ileum were identified as moderately well-differentiated adenocarcinomas. Multiple random sections from the colon revealed two lesions with the histologic characteristics of carcinoma. In both locations, premalignant changes of the basal cell proliferation type were seen adjacent to and remote from the tumors. The multiplicity of tumors in the setting of premalignant changes suggests that "backwash" ileitis increases the risk of small bowel carcinoma. (40 refs)

- 79-4720 Hypernephroma and Hypertension: Observations.** (Eng) Pillari, G. (Dept. Radiology, Long Island Jewish-Hillside Medical Center, Long Island, NY); Fulco, J. D.; Lee, W. J. *NY State J Med* 79(6): 865-867; 1979.

Fifty-six consecutive, surgically proved cases of hypernephroma were reviewed with special attention to the incidence of significant associated hypertension and presenting signs and/or symptoms. The neoplasms were discovered incidentally by timed-sequences urography, nephrotomography, sonography, and selective renal angiography. Presenting signs and symptoms included flank pain and/or back pain (16 cases), hematuria (10), hypertension (7), wt loss (6), and mixed signs and symptoms (17). Twenty-eight patients had significant hypertension preoperatively, and in seven, hypertension was the only physical sign. At surgery, there was no evidence of metastatic disease in any of these seven patients. The characteristics of the tumors found in the seven patients included small tumor size, contained cortical location, and absence of identifiable tumor necrosis. Remarkable polycythemia was present in 2/7 patients but in only one other patient in the entire series. This investigation suggests at least an empiric relationship of hypernephroma and hypertension; however, the etiologic relationship between the two conditions requires further study. It is concluded that neoplasm should be included in the differential diagnosis in the hypertensive patient and that radiology

and selective angiography are imperative in identifying this neoplasm. (10 refs)

- 79-4721 State of RBC Surface (Echinocytosis) During Experimental Carcinogenesis.** (Rus) Kashulina, A. P. (Lab. Pathophysiology, P. A. Hertsen Res. Oncological Inst., Moscow, USSR); Tereshchenko, I. P. *Biull Eksp Biol Med* 87(5): 455-457; 1979.

An attempt was made to determine whether alterations of the surface of RBC such as echinocytosis occur not only during certain liver diseases and various types of muscle dystrophies but also during experimental carcinogenesis. Experiments were carried out in C3H mice with spontaneous carcinomas of the mammary gland and in C57BL/6 mice with sc transplanted Ehrlich adenocarcinomas. In 3- to 4-mo-old C3H mice (early pretumor period), echinocytes comprised 58.6% of all RBC, compared with 91.1% in 11- to 12-mo-old mice (late pretumor period) and 96.6% in mice with mammary gland tumors. In control C57BL/6 mice, echinocytes comprised only 18.7% of all RBC, but the development of Ehrlich adenocarcinoma was associated with a marked increase in the number of echinocytes (up to 65.3%). (6 refs)

- 79-4722 Morphometric and Cytophotometric Investigations of Lobular Neoplasia of the Breast with Ductal Involvement.** (Eng) Zippel, H. H. (Universitäts-Frauenklinik Köln, Kerpener Strasse 34, D-5000 Cologne 41, W. Germany); Henatsch, H. J.; Kunze, W. P. *J Cancer Res Clin Oncol* 93(3): 265-274; 1979.

The DNA contents of neoplastic ductal and lobular cells obtained from 11 cases of lobular neoplasia of the breast were determined. In the nine cases corresponding to type A neoplasia, the nuclei were primarily diploid, whereas in the two cases corresponding to type B, the DNA content showed broad variation, extending into the hypertetraploid range. Compared with the type A cells, the type B cells showed a much more irregular distribution and marked variations in nuclear size and shape. The mean nuclear size in the type A cells was 73.3 arbitrary Units (AU), whereas that in the type B cells was 95.6 AU. The cell density (number of nuclei per lobular area) was much higher in the type B lesions. The data support the contention that infiltrating ductular carcinoma originates in lobular changes via ductal alterations. (19 refs)

- 79-4723 Precancerous Alterations of the Breast and Early Breast Carcinoma. Clinical, Histological, Morphological and Cytophotometric Investigations at the Cologne University Gynecological Clinic.** (Ger) Zippel, H. H. (Universitäts-Frauenklinik

Koln, Kerpener Strasse 34, 5000 Koln 41, W. Germany)
Fortschr Med 97(4): 159-164; 1979.

Clinical and morphological methods for defining and clarifying precancerous alterations of the mammary gland and early breast carcinoma were applied to 822 breast carcinoma patients and 1,691 patients with benign breast lesions admitted to a German clinic during 1957-1974. Patients with tumors up to 2 cm in diameter, which comprised 51.8% of all tumors during 1970-1974, had a 5-yr survival rate of 84.2% if there were no axillary metastases. Only tumors < 0.5 cm in diameter could be classified as the true early form of carcinoma (microcarcinoma). In microcarcinoma patients, there were no additional sites of carcinoma in the mammary gland and lymph node tissue. Facultative precancerous alterations were considered intraductal cellular proliferations with or without sporadic atypical cells and type A lobular neoplasia (diploid DNA distribution). Obligatory precancerous alterations were considered intraductal cellular proliferations with many atypical cells and type B lobular neoplasia (aneuploid DNA distribution). Among the study population, the risk of carcinoma was found to be 2.2% for the type A lobular neoplasia and 3.4% for type B. The most important method for detecting the premalignant and early stages of breast cancer is mammography. In >60% of all patients, microcalcification had been the indication for histological examination. (no refs)

79-4724 Adenofibroma of the Breast and Pubertas Praecox. (Spa) Arbues, F. (Servicio de Tocoginecologia, Residencia Sanitaria "Ortiz de Zarate" de la Seguridad Social, Calle Sandazuri 17, Vitoria, Spain); Gimeno, S.; Vilela, C. *Tokoginecol Pract* 37(419): 279-288; 1978.

A tumor of the left breast was found in a 10-yr-old girl with pubertas praecox who had menarche at the age of 8 yr, 9 mo. The tumor, measuring 6.5 x 5.5 x 3.5 cm, was identified histologically as a pericanalicular fibroadenoma. Abundant proliferation of ducts and acini and normal mitoses were seen. There was no epithelial atypia. (12 refs)

79-4725 Factors Associated with Breast Structure in Breast Cancer Patients. (Eng) Grove, J. S. (Pacific Health Res. Inst., Honolulu, HI 96813); Goodman, M. J.; Gilbert, F.; Clyde, D. *Cancer* 43(5): 1895-1899; 1979.

Variables that seem to affect the breast structure of women with diagnosed breast cancer were evaluated. The breast duct patterns and radiographic density, or dysplasia, of 104 breast cancer patients in Hawaii were examined by mammography. The proportions of the four types of breast structure were analyzed for possible relation with age,

menopausal state, height, wt, and race. Multiple regression analysis indicated that menopausal state appears to be more important than age per se for the general change in breast structure. Low body wt, but not race, is associated with prominent duct patterns and dysplasia. Breast tissue type, age at diagnosis, ethnic group, age at menopause, artificial or natural menopause, height, wt, and menopausal status at diagnosis are tabulated for each patient. (6 refs)

79-4726 Dermatomyositis and Visceral Neoplasia. A Case Report. (Ita) Depaoli, M. (Ospedale Dermatologico S. Lazzaro, Turin, Italy); Zina, A. *G Ital Dermatol* 114(1/2): 57-60; 1979.

Dermatomyositis was diagnosed in five women and one man, four of whom (all women) also had cancer (carcinomas of the breast, uterus, and meningioma). In these four patients, the dermatosis was present for only a few months. The findings suggest a relationship between cancer and dermatomyositis. (14 refs)

79-4727 Cystosarcoma Phyllodes of the Breast. Report of 12 Cases. (Spa) Duque Gallo, J. A. (Ciudad Sanitaria de la Seguridad Social "La Fe", Valencia, Spain); Valls Valero, F.; Garcia Ferrer, J. L.; Cabo Valle, A.; Jorda Cuevas, M.; Rivas Roder, S. *Tokoginecol Pract* 37(419): 313-320; 1978.

Cystosarcoma phyllodes (CP) of the breast was diagnosed in 12 patients among a series of 1,512 women with breast tumors, including 988 benign tumors and 524 malignant tumors. Eleven CP were benign and one turned malignant with two recurrences. The average age of the 12 patients was 40.8 yr. Nine patients were nulliparas; the remaining three patients had breast-fed their babies. Four patients had a history of fibrocystic disease and one had a history of polycystic ovary. The background of the seven other patients was not remarkable. (6 refs)

79-4728 Breast Cancer and Pregnancy. (Spa) Muxi, M. (Residencia Maternal, Ciudad Sanitaria de la Seguridad Social "Francisco Franco", Barcelona, Spain); Perez-Soler, J.; Salvador-Monte, R.; Rodriguez, C.; Monguio, A.; Duran, A.; de la Riva, A.; Xercavins, J.; Somoza, J. A.; Garriga, J. H. *Tokoginecol Pract* 37(419): 295-306; 1978.

Four cases of breast cancer that occurred during pregnancy were reported, and the related literature is reviewed. These four cases were observed among a series of 191,798 pregnancies, corresponding to a frequency of 1/47,949 births. The patients were aged 30, 33, 40, and 43 yr, respectively. None of them had an unusual general or

gynecological background. Advanced pulmonary and pleural metastases were found in two patients at diagnosis. One patient developed a bone metastasis that caused a pathological fracture. (15 refs)

- 79-4729 Breast Cancer and Pregnancy: Results of a Survey Conducted by the Social Security Administration of Barcelona, Spain.** (Spa) Muxi, M. (Residencia Sanitaria Seguridad Social "Francisco Franco," Barcelona, Spain); Perez-Soler, J.; Garriga, J. H.; Rodriguez, C.; de la Riva, A. M.; Xercavins, J.; Monguio, A.; Somoza, J. A.; Aguilar, I. *Tokoginecol Pract* 37(419): 307-312; 1978.

Breast cancer was diagnosed during pregnancy in four women. They were treated only after delivery. The babies had a normal development. (no refs)

- 79-4730 A Report of the Histological Features in 12 Cases of Gonadoblastoma.** (Eng) Woodcock, A. S. (Royal Coll. Obstetricians and Gynaecologists, Sussex Place, Regent's Park, London, NW 1 4RG, England); Govan, A. D.; Gowing, N. F.; Langley, F. A.; Anderson, M. C. *Tumori* 65(2): 181-189; 1979.

The histological features of 12 cases of gonadoblastoma submitted to the Ovarian Tumour Panel of the Royal College of Obstetricians and Gynaecologists are reported. The patients were 9-26 yr old. Children may present with obvious genital malformations, retarded growth, or precocious puberty. In adults, the main complaint is amenorrhea, but sometimes there is associated masculinization. Histologically, gonadoblastomas have a distinctive structure that is easily recognized in most instances. The most important feature is the instability of the germ cells in these tumors. Nine of the patients showed an associated dysgerminoma that was bilateral in four. In any case of suspected gonadal dysgenesis, presumptive evidence of a diagnosis is suggested by the presence of a Y chromosome, raised gonadotropin levels, and pelvic calcification on x-ray examination. At operation, all streak tissue representing both gonads must also be removed, since these tumors are frequently microscopic in size and bilateral. For the same reasons, the excised tissue should be serially sectioned. (7 refs)

- 79-4731 Association of Brenner's Tumor of the Ovary with Endometrial Adenocarcinoma.** (Fre) Bremond, A. (Clinique gynecologique, Hopital Edouard Herriot, Place d'Arsonval, 69300 Lyon Cedex 2, France); Frappart, L. *Rev Fr Gynecol Obstet* 74(5): 365-367; 1979.

The association of differentiated endometrial adenocar-

cinoma with typical Brenner's tumor of the left ovary was found in a 62-yr-old woman. The estradiol level in the venous blood of the tumor-bearing ovary was 15 p(pico)g/ml, and in the peripheral blood, 9 pg/ml. The findings suggest the possible endocrine action of the stroma in a few cases of Brenner's tumor; the coexistence of the two tumors may not be a coincidence. (5 refs)

- 79-4732 Pure and Mixed Brenner Tumors of the Ovary. Clinicopathologic and Histogenetic Observations.** (Eng) Waxman, M. (Inst. Pathology, Downstate Medical Center, Kings County Hosp., 450 Clarkson Ave., Brooklyn, NY 11203). *Cancer* 43(5): 1830-1839; 1979.

An analysis was made of 56 Brenner tumors in 51 patients and of cases reported in the literature, with particular attention being given to the problem of histogenesis. The incidence of bilaterality was 9.8%. Most tumors were < 2 cm, and they constituted an incidental microscopic finding. Most of the patients were 30-49 yr old (mean age 49.7, main age 44). A high incidence of associated malignant neoplasms was noticed among the patients with Brenner tumor (12 patients had 16 malignancies), but this finding was considered coincidental. Among the 16 associated tumors were 7 squamous cell carcinomas of the cervix, 4 breast carcinomas, and 2 endometrial carcinomas. There was no evidence of hormone secretion by the tumors. Examination of 31 tumors revealed that 27 occupied the cortex. Urothelial metaplasia of the ovarian surface epithelium was demonstrated in one case, and continuation of the Brenner column with the surface of the ovary in another. There were 12 cases of mixed Brenner tumor in this series. The most frequent associated constituents of the mixed Brenner tumors are mucinous cystadenomas. These tumors represent a single mixed neoplasm originating in a multipotential celomic cell that proliferates and differentiates into several mullerian forms. The histogenesis of medullary and hilar Brenner tumors can be explained on the basis of celomic metaplasia. Uncertainty persists in explaining the histogenesis of Brenner tumors mixed with mature cystic teratoma. (21 refs)

- 79-4733 Characterization of Normal Cervical Epithelium, Intraepithelial Neoplasias and Cervical Carcinomas In Vitro by Quantitative Studies on Nuclear Overlap Behaviour.** (Eng) Ebeling, K. (Acad. Sciences German Democratic Republic, Central Inst. Cancer Res., DDR 1115 Berlin-Buch., E. Germany); Tanneberger, S. *Int J Cancer* 23(5): 632-638; 1979.

Quantitative studies were made of the nuclear overlap (NO) behavior of cells from normal cervical epithelium, dysplasia, carcinoma in situ (CIS), and cervical carcinoma (CVC) cultivated in vitro. In outgrowth cultures of normal

epithelium, a monolayer composed of cohesive polygonal epithelial cells was seen. In cultures derived from dysplasia, CIS, and CVC, there was a decrease in cell cohesion followed by an alteration of cell form. In all cultures, the number of NO observed was lower than the number expected. The NO index (the actual number of NO's, expressed as a percentage of the number to be expected when the cells are distributed randomly) of cultures from normal cervical epithelium was significantly lower than that of all other tissues examined. There was no significant difference between the NO indices of CVC and ovarian carcinoma nor between the NO-indices of CIS or dysplasia and CVC. Both the significant difference in NO index between cells cultured from normal cervical epithelium and intraepithelial atypias and CVC's, and the nonsignificant difference between intraepithelial atypias and CVC's could be considered an expression of the same biological deviation in preinvasive and invasive lesions of the cervix uteri. In spite of their different morphology, dysplasia, CIS, and invasive CVC have biologically common features, and the different morphological classification of the preinvasive lesions of the cervix uteri does not always reflect their real invasive potential. (48 refs)

79-4734 Cancer of the Vulva Seen at One Department. (Spa) Miguez, F. O. (Departamento de Tocoginecologia, Seguridad Social "Nuestra Senora de la Candelaria," Residencia Sanitaria, Santa Cruz de la Tenerife, Spain); Cotter y Cotter, J. M. *Tokoginecol Pract* 37(415): 141-146; 1978.

Eight cases of cancer of the vulva were diagnosed among a series of 27,390 women treated for various conditions during an 8-yr period. Seven patients were 40-79 yr old and one was >80 yr old. All patients were multiparous. The tumor was localized to the labia in 5 patients, to the clitoris in 2, and to the urethra in 1. (7 refs)

79-4735 The Detection of Malignant Prostatic Cells in the Urine. Definition of a High-Risk Population. (Fre) Benoit, G. (Clinique Urologique, Hopital Cochin, 27 rue du Faubourg St. Jacques, F75014

Paris, France); Boccon-Gibod, L.; Dalian, G.; Desligneres, S. *Nouv Presse Med* 8(19): 1604; 1979.

Experience over the last 20 yr at a urology clinic is reviewed briefly. Correlations between urine cytology findings suggestive of malignancy (Grade IV) and clinical and histological data have made it possible to single out two populations: one in which histological examination confirmed the diagnosis of prostatic carcinoma and one in which histological confirmation was not obtained. Of the latter, approx 20% had prostatic or bladder carcinoma. (6 refs)

See also:

*(Rev.): 79-4202, 79-4208, 79-4214, 79-4221, 79-4248, 79-4255, 79-4256, 79-4257, 79-4260, 79-4261, 79-4262, 79-4277, 79-4289, 79-4290, 79-4291, 79-4292, 79-4293, 79-4294, 79-4295, 79-4296, 79-4297, 79-4298, 79-4299, 79-4300, 79-4301, 79-4316.

*(Chem.): 79-4330, 79-4338, 79-4339, 79-4352, 79-4354, 79-4356, 79-4362, 79-4374, 79-4378, 79-4385, 79-4389, 79-4394, 79-4400, 79-4404, 79-4405, 79-4410, 79-4412, 79-4413, 79-4415, 79-4416, 79-4436, 79-4451, 79-4455, 79-4457, 79-4464, 79-4487, 79-4491, 79-4496, 79-4499, 79-4500, 79-4501, 79-4508.

*(Phys.): 79-4510, 79-4514, 79-4516, 79-4517, 79-4518, 79-4525, 79-4526, 79-4528, 79-4529, 79-4530, 79-4531, 79-4534, 79-4536, 79-4547, 79-4550, 79-4552, 79-4553.

*(Viral): 79-4617, 79-4618, 79-4620, 79-4630, 79-4631.

*(Immun.): 79-4662, 79-4669, 79-4672, 79-4678, 79-4681, 79-4689, 79-4690, 79-4692.

*(Epid.-Biom.): 79-4738, 79-4749, 79-4760, 79-4762, 79-4772, 79-4776, 79-4780, 79-4781, 79-4786.

EPIDEMIOLOGY AND BIOMETRY

- 79-4736 **Geographical Distribution of Burkitt's Lymphoma in Nigeria.** (Eng) Durodola, J. I. (Dept. Surgery, Oncology Unit, Univ. Coll. Hosp., Ibadan, Nigeria). *Trop Geogr Med* 30(4): 463-466; 1978.

The geographic distribution of Burkitt's lymphoma (BL) in Nigeria was studied based on 880 cases recorded in an Ibadan hospital's cancer registry during 1961-1975. Of the 880 patients, 550 (62.5%) were males and 330 (37.5%) were females. The peak incidence was at age 8-9 yr. The 87 towns/localities from which the cases were reported were distributed all over Nigeria except the most northern Sudan savanna zone. No case of BL has been reported from areas at a latitude 11 degrees north of the equator. Of the 880 cases, 104 were from Ibadan, 36 from Ogbomoso, 24 from Abeokuta, 22 from Ife, and 20 from Ilesha; all these areas are in the southern forest zone. The high concentration of BL cases in these localities is probably due to the proximity of hospitals and diagnostic facilities. (4 refs)

- 79-4737 **Clinical and Quantitative Staging and Monitoring of Multiple Myeloma. Analysis of 145 Clinical Cases.** (Ita) Di Guglielmo, R. (Istituto di Clinica Medica II, Università degli Studi, Florence, Italy); Vercelli, D.; Guidi, G. *Recent Prog Med* 66(4): 382-417; 1979.

A clinical and quantitative staging system for multiple myeloma (MM) was used to assess the myeloma tumor cell mass in 145 patients (55.86% men, 44.12% women, aged 33-82 yr). The extent of the bone lesions is the most important parameter in both staging systems. The quantitative staging is more reliable than the clinical because it takes into account all parameters at the same time, never relying on a single variable. According to the clinical staging, 20 patients were in Stage I, 55 in Stage II, and 70 in Stage III. According to the quantitative staging, 15 patients were in Stage I, 78 in Stage II, and 52 in Stage III. The mean survivals showed good correlation with the quantitative staging. (88 refs)

- 79-4738 **Cancer Morbidity in Rheumatoid Patients.** (Fin) Isomaki, H. (Rheumatism Foundation's Hosp., 18120 Heinola 12, Finland); Hakulinen, T.; Joutsenlahti, U. *Duodecim* 95(4): 157-163; 1979.

The incidence of cancer among patients with rheumatoid arthritis was studied using the Social Security and Finnish Cancer Registers. The results were compared with in-

cidence among the general population according to age and sex. A total number of 11,483 men and 34,618 women suffered from rheumatoid arthritis. Four hundred and four men had tumors (expected number was 354). The difference was most notable for leukemia, lymphoma, myeloma and cancer of the respiratory tract. Seven hundred and ninety-five women had cancer (expected number, 784). The incidence was higher for leukemia, lymphoma, Hodgkin's disease and myeloma in particular with the women also, but the incidence of stomach and intestinal cancer was lower in the women than expected. The incidence of cancer among the men was higher than in the general population; among the women, it was not elevated. (18 refs)

- 79-4739 **Risk Factors in Lip Cancer: A Questionnaire Survey.** (Eng) Lindqvist, C. (Finnish Cancer Registry, Liisankatu 21 B, 00170 Helsinki 17, Finland). *Am J Epidemiol* 109(5): 521-530; 1979.

An analysis was made of potential risk factors for lip cancer in Finland. The series comprised 290 patients with epidermoid carcinoma of the lip and 254 controls with squamous cell carcinoma of the skin of the head and neck, all reported to the Finnish Cancer Registry during 1973. The results obtained in a questionnaire survey (response rate, 75%; 54% of the total series) indicated that male lip cancer patients had engaged in outdoor work and smoking with significantly greater frequency than had the male controls. Together, these two risk factors posed a markedly increased risk (relative risk = 15.4). However, when the factors were analyzed separately, to exclude the effect of one on the other, no significant risk was noted. The frequency of recurrent herpetic infections was significantly greater in the men with lip cancer than in the men with head and neck cancer. No significant differences were apparent with respect to urban or rural residence. A negative geographic correlation exists between the amount of total solar radiation and lip cancer in Finland. Therefore, the main risk of lip cancer is probably the synergistic effect of another climatic factor and smoking. The risk associated with outdoor work (and perhaps also with herpetic ulcers) may lower the resistance of the vermilion border of the lip, thus increasing its susceptibility to tobacco carcinogens. (35 refs)

- 79-4740 **Nickel as Carcinogenic Factor in Nasal Carcinoma.** (Eng) Torjussen, W. (Central County Hosp., N-4601 Kristiansand, Norway). *Acta Otolaryngol [Suppl]* (Stockh) 360: 125; 1979.

The nickel concentrations in the plasma, urine, and nasal mucosa of 318 Norwegian nickel workers, 15 retired nickel workers, and 57 age-matched controls were studied. The mean nickel concentrations in the plasma, urine, and nasal mucosa of the nickel workers (6.3 $\mu\text{g/liter}$, 49.1 $\mu\text{g/liter}$, and 273.9 $\mu\text{g/100 g}$, respectively) were higher ($p < 0.01$) than those of the controls (1.9 $\mu\text{g/liter}$, 4.9 $\mu\text{g/liter}$, and 12.9 $\mu\text{g/100 g}$, respectively). The values for the retired nickel workers (2.9 $\mu\text{g/ml}$, 11.3 $\mu\text{g/liter}$, and 114.4 $\mu\text{g/100 g}$, respectively) were also higher than those of the controls ($p < 0.01$). The half-life for nickel in the nasal mucosa of the retired workers was estimated to be 3.5 yr. Workers exposed to heavy aqueous soluble nickel compounds retained more nickel in the nasal mucosa and experienced a higher risk for nasal carcinoma than those exposed to soluble nickel compounds. (3 refs)

- 79-4741 Occupational Etiology and Nasal Cancer. An Internordic Project.** (Eng) Engzell, U. (Dept. Otolaryngology, Huddinge sjukhus, S-14186 Huddinge, Sweden). *Acta Otolaryngol [Suppl] (Stockh)* 360: 126-128; 1979.

The occupations and exposures of 44 Swedish men with nasal adenocarcinoma and 127 Swedish men with squamous cell and poorly differentiated nasal carcinoma were studied. Of the men with adenocarcinoma, 22/44 were joiners, most of them cabinet makers. The number of subjects working in the flour industry (3/44) was also higher than expected. The latency period from first exposure to diagnosis was 22-70 yr (mean, 44.7 yr), and the exposure period was generally more than 30 yr in the case of the joiners. The series of men with squamous cell and poorly differentiated carcinoma has not yet been fully investigated. (12 refs)

- 79-4742 Relationship Between the Epstein-Barr Virus Genome and Nasopharyngeal Carcinoma in Caucasian Patients.** (Eng) Andersson-Anvret, M. (Dept. Tumor Biology, Karolinska Institutet, S 10401 Stockholm 60, Sweden); Forsby, N.; Klein, G.; Henle, W.; Bjorklund, A. *Int J Cancer* 23(6): 762-767; 1979.

To determine whether the association between undifferentiated nasopharyngeal carcinoma (NPC) and Epstein-Barr virus (EBV) is constant, regardless of the geographical and ethnic origin of the patient, a correlated histopathological and nucleic acid hybridization study was made of biopsies from Swedish and Belgian Caucasian patients with NPC and from various controls. Among 12 undifferentiated NPC's, 11 were positive for EBV-DNA, with each cell containing multiple copies of the viral genome. Serological tests showed elevated anti-viral capsid antigen and anti-early antigen (diffuse component) titers. Six NPC's with various degrees of squamous differentiation, four malig-

nant lymphomas of the nasopharynx, and seven carcinomas located outside the nasopharynx were EBV-DNA negative. These findings further stress the uniqueness and regularity of the association between EBV-DNA and undifferentiated NPC. Clearly, the association extends over geographical barriers and holds true not only in the previously studied, moderate-incidence African ethnic group, but also in low-incidence Western patients. (20 refs)

- 79-4743 Irradiation-induced Tumours of the Head and Neck.** (Eng) Aanesen, J. P. (Dept. Otolaryngology, Linköping Univ. Hosp., S-58185 Linköping, Sweden); Olofsson, J. *Acta Otolaryngol [Suppl] (Stockh)* 360: 178-181; 1979.

The occurrence of 14 radiation-induced tumors of the head and neck among 11 patients seen at a Swedish hospital between 1968 and 1977 is reported. The four women and seven men ranged in age from 46 to 77 yr (mean, 65 yr) at the time of diagnosis. The mean interval between radiotherapy and tumor diagnosis was 32 yr. The tumors were: 4 squamous cell carcinomas (SCC) of the hypopharynx, 3 SCC of the buccal mucosa, 2 SCC of the skin, 1 SCC of the epiglottis, 2 thyroid carcinomas, 1 poorly differentiated carcinoma of the parotid gland, and 1 fibrosarcoma of the sternocleidomastoid muscle. Although radiation-induced tumors are uncommon, all patients given radiotherapy should be followed throughout life. (11 refs)

- 79-4744 Case-Control Study: Soft-Tissue Sarcomas and Exposure to Phenoxyacetic Acids or Chlorophenols.** (Eng) Hardell, L. (Center Oncology, Univ. Hosp., S-901 85 Umea, Sweden); Sandstrom, A. *Br J Cancer* 39(6): 711-717; 1979.

Exposure to phenoxyacetic acids (PAA) and chlorophenols (CP) was studied in 52 patients with soft-tissue sarcomas (STS) and 208 controls. Thirty-one patients were deceased at the time of the study. Exposure to PAA or CP was registered in 36.5% of the STS patient and 9.2% of the controls. The relative risk for STS after exposure to PAA was calculated as 5.3. The relative risk associated with exposure to CP was 6.6. Four (7.7%) patients and 14 (6.8%) controls reported the use of paints containing CP during their leisure. Four patients and 14 controls also reported exposure to dichlorodiphenyltrichloroethane (DDT), the relative risk for which was calculated as 1.2. The risk of STS among unexposed persons involved in the same occupations as the exposed patients was 0.6, suggesting that the excess risk associated with PAA and CP was indeed related to these chemicals. There was no apparent difference in smoking habits between patients and controls. It is possible that the increased risk of STS associated with exposure to PAA or CP is caused by the pure chemical

substances themselves and/or by impurities in the commercial preparations. (10 refs)

- 79-4745 Occupational and Bronchial Carcinoma. (Eng) Hillerdal, G. (Dept. Lung Medicine, Univ. Uppsala, S 750 14 Uppsala, Sweden); Nou, E. *Scand J Respir Dis* 60(2): 76-82; 1979.

All bronchial carcinoma patients from Uppsala County, Sweden, that were diagnosed from 1971 to 1976 were studied. There were 212 male and 61 female patients. A careful occupational history was taken, and the occupations of the patients in 1950 were compared with the County's official occupational statistics of that year. Chest x-rays were scrutinized for pleural plaques. Ninety-six percent of the men and 36% of the women were smokers. A significantly higher proportion of metal workers and workers from the building industry was found among the patients, and agricultural workers were underrepresented. Smoking habits did not explain the difference. Of the male patients, 53 had a history of exposure to asbestos. All 53 were current smokers or exsmokers. The number of patients with pleural plaques was four times higher than expected. It is concluded that dust in certain occupations has an additive effect on the carcinogenic effect of smoking. (34 refs)

- 79-4746 Lung Cancer Mortality in Aluminum Reduction Plant Workers. (Eng) Gibbs, G. W. (Dept. Epidemiology and Health, McGill Univ., 3775 University St., Montreal, Quebec H3A 2B4, Canada); Horowitz, I. *J Occup Med* 21(5): 347-353; 1979.

The lung cancer mortality (LCM) of 5,406 men (Cohort 1) employed at one aluminum smelter in Quebec on January 1, 1950, and of 485 men employed at a second Quebec smelter (Cohort 2) on January 1, 1951, is reported. The total number of years of exposure to tars, number of years since first exposure to tars, and an index of exposure to tars expressed in tar-years were calculated for each man. More than 99% of the men in Cohort 1 and approx 98% of the men in Cohort 2 were traced. As of December 31, 1973, 1,070 men in Cohort 1 had died, and death certificates were obtained for 990; and 64 men in Cohort 2 had died, and death certificates were obtained for 58. There were 84 lung cancer deaths in Cohort 1 and 11 in Cohort 2. The results showed that the LCM of that portion of Cohorts 1 plus 2 who had ever been exposed to tars was similar to that of workers never exposed to tars. The LCM of men in Cohort 1 was greater than that expected according to Quebec provincial rates, but this was probably due to the slightly increased LCM in the communities serving the industries. Although the total number of cases in Cohort 2 was small, the LCM was well in excess of that expected at Quebec rates and it could not be explained on the basis of com-

munity experience. There was a definite dose-response relationship between LCM and tar-years and years of exposure. The standardized mortality ratio for persons exposed for >21 yr to the higher levels of tars was 2.3 times that of persons not exposed to tars. Although smoking may still be a factor, the evidence suggests that the increased risk of lung cancer is related to employment in definite tar-exposed occupations. (7 refs)

- 79-4747 Radiotherapy of Early Glottic Cancer--I. (Eng) Harwood, A. R. (Dept. Radiation Oncology, Princess Margaret Hosp., 500 Sherbourne St., Toronto, Ontario, M4X 1K9, Canada); Hawkins, N. V.; Rider, W. D.; Bryce, D. P. *Int J Radiat Oncol Biol Phys* 5(4): 473-476; 1979.

Retrospective analysis of the results of treatment of 378 patients with early glottic cancer at one hospital between 1965 and 1974 revealed that increasing the size of the treatment volume (field) while retaining the same dose reduced the local recurrence rate after radiotherapy from 18% to 9% ($p = 0.034$). (13 refs)

- 79-4748 A Mortality Study of Oil Refinery Workers. (Eng) Theriault, G. (Dept. Social and Preventive Medicine, Laval Univ., Quebec G1K 7P4, Canada); Goulet, L. *J Occup Med* 21(5): 367-370; 1979.

The survival status of 1,205 men employed for >5 yr in a Canadian oil refinery from 1928 through 1976 was assessed, and death certificates were reviewed. Expected numbers of deaths were estimated based upon age- and cause-specific death rates for the Province of Quebec applied to person-years at work. The standard mortality ratio for the oil refinery workers was lower than that expected for all causes of death (SMR = 78.43). Three cancers of the brain were found among young people who died <20 yr since the start of exposure. This incidence was statistically higher than that expected. Although the incidence of cancer of the digestive system was not significantly higher than that expected, these cancers remain suspect of being occupationally associated. (11 refs)

- 79-4749 Etiology of Pleural Calcification: A Study of Quebec Chrysotile Asbestos Miners and Millers. (Eng) Gibbs, G. W. (Dept. Epidemiology, Occupational Health and Safety Unit, McGill Univ., Montreal, Quebec, Canada). *Arch Environ Health* 34(2): 76-83; 1979.

A review of 15,689 chest radiographs of Quebec chrysotile miners and millers identified 206 men with pleural calcification. Of these, 198 had worked in the Thetford

Mines area, 6 at Asbestos, and 2 at St. Remi de Tingwick; 2.5%, 0.08%, and 1% of the chest films from these areas, respectively. A series of case-control studies revealed that pleural calcification was concentrated in men employed at a small group of mines in Thetford Mines and occurred more often among miners and maintenance personnel than among millers. Calcification was not related to past history of illness or injury, place of residence, or employment in other industries. The distribution of pleural calcification suggested that the condition is related to some characteristic of airborne dust or mineral closely associated with the chrysotile that is encountered in the Thetford Mines but not in other mining areas. Possible minerals include mica talc, and breunnerite. (36 refs)

79-4750 Should Lung Cancer in Iron Ore Miners in Lorraine Be Considered an Occupational Disease? (Fre) Anthoine, D. (Clinique pneumophtisiologique de Nancy, Hopital Villemin, rue de Nabecor, Nancy 54000, France); Braun, P.; Cervoni, P.; Schwartz, P.; Lamy, P. *Rev Fr Mal Respir* 7(1): 63-65; 1979.

Lung cancer was found in 270 underground iron ore mine workers in the Lorraine area. Seventy-five per cent of the miners had been on the job for at least 20 yr, and 73% smoked at least 20 cigarettes a day. Epidermoid carcinoma was found in 80%, anaplastic carcinoma in 17.5%, other histological types in the rest. The workers had been exposed to carcinogenic dust and diesel exhaust gases, but radon exposure was not found. The findings indicate the simultaneous involvement of several cocarcinogenic factors in the development of lung cancer in the miners and suggest the carcinogenic effect of iron. Lung cancer in iron ore miners should be considered an occupational disease. (9 refs)

79-4751 Lung Cancer Mortality of Workers in Chromate Pigment Manufacture: An Epidemiological Survey. (Eng) Davies, J. M. (Div. Epidemiology, Inst. Cancer Res., Sutton, Surrey SM2 5PX, England). *J Oil Colour Chem Assoc* 62(5): 157-163; 1979.

An epidemiologic survey of lung cancer mortality among the workers in three small pigment factories was undertaken. In two factories in which the workers had mixed exposure to both lead chromate and zinc chromate, men who had experienced high and medium exposures showed more than twice the expected number of lung cancer deaths ($p < 0.01$). There was no excess mortality from lung cancer among men with low exposures or among those who had been employed for < 1 yr. In one factory, the excess exposures occurred within 25 yr of the first exposure; 4/22 occurred within 5-9 yr and 5/22 occurred within 10-14 yr. Few employees had worked in the second factory for 25 yr.

There have been no lung cancer deaths among men who entered employment at either factory during 1968-1974. There was no excess mortality among the employees of the third factory, where only lead chromate was manufactured. (9 refs)

79-4752 Re: "A Cluster of Hodgkin's Disease in a Small Community" (Letter to Editor). (Eng) Zack, M. (Center Disease Control, Atlanta, GA 30333); Heath, C. W., Jr.; Isbister, J. L.; Van Amburg, G. *Am J Epidemiol* 109(5): 621-622; 1979.

A study of cancer incidence in similar communities with and without navy bean elevators revealed that both communities had significantly fewer lung cancer deaths than expected and fewer deaths due to Hodgkin's disease. (6 refs)

79-4753 X-linked Lymphoproliferative Syndrome Registry (Letter to Editor). (Eng) Purtilo, D. T. (Dept. Pathology, Univ. Massachusetts Medical Center, Worcester, MA 01605); Hamilton, J. *Ann Intern Med* 90(6): 995; 1979.

The X-Linked Lymphoproliferative Syndrome Registry was recently established, and it is funded by the National Cancer Institute. Referrals of possible cases of the syndrome as well as unusual complications associated with infectious mononucleosis are invited. Three criteria to be used to ascertain whether a patient is a candidate for the registry are given. (1 ref)

79-4754 An Association of Upper Respiratory Cancer with Exposure to Diethyl Sulfate. (Eng) Lynch, J. (Exxon Corp., P.O. Box 45, Linden, NJ 07036); Hanis, N. M.; Bird, M. G.; Murray, K. J.; Walsh, J. P. *J Occup Med* 21(5): 333-341; 1979.

A morbidity and mortality study was made of workers at an alcohol manufacturing plant that included several weak acid isopropyl alcohol units and a strong acid ethanol unit. An excess mortality of laryngeal cancer was found, and it was associated with work on the strong acid ethanol unit. The strong acid ethanol process used resulted in the formation of high concentrations of diethyl sulfate, a compound that has been shown to be carcinogenic in animals. The unit, which closed in 1975, had significant opportunities for worker exposure to diethyl sulfate. There have been previous reports of excess upper respiratory cancer associated with work on strong acid isopropyl alcohol units with similarly high concentrations of the animal carcinogen diisopropyl sulfate. These facts lead to the tentative conclusion that diethyl sulfate was primarily responsible for the

ethanol unit cancer cases. In modern weak alcohol plants, where only trace amounts of diisopropyl sulfate are present and exposures are much lower, the problems found with the old strong acid units do not exist. (40 refs)

- 79-4755 Malignancy in Uremia: Dialysis Versus Transplantation.** (Eng) Herr, H. W. (Dept. Surgery, Univ. California, Irvine, CA 92717); Engen, D. E.; Hostetler, J. *J Urol* 121(5): 584-586; 1979.

The incidence of cancer was compared in 499 dialysis patients and 121 renal transplant recipients. De novo malignancy developed in 15 patients on chronic dialysis and in 6 transplant recipients, a significant increase over the expected number in the age-matched general population. The mean duration of dialysis before diagnosis of cancer was 27.3 mo (range 2-74 mo), and the mean tumor latency time after transplantation was 30.8 mo (range 13-54 mo). There was no statistical difference between the groups with respect to age, sex, underlying renal disease, duration of treatment before diagnosis of cancer, or incidence of cancer in uremic patients on dialysis or after transplantation. A total of 10 dialysis patients and 1 transplant patient died of cancer. Neoplasms in the dialysis patients comprised a broad spectrum of common mesenchymal tumors, but superficial skin cancers were seen more frequently in the transplant recipients. The differences in tumor types accounted for the high mortality rate from cancer in the dialysis patients, and they may reflect different patterns of immunosuppression in these two patient populations. (9 refs)

- 79-4756 Is Low-Dose Radiation Associated with Myeloma (Letter to Editor)?** (Eng) Najarian, T. (193 Lewis Road, Belmont, MA 02178); Castleman, B. *N Engl J Med* 300(22): 1278; 1979.

The incidence of prior exposure to radiation and solvents was determined among 44 men and 45 women with myeloma. Six of the men and six of the women had had previous radiation exposure compared with 4/120 male controls and 3/108 female controls. Ten of the female myeloma patients and six of the female controls had worked with solvents (mostly in dry cleaning). (1 ref)

- 79-4757 Immunohistochemical Evidence for RNA Virus Related Components in Human Breast Cancer.** (Eng) Mesa-Tejada, R. (Inst. Cancer Res., 701 West 168th St., New York, NY 10032); Keydar, I.; Ramanarayanan, M.; Ohno, T.; Fenoglio, C.; Spiegelman, S. *Ann Clin Lab Sci* 9(3): 202-211; 1979.

An indirect immunoperoxidase method was used to localize antigenic components with cross-reactivity to a 52,000-

dalton group-specific glycoprotein (gp52) of the mouse mammary tumor virus (MMTV) in paraffin sections of human breast carcinomas. A positive staining reaction was observed with 171/376 (45.5%) of the cancers tested. Invasive carcinomas with intraductal components were more likely to contain cross-reactive antigen than were either pure intraductal or invasive tumors. The pattern of staining tended to be focal, intracellular, and cytoplasmic with considerable variability even within the same tumor. Absorption with purified gp52 eliminated the staining reaction, but absorption with numerous other related and unrelated substances was ineffective. Of 99 carcinomas from other organs and 8 cystosarcoma phyllodes, only 1 (a mucoepidermoid carcinoma of the parotid gland) gave a positive reaction in this test. Normal breast tissue and benign breast tumors also gave negative results, with the exception of an apocrine metaplasia associated with cystic disease. (36 refs)

- 79-4758 Increased Incidence of Breast Carcinoma in Patients with Irradiation for Post-Partum Mastitis: A Screening Situation.** (Eng) Logan, W. W. (1351 Mt. Hope Ave., Suite 108, Rochester, NY 14620); Mansur, P. S.; Cullinan, A.; Kowaluk, E.; Hutchings, J.; Hempleman, L. H. *J Surg Oncol* 11(3): 239-242; 1979.

Mammography examinations were performed on 265/606 women treated with x-rays for post-partum mastitis during 1940-1955 in Rochester, NY. The av dose for each treated breast was 377 rads (at 2.5 cm depth). No biopsies were recommended on the basis of physical examination alone, since no masses were palpated. Thirteen biopsies were recommended on the basis of the mammographic findings. One biopsy revealed an adenocarcinoma and a second showed a noninfiltrating lobular carcinoma in situ (in the untreated breast). Both cases were free of axillary node metastases. No carcinomas were found in 11 patients on whom biopsies were recommended because of fine calcifications, but the biopsies revealed a high proportion of precancerous lesions. It is recommended that women in this high-risk category have close medical supervision, yearly mammography, and perform careful self-breast examination. (2 refs)

- 79-4759 Risk of Breast Cancer Following Low-Dose Radiation Exposure.** (Eng) Boice, J. D. (Environmental Epidemiology Branch, NCI, NIH, Landow Building, Room 3C07, Bethesda, MD 20014); Land, C. E.; Shore, R. E.; Norman, J. E.; Tokunaga, M. *Radiology* 131(3): 589-597; 1979.

The risk of breast cancer following radiation exposure was determined based on surveys of 1,047 tuberculous women who had had multiple fluoroscopic examinations of the chest, 571 women irradiated for postpartum mastitis, and 63,263 female survivors of the atomic bomb. Among the

bomb survivors, 108/12,843 who received estimated breast doses of >10 rads developed cancer, compared with 243/49,095 who received 0-10 rads, for a relative risk factor of 1.7 ($p < 0.0001$). Forty-one of the tuberculosis patients developed breast cancer compared with 23.3 expected on the basis of population rates. The av cumulative dose per patient was estimated to be 150 rads. Among the mastitis patients, 36/571 developed breast cancer compared with 32/993 nonirradiated mastitis patients; the relative risk factor was 2.0 ($p < 0.001$). The av cumulative dose was 377 rads/breast and 247 rads/patient. Risk increased significantly with increasing dose ($p < 0.00002$). Fractionation did not appear to diminish the radiation risk, nor did time since exposure (even after 45 yr of observation). The interval between exposure and the clinical appearance of radiogenic breast cancer may be mediated by hormonal or other age-related factors, but it was unrelated to dose. The best estimate of risk among American women exposed after age 20 is 6.6 excess cancers/ 10^6 woman years-rad. (39 refs)

- 79-4760 Low Urinary Estrogen Glucuronides in Women at Risk for Familial Breast Cancer. (Eng) Fishman, J. (Rockefeller Univ., New York, NY 10021); Fukushima, D. K.; O'Connor, J.; Lynch, H. T. *Science* 204(4397): 1089-1091; 1979.

Daily (12-hr) urine collections taken throughout the menstrual cycle were obtained from 30 young women who, by genetic analysis, were at risk for familial breast cancer and from 30 control women matched for age, height, reproductive history, and ethnic background. Steroids in the urine, were extracted by glucuronidase hydrolysis, and estrone, estradiol, tetrahydrocortisol, allotetrahydrocortisol, testosterone, dihydrotestosterone, androsterone, etiocholanolone, and dehydroisoandrosterone were measured by radioimmunoassay. No significant differences were found between the high-risk subjects and the controls in the urinary concentration of any of the corticosteroids and androgens measured. Highly significant differences were observed in estrone and estradiol. The controls had a higher mean full-cycle concentration of estrone (9.8 mg/kg creatinine vs 7.9 for high-risk patients) and estradiol (6.8 mg/g creatinine vs 5.8 for high-risk patients). There were no significant differences between the two groups in the plasma concentrations of any of the hormones. This suggests that there may be a change in conjugative pattern of estrone and estradiol in the high-risk subjects with decreased glucuronidation and increased sulfation. If this speculation is valid, it suggests that the risk factor is linked to the metabolic fate of secreted estrogen, which is dependent on enzymes under genetic control. (20 refs)

- 79-4761 Growth Rates of Primary Breast Cancers. (Eng) Heuser, L (Dept. Surgery, Univ.

Louisville, Louisville, KY 40232); Spratt, J. S.; Polk, H. C. *Cancer* 43(5): 1888-1894; 1979.

Breast cancer growth rates are a critical aspect of the natural history of the disease. The growth rates of 32 primary breast cancers were determined from serial mammographic views of tumor nucleus shadows in a population of 109 cancers. These cancers were found in a screening population of 10,120 women receiving $>30,000$ mammograms over 3 yr. In 23 patients, tumor volume doubling times ranged from 109 to 944 days, with the mean doubling time being 325 days; in 9 patients, the tumors showed no growth. Additional cancers surfaced that were growing too fast to be measured. These cancers were significantly more likely to metastasize. However, because of the small sample size, the absolute percentage of tumors in the fast-growing subset was not determinable, but it ranged between 17% and 77% of the 109 cancers. (6 refs)

- 79-4762 Sulfur Dioxide Exposure in a Smelter. III. Acute Effects and Sputum Cytology. (Eng) Archer, V. E. (Nat'l. Inst. Occupational Safety and Health, Center Disease Control, PHS, DHEW, Room 433, 350 S. Main St., Salt Lake City, UT 84101); Fullmer, C. D.; Castle, C. H. *J Occup Med* 21(5): 359-364; 1979.

Although acute effects from exposure to low levels of sulfur dioxide have frequently been observed in acute exposure experimental studies, it was not known whether or not such effects occur among workers chronically exposed to 0.3-4 ppm of SO_2 . Measurements of forced vital capacity (FVC), mean forced expiratory volume (FEV₁), forced expiratory flow (FEF₅₀), FEF₇₅, and FEF₅₀₋₇₅ and closing volume were made before and after the workshift for 195 copper smelter workers and 54 controls. Sputum samples were collected for cytological examination. Mean FEV₁ and FVC values were significantly decreased during a day's work in the smelter. Significantly more smelter workers had a decrease in FEV₁ and FEF₅₀ during the day than did controls. More of the smelter workers felt "chest tightness." No change in closing volumes was seen. Smelter workers tended to have a higher percentage of sputum samples with moderate and marked atypia than did controls, but the difference was not statistically significant. The results of this study and a previous chronic effects study suggest that transient acute respiratory effects may be a harbinger of chronic effects if exposure is prolonged. (41 refs)

- 79-4763 Population Dose and Health Impact of the Accident at the Three Mile Island Nuclear Station. A Preliminary Assessment for the Period March 28 Through April 7, 1979. (Eng) Battist, L. (U.S. Nuclear Regulatory Commission, Washington, DC 20555); Buchanan, J.; Congel, F.; Nelson, C.; Nelson, M.; Peterson, H.; Rosenstein, M. (Washington, D.C.: U.S. Government Printing Office) 103 pp.; 1979.

The collective radiation dose from the March 28, 1979 accident at the Three Mile Island Nuclear Station was estimated for the approx 2 million people living within 50 miles of the station. The estimates cover the offsite releases that occurred from March 28 through April 7, and they are based on ground-level radiation measurements from thermoluminescent dosimeters located within 15 miles of the site. The dose estimates assume that the accumulated exposure recorded by the dosimeters was from gamma radiation, ie, radiation that penetrates the internal body organs. The estimated collective dose is 3,300 person-rem (roentgen-equivalent-man), which represents an av of four separate estimates that are 1,600, 2,800, 3,330, and 5,300 person-rem. The range of the collective dose values is due to different methods of extrapolating from the limited number of dosimeter measurements. The av dose to an individual in the affected population is 1.5 millirem, based on the av collective dose. The projected number of excess fatal cancers that could occur over the remaining lifetimes of the population is approx 1. Had the accident not occurred, approx 325,000 persons in the area would normally die of cancer. The projected total number of excess health effects, including fatal and nonfatal cancers and genetic effects in future generations, is approx two. (16 refs)

- 79-4764 Long-Term Effects of Radium Exposure in Female Dial Workers: Liver Function and Liver Disease.** (Eng) Polednak, A. P. (Center Human Radiobiology, Radiological and Environmental Res. Div., Argonne Natl. Lab., Argonne, IL 60439). *Environ Res* 18(2): 454-465; 1979.

To study the long-term effects of α -emitting radionuclides on the liver, the results of liver function tests and data on the prevalence of mortality from diseases of the liver and biliary tract among women first employed before 1930 in the US radium watch-dial painting industry were examined. There was little evidence for a relationship between radium intake dose (initial system burden) and serum levels of albumin, total bilirubin, glutamic oxaloacetic transaminase (SGOT), or cholesterol in 142 long-term survivors. Mean SGOT level was significantly higher in the highest intake dose group ($\geq 50 \mu\text{Ci}$) than in lower intake-dose groups, suggesting the need for continued clinical follow-up. There was no significant association between intake-dose and serum alkaline phosphatase in 264 women in whom the enzyme was measured. The prevalence of diagnosed diseases of the liver was not significantly related to intake-dose level in 683 women, nor were the observed numbers of deaths from cirrhosis of the liver or liver cancer increased relative to the US white female population. (32 refs)

- 79-4765 Viets and Vets Fear Herbicide Health Effects. Vietnamese Official Brings Concerns to**

Washington. (Eng) Wade, N. (No affiliation given). *Science* 204(4395): 817; 1979.

The possibility that the (2,4,5-trichlorophenoxy)acetic acid herbicide contaminant dioxin may be carcinogenic has been raised by a Vietnamese surgeon. The incidence of liver cancer during 1974-1977 apparently increased in areas of South Vietnam that had received the bulk of the spraying, whereas the incidence in North Vietnam remained the same. Chloracne, birth defects, miscarriages, and chromosome breaks have also been observed in Vietnamese who inhabited sprayed areas. The chloracne, numbness of the digits, irritability, and other toxic reactions noted in significant numbers of Vietnam veterans may also be related to dioxin exposure in Vietnam veterans. (1 ref)

- 79-4766 Correlations in Mortality Data Involving Cancers of the Colorectum and Esophagus.** (Eng) Lipworth, L. L. (Dept. Family and Community Medicine, Univ. Massachusetts Medical Sch., 55 Lake Ave. North, Worcester, MA 01605); Rice, C. A. *Cancer* 43(5): 1927-1933; 1979.

Massachusetts vital event data for 1969-1972 were used to develop correlations between mortality rates for malignant diseases and other causes of death over the 34 health planning subdivisions of the state. Ischemic heart disease (IHD) was significantly correlated with cancers of the colon, rectum, and colorectum. These associations were not a function of population size, but both IHD and colorectal cancer were significantly negatively correlated with median family income. Cancer of the esophagus was significantly correlated with cirrhosis and lung cancer, although the data suggest a greater etiologic similarity between esophageal cancer and cirrhosis than between esophageal cancer and lung cancer. Cancers of the lung and colorectum, breast and bladder, uterine cervix and lung, stomach and uterine cervix, and stomach and uterine body/"uterus unspecified" were correlated at the 5% level of significance. (30 refs)

- 79-4767 Cancer Morality in U.S. Counties with Shipyard Industries During World War II.** (Eng) Blot, W. J. (Environmental Epidemiology Branch, NCI, Bethesda, MD 20014); Stone, B. J.; Fraumeni, J. F.; Morris, L. E. *Environ Res* 18(2): 281-290; 1979.

To evaluate the possibility of a carcinogenic hazard from asbestos used in ship construction and repair, cancer mortality rates for US counties, in which large shipyards were located during World War II, were determined. The 49 counties included both metropolitan areas and small coastal counties with deep-water ports. For the period 1950-1969, rates among white males in most shipyard counties (SYC) were higher than the corresponding US rate or

the rate in 80 urban control counties. The excess was most consistent in the southern SYC. The rate of increase in lung cancer mortality was only marginally greater for SYC in the Northeast and West but was pronounced for SYC in the South. The distribution of lung cancer in white females in the SYC tended to parallel that in white males. Mortality from laryngeal, oropharyngeal, esophageal, and stomach cancers also tended to be high in over half of the SYC. The rates for oropharyngeal and lung cancers were particularly high in urban SYC in the south, whereas the rates for esophageal cancer were highest in urban counties in the northeast and the rates for stomach cancer were highest in the northeast and north central regions. The data suggest that shipyard exposures to asbestos have contributed to the clustering of cancer mortality in coastal areas of the country. (23 refs)

- 79-4768 U.S. Trends in Mortality from Carcinoma of Cervix (Letter to Editor). (Eng) Anello, C. (Div. Biometrics, Food and Drug Admin., Rockville, MD 20857); Lao, C. *Lancet* 1(8124): 1038; 1979.

Although cervical cancer mortality rates for 1970-1976 decreased among US women >25 yr old, there was a consistent increase in the 1974-1976 rates among women <25 yr old. Based on data from two hysterectomy surveys, this apparent increase did not reflect different hysterectomy patterns in different age groups. Between 1973 and 1976, the percentage of hysterectomies increased in all age groups from 15-44 yr. (1 ref)

- 79-4769 Incidence of Ovarian Neoplasms at the Bloemfontein Academic Hospitals, 1972-1977. (Eng) Venter, P. F. (Dept. Obstetrics and Gynaecology, Univ. Orange Free State, Bloemfontein, S. Africa); Anderson, J. D.; Van Velden, D. J. *SAfr Med J* 55(23): 911-913; 1979.

The incidence of ovarian neoplasms among black patients seen at the Pelonomi Hospital and white patients seen at the National Hospital in Bloemfontein, South Africa, during 1972-1977 was studied. There were 47 benign and 61 malignant ovarian tumors among the 9,807 black patients and 12 benign and 85 malignant ovarian tumors among the 1,895 white patients. The relatively low incidence of ovarian cancer among black patients remains to be explained. (15 refs)

- 79-4770 A Study on the Aetiological Factors of Bilharzial Bladder Cancer in Egypt. 3. Urinary β -Glucuronidase. (Eng) El-Aaser, A. A. (Dept. Cancer Biology, Cancer Inst., Cairo Univ., Cairo, Egypt); El-Merzabani, M. M.; Higgy, N. A.; Kader, M. M. *Eur J Cancer* 15(4): 573-583; 1979.

The β -glucuronidase (BG) from human liver, spleen, kidney, and normal bladder mucosa was compared with that from bladder cancer tissue, bladder tissue from patients with bilharzial infections, and intact *Escherichia coli*. The BG activity of bladder tumor tissue (18.4 units) was higher than that of normal tissue, but intact *E. coli* showed that the highest level (27.2 units). The human and *E. coli* enzymes reacted differently to different buffers and urine constituents and showed different pH optima. Of the different species of bacteria isolated from the urine of patients with bladder cancer or bilharzial infection, *E. coli* showed the highest BG activity, followed by *Staphylococcus*, *Proteus*, *Klebsiella*, and *Streptococcus faecalis*. With increasing incubation time in citrate-phosphate buffer at optimum pH, liver BG activity increased whereas *E. coli* BG showed a lag time. The BG activity of the urine from bladder cancer patients and those infected with bilharzia was higher than that of urine from normal subjects. There was a positive correlation between type and severity of bacterial infection and BG activity. (42 refs)

- 79-4771 Geographic Epidemiology of Bladder Cancer Deaths in Japan: Focus on City and Country Distribution. (Jpn) Ohno, Y. (Dept. Preventive Medicine, Nagoya Univ. Sch. Medicine, Nagoya, Japan); Aoki, K.; Shimizu, H.; Tominaga, S. *Acta Urol Jpn* 25(2): 121-132; 1979.

Geographic variations in bladder cancer mortality in Japan during 1969-1971 were analyzed on a city and county basis. Of 1,123 areas, the mortality rates among men aged 40 yr and older were significantly higher than the national av in 71 areas (10.29-50.15/100,000); the rates among women were significantly higher in 63 areas (6.97-26.00/100,000). The mortality was often higher in rural areas than in industrial areas along the Pacific coast. To determine the industrial makeup of areas with high bladder cancer mortality, the percentage of men working in 46 industries was compared with the corresponding percentage in low-risk areas. The comparisons were restricted to (1) cities in which lung cancer mortality in men did not exceed the national av and (2) to cities with an excess bladder cancer risk for men (male:female ratio >1.50). The industries associated with the high incidence of bladder cancer in men were those manufacturing textiles; pulp and paper products; rubber; leather and fur products; iron, steel, and nonferrous products; fabricated metal products; electric machinery and precision machines; and the printing industry. Other environmental and socioeconomic factors may contribute to the high bladder cancer incidence in rural areas. (27 refs)

- 79-4772 Multiple Primaries in Patients with Oral Cavity Malignancies. Autopsy Examination. (Jpn) Takahashi, H. (Dept. Oral Pathology, Tohoku Univ. Sch.

Dentistry, Sendai 980, Japan); Okabe, H.; Wakasa, H. *Gan No Rinsho* 25(4): 267-272; 1979.

Of 97 patients with oral cavity malignancies autopsied at Tohoku University, Japan, during 1961-1975, 7 (all men 54-83 yr old) had multiple tumors. This rate was higher than that reported at other institutions (108/2,872). The mortality of these multiple tumor patients was higher than that of the single tumor patients. The tumors occurred simultaneously in 3/7 patients: squamous cell carcinoma (SCC) of the maxillary sinus and adenocarcinoma (AC) of the stomach, SCC of the tongue and SCC of the esophagus, and SCC of the soft palate and transitional cell carcinoma (TCC) of the ureter. In the remaining patients, the non-simultaneous tumors were AC of the rectum, AC of the stomach, and anaplastic carcinoma of the gingiva; SCC of the gingiva and AC of the stomach; TCC of the urinary bladder and SCC of the tongue; and SCC of the soft palate and AC of the kidney. There were four oral cavity/digestive tract tumor combinations and three oral cavity/urinary tract tumor combinations. Pharyngeal malignancies occurred more often than maxillary tumors in single oral cavity tumor patients (47% vs 2%), but the reverse was true in multiple oral cavity tumor patients (4% vs 11%). For the entire country of Japan, urinary tract tumors were more frequently associated with oral cavity tumors than digestive tract tumors (0.17% vs 0.1%). (12 refs)

79-4773 Relationship Between Double Primary Malignant Tumors and History of Therapeutic Radiation: Analysis of 22 Patients with Radiation-Induced Cancer. (Jpn) Mitsuhashi, N. (Dept. Radiology, Gunma Univ. Sch. Medicine, Maebashi, Gunma Prefecture 371, Japan); Itoh, J.; Niibe, H. *Nippon Acta Radiol* 39(3): 243-251; 1979.

Of 4,186 patients irradiated during 1952-1977 for primary malignant tumors, 38 developed a second primary (radiation-induced) cancer (SPC). These patients were divided into three groups: (1) 22 patients (12 women) with SPC's in the irradiated area; 16/22 SPC's were histologically identical to the initial primary cancer (IPC); (2) 4 patients (2 women) with SPC's following intracavitary or interstitial radium therapy to IPC's at other sites; and (3) 12 patients (7 women) with SPC's following external irradiation to IPC's at other sites. Group 4 consisted of 29 patients (17 women) with SPC's but without a history of therapeutic irradiation. In Group 1, the IPC's were in the head and neck (12 cases), female genital organs (6), breast (3), and skin (1); the SPC's were in the head and neck (9), female genital organs (5), skin (4), and esophagus (4). In Groups 2-4, most of the SPC's were in the female genital organs (14), stomach and colon (9), breast (7), and head and neck (7) and most of the SPC's were in the lung (16), stomach and colon (8), head and neck (7), and female genital organs (5). The latent period between development

of the IPC and the SPC was longer in Group 1 than in the other groups (av of 14.7, 2.3, 5.8, and 6.4 yr, respectively). There was no significant correlation between latent period and age, radiation source, or radiation dose. (42 refs)

79-4774 Cancer Statistics in Japan and in Osaka: Incidence, Therapy, and Survival. (Eng) Fujimoto, I. (Dept. Field Res., Osaka Cancer Registry, Center Adult Diseases, Nakamichi 1-3-3, Higashinari-ku, Osaka 537, Japan); Hanai, A. *Gann Monogr Cancer Res* 22: 201-215; 1979.

The cancer incidence in Japan was estimated using data from five population-based cancer registries: Miyagi, Kanagawa, Osaka, Tottori, and Okayama. The population of the five prefectures was 16.1 million, or 16% of the entire Japanese population. In 1972, estimated incidence rates for all sites in Japan were 193.6/100,000 population in men and 167.8/100,000 in women. The most frequent sites in men were the stomach, lung, liver, esophagus, rectum, colon, and pancreas, in that order. In women they were the stomach, uterus, breast, lung, liver, colon and rectum. The age-specific incidence rates by site and sex are presented. The following were observed in the Osaka Cancer Registry: during 1963-1973, cancer of the stomach and uterus decreased by 20%-25% and cancer of the lung, colon, and pancreas increased by >25%; the 5-yr survival rate for cancer of all sites diagnosed during 1968-1971 was 17.6% for men and 28.1% for women. (11 refs)

79-4775 Cancer of the Large Bowel in a Defined Population: Canterbury, New Zealand, 1970-4. (Eng) Stewart, R. J. (University Dept. Surgery, Clinical Sch. Medicine, Christchurch Hosp., Christchurch 1, New Zealand); Robson, R. A.; Stewart, A. W.; Stewart, J. M.; Macbeth, W. A. *Br J Surg* 66(5): 309-314; 1979.

A total of 1,024 new cases of large bowel cancer occurred in Canterbury, New Zealand (population 400,796), during 1970-1974, and the 992 cases that were diagnosed before death are reviewed. There were 484 men and 508 women, most of whom were 60-80 yr old. The high incidence of this disease in New Zealanders of European origin is illustrated. There was only one Maori patient in the group. A significant difference in the site distribution of primary tumors between the sexes was found, with a female preponderance of cancer of the proximal colon gradually changing to a male preponderance of cancer of the rectum. In all, 61.5% of the patients had lymph node metastases or advanced disease at the time of diagnosis or treatment. The estimated crude 5-yr survival rate of the whole group was 32.7% (relative rate 42.8%), and the crude 5-yr survival rate after potentially curative surgery was 48.4% (relative rate 62.4%). Comparison of the results with those for other populations indicates that there may be a higher rate of col-

on cancer in New Zealand and North America than in Europe. (27 refs)

79-4776 Ulcerative Colitis and Liver Disease: The Spectrum of Disease in 69 Patients (Meeting Abstract). (Eng) Siegel, J. H. (Academic Dept. Medicine, Royal Free Hosp., London, England); Morris, J. S. *Gastroenterology* 76(5, part 2): 1247; 1979 (no refs)

79-4777 Radiation Doses after Three Mile Island (Letter to Editor). (Eng) McLean, A. S. (Natl. Radiological Protection Board, Harwell, Didcot, Oxfordshire OX11 0RQ, England). *Lancet* 1(8124): 1039; 1979.

The collective radiation dose to the population of approx 2 million living within a 50-mile radius of the Three Mile Island nuclear power station in Pennsylvania has been estimated at 2,000-3,000 person-rem (roentgen-equivalents-man). This implies that there is approx one chance in four that there will be one extra death from cancer in this population, in addition to the approx 500,000 expected cancer deaths from all causes. (no refs)

79-4778 Cholesterol and Colon Cancer (Letter to Editor). (Eng) Thornton, J. R. (Dept. Medicine, Univ. Bristol, Bristol Royal Infirmary, Bristol BS2 8HW, England). *Lancet* 1(8123): 971; 1979.

The authors of an article in which dietary cholesterol was proposed to be cocarcinogenic for human colon cancer neglected to mention the possible roles of colonic bacteria and dietary fiber. They also indicated that cholesterol is a more likely colon tumor promoter than its bile-salt derivatives, while accepting the fact that the metabolic pathways of these substances are inextricably interrelated. Finally, their postulated mechanisms of cholesterol cocarcinogenesis are ambiguous. (10 refs)

79-4779 Faecal Bile Acids and Clostridia in the Aetiology of Colorectal Cancer (Meeting Abstract). (Eng) Murray, W. R. (Univ. Dept. Oncology, Western Infirmary, Glasgow, Scotland); Blackwood, A.; Calman, K. C.; MacKay, C. *Br J Surg* 66(5): 364; 1979 (1 ref)

79-4780 Primary Malignant Tumors of the Small Intestine. (Ger) Hohn, D. (Chirurgischen Klinik, Städtisches Klinikum Karlsruhe, Moltkestrasse 14,

7500 Karlsruhe, W. Germany). *Fortschr Med* 97(22): 1029-1032; 1979.

In a series of 34,394 laparotomies performed during 1960-1978, including 3,505 for malignant tumors of the gastrointestinal tract, primary malignant tumors of the small intestine were found in 21 patients. The average age of the patients was 66.4 yr (65.3 yr for the 11 men, and 64.1 yr for the 10 women). The tumors included adenocarcinomas (9), sarcomas (6), malignant lymphomas (4), and carcinoid tumors (2). The average age was 65.1 yr for the carcinoma patients, 59.3 yr for the sarcoma patients, and 70.7 yr for the lymphoma patients; the 2 patients with carcinoid tumors were 68 and 69 yr old. The tumors were localized in the duodenum in 2 cases, in the jejunum in 7, in the ileum in 10, and multiple tumors were seen in 2 cases. The duration of symptoms was 12.3 mo (8.2 mo after excluding 1 patient with a 9-yr delay between tumor onset and surgery). Only 5 patients were free from metastases at the time of surgery. (no refs)

79-4781 Development of Gastric Stump Cancer Following Resection of the Stomach. (Bul) Popov, P. (Inst. Gastroenterology, Sofia, Bulgaria); Brailski, Kh. *Vutr Bulest (Sofia)* 18(1): 33-38; 1979.

Of 654 patients who underwent stomach resection for benign tumors of the stomach and duodenum, 25 (20 men, 5 women; mean age 63 yr) developed gastric stump cancer. Seventeen patients underwent primary surgery for a duodenal lesion, 6 for gastric ulcer, and 2 for gastric polyps; 7 patients underwent Billroth I resection, 18 Billroth II resection. The average period between the surgery and the development of cancer was 22 yr. Following the stomach resection, 22 of the patients developed atrophic gastritis. (20 refs)

79-4782 The Incidence of Gastro-intestinal Cancer in North Baden (West Germany) 1971-1977. (Eng) Kayser, K. (Dept. Documentation, Historic and Social Pathology, Univ. Heidelberg, Heidelberg, W. Germany); Burkhardt, H. U. *J Cancer Res Clin Oncol* 93(3): 301-321; 1979.

The incidences of cancer of the esophagus, stomach, small intestine, colon, and rectum during 1971-1977 in the district of North Baden, West Germany, are presented, and their relation to environmental factors are discussed briefly. Esophageal cancer is rare in North Baden, and the male:female ratio is about 5:1. The causes are unknown, but there is some indication that high alcoholic consumption together with smoking leads to an increased risk. The

risk decreases at >70 yr of age. Gastric cancer comprises about one-third of all gastrointestinal (GI) cancers in this region. The male:female ratio has remained nearly constant over the study period, and it is about 1.6:1. Max risk occurs in the age groups 70-74 yr (men) and 75-79 yr (women). Approx 30% of these cancers are first diagnosed at surgery. Although great changes in food and living habits have occurred since 1910, the risk for developing gastric cancer has remained the same. Cancer of the small intestine is rare, with a total of 284 cases being found over the 7-yr period. Colon cancer comprises 25% of all the GI cancers, and there was a continuous increase in incidence from 1971 to 1977. More than one-half of all colon tumors are located in the sigmoid colon. Rectal cancer comprises approx 30% of all GI cancers, and max risk occurs at age 80-84 for men and 70-74 for women. The male:female sex ratio for this cancer increases more sharply with ascending age than that for colon cancer. The incidence is increasing much more slowly than that of colon cancer. The colon cancer incidence (per 100,000) increased from 18.2 in 1971 to 27.6 in 1977 in men and from 18.9 to 35.0 in women. The rectal cancer incidence increased from 31.9 to 36.9 in men and from 23.5 to 32.2 in women. The results of the pathoanatomic registry agree well with the data from clinical registries. (41 refs)

- 79-4783 Epidemiology of Malignant Neoplasms of the Breast, Uterine Cervix, Endometrium, and Rectum in Females in East Germany, 1962-1972. (Ger) Schott, J. (Lehrstuhl Soziale Gynakologie des Bereiches Medizin (Charite), Humboldt-Universitat zu Berlin, Tucholskystr. 2, DDR-104 Berlin, E. Germany). *Arch Geschwulstforsch* 49(1): 54-82; 1979.

Incidence, mortality, duration of medical care, and 5-yr survival rate were calculated for chorioepithelioma and other malignant neoplasms of the breast, uterine cervix, corpus uteri, and rectum in the female population of East Germany for the 1962-1972 period on the basis of a model. The calculated values show good agreement with the registered ones. (12 refs)

- 79-4784 An Epidemiologic Study of Breast Cancer and Benign Breast Neoplasias in Relation to the Oral Contraceptive and Estrogen Use. (Eng) Ravnihar, B. (Inst. Oncology, Medical Faculty, Univ. Ljubljana, Ljubljana, Yugoslavia); Seigel, D. G.; Lindtner, J. *Eur J Cancer* 15(4): 395-405; 1979.

The relationship between breast disease and use of oral contraceptives (OC's) and other estrogens (E's) was investigated in a case-control study involving 2,503 subjects. Of these patients, 371 had never used OC's although 16 of the 371 had used E's for other indications. E's as medication had been given to 290/2,503 patients, and 218/2,503 had taken non-E sex hormones. The proportion of OC users and long-term (≥ 2 yr) users among women ≤ 49 yr with cystic breast disease was significantly lower than among their matched controls; there were no significant differences between women with fibroadenoma and controls. Among patients with cystic disease and fibroadenoma, there was a higher proportion of women who had been pregnant or parous than among the controls. Fewer menopausal women than were expected were found among the cystic disease patients. Other factors usually associated with breast cancer were not associated with benign breast disease. (19 refs)

- 79-4785 Oesophageal Cancer in Greenland: Selected Epidemiological and Clinical Aspects. (Eng) Nielsen, N. H. (Inst. Forensic Medicine, Frederik den V's vej, DK-2100 Copenhagen O, Denmark); Mikkelsen, F.; Hansen, J. P. *J Cancer Res Clin Oncol* 94(1): 69-80; 1979.

Epidemiological and clinical aspects of 40 cases of esophageal cancer diagnosed among indigenous Greenlanders during 1955-1974 are reviewed. The annual incidence rates per 100,000, age adjusted to the world population, were 16.3 for men and 11.8 for women in 1955-1964. The corresponding rates in 1965-1974 were 15.9 for men and 6.7 for women, with a male:female ratio of 2.4:1. These rates rank among the moderately high rates found in India, Puerto Rico, and France and in US blacks. Distribution, anatomical location, and prognosis followed the normal pattern for esophageal cancer. No particular occupational trend was apparent, and there was no difference between towns and settlements. However, a statistically significant geographical gradient of frequency was found, with higher rates occurring in the southernmost three districts. The traditional Greenland diet may contain concentrations of precursors sufficient to create carcinogenic levels of nitrosamines. Further studies are needed of environmental factors such as foodstuffs, home-brewed beer, drinking water, and cigarette smoking. Attention should be focused on the special ecological conditions in southern Greenland. (57 refs)

- 79-4786 High Incidence of Undetected Neoplasia in Maldescended Testes. (Eng) Krabbe, S. (Children's Dept., Centralsygehuset, Hillerod, Denmark); Berthelsen, J. G.; Volsted, P.; Eldrup, J.; Skakkebaek, N.

E.; Eyben, F. V.; Mauritzen, K.; Nielsen, A. H. *Lancet* 1(8124): 999-1000; 1979.

The role of testicular biopsy in the early detection of testicular cancer in patients at risk was evaluated by studies of 50 previously cryptorchid men. Testicular biopsy specimens from 4/50 men previously treated for undescended testes had a carcinoma-in-situ pattern. Two of these men had adjacent invasive carcinoma (seminoma and embryonal carcinoma). The patient with embryonal carcinoma had an enlarged testis, but the three other patients with neoplasia had no clinical signs or symptoms of malignancy. Routine follow-up, including testicular biopsy, is essential in young men with undescended testes because of the increased risk of malignancy in this population and because this procedure may detect testicular neoplasia at a stage when orchidectomy alone is curative. (11 refs)

See also:

- *(Rev.): 79-4201, 79-4203, 79-4206, 79-4210, 79-4216, 79-4219, 79-4224, 79-4230, 79-4231, 79-4232, 79-4235, 79-4239, 79-4241, 79-4245, 79-4247, 79-4250, 79-4251, 79-4265, 79-4267, 79-4269, 79-4271, 79-4298, 79-4302, 79-4303, 79-4304, 79-4305, 79-4306, 79-4307, 79-4308, 79-4309, 79-4310, 79-4311, 79-4312, 79-4313, 79-4314, 79-4315, 79-4316, 79-4317, 79-4318, 79-4319, 79-4320, 79-4321, 79-4322, 79-4323, 79-4324, 79-4325.
- *(Chem.): 79-4351, 79-4409, 79-4418, 79-4425, 79-4430, 79-4431, 79-4432, 79-4447, 79-4463, 79-4506, 79-4508.
- *(Phys.): 79-4509, 79-4510, 79-4515, 79-4522, 79-4533, 79-4550.
- *(Immun.): 79-4678.

MISCELLANEOUS

- 79-4787 Biochemistry of Terminal Deoxynucleotidyltransferase. Mechanism of Manganese-dependent Inhibition by Deoxyadenosine 5'-Triphosphate and Biological Implications. (Eng) Modak, M. J. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021). *Biochemistry* 18(12): 2679-2684; 1979.

Studies showing that terminal deoxynucleotidyltransferase (TdT)-catalyzed DNA synthesis is strongly inhibited in the presence of all four ribonucleoside triphosphates, (rNTP's), with ATP being the most potent, are reported. In an activated DNA-primed reaction, the effective order of triphosphate inhibition was $A > G > C > U$. Furthermore, inhibition appeared to be more severe in the presence of Mn^{2+} than in the presence of Mg^{2+} . Extension of these findings to deoxynucleoside triphosphates (dNTP's) revealed that only dATP, in the presence of Mn^{2+} , strongly inhibited TdT-catalyzed incorporation of the remaining three triphosphates. In contrast to the inhibitory effect of rNTP's and Mn-dATP on TdT, replicative DNA polymerases were resistant to the addition of rNTP's and were either unaffected or stimulated by the addition of dNTP's. The Mn^{2+} -dependent dATP inhibition of TdT was found to be a result of multiple effects involving the three important components of the catalysis; namely, enzyme, primer, and substrate. The rates of incorporation of Mn-dATP were approx 50-fold lower than those observed with Mn-deoxyguanosine triphosphate (Mn-dGTP), although the affinity for binding Mn-dATP to enzyme was approx 1-2 orders of magnitude higher than that of Mn-dGTP. This probably results in the blockage of the substrate binding site on TdT by Mn-dATP. Increasing the primer concentration afforded partial protection from dATP inhibition; kinetic studies performed with changing primer concentration and inhibitor dATP yielded noncompetitive plots. A direct involvement of Mn-dATP as a competitor for the substrate dGTP for binding to the enzyme was revealed by the partial competitive modes of inhibition observed with kinetics studies performed by using these two substrates. Furthermore, the addition of Mn^{2+} to a reaction proceeding in the presence of Mg^{2+} may be instantly inhibited. Biological implications of the adenine nucleotide-mediated inhibition of TdT are discussed, along with the possible involvement of TdT in lymphocyte differentiation and immunodeficiency. (23 refs)

- 79-4788 Characterization of DNA Damages by Filtration Through Nitrocellulose Filters: A Simple Probe for DNA-modifying Agents. (Eng) Kuhnlein, U.

(Environmental Carcinogenesis Unit, British Columbia Cancer Res. Centre, 601 W. 10th Ave., Vancouver, British Columbia, Canada); Tsang, S. S.; Edwards, J. *Mutat Res* 64(3): 167-182; 1979.

The possibility of detecting ultimate carcinogens (those that do not need metabolic activation) was explored by exposing purified DNA to the carcinogenic agent and then measuring the introduction of DNA lesions. The method makes use of the property of nitrocellulose filters to selectively retain denatured DNA or DNA containing single-stranded regions. It allows the measurement of single- and double-stranded breaks, cross-links, damages that locally denature the DNA helix, damages that render the phosphodiester bonds of the DNA sensitive to hydrolysis, and damages that increase the depurination or the depyrimidination rate of the DNA. The method uses the double-stranded covalently closed circular DNA of phage PM2 as a substrate. Since the analysis involves only incubation and filtration steps, it is quick and well-suited as a routine method for screening chemicals with DNA-modifying and, therefore, potentially carcinogenic activity. (25 refs)

- 79-4789 Amplification of α -Fetoprotein Complementary DNA by Insertion into a Bacterial Plasmid. (Eng) Innis, M. A. (Roche Inst. Molecular Biology, Nutley, NJ 07110); Harpold, M. M.; Miller, D. L. *Arch Biochem Biophys* 195(1): 128-135; 1979.

The synthesis of duplex DNA-AFP from complementary DNA-AFP (cDNA-AFP), its insertion into a bacterial plasmid, and the identification of the cloned sequence are described. A fragment of the α -fetoprotein (AFP) structural gene was purified and amplified by bacterial cloning techniques. Double-stranded DNA-AFP was constructed from a cDNA copy of >95% pure messenger RNA-AFP (mRNA-AFP) and inserted into *Escherichia coli* plasmid pBR322 by poly(dA-dT)linkers. Chimeric plasmic DNA isolated from transformants of *E. coli* strain χ 1776 have been shown to contain α -fetoprotein sequences by hybridization to labeled mRNA-AFP. One clone, designated pA5 (chimeric plasmid pBR322 containing a cDNA-AFP sequence isolated from clone 5), was studied in more detail. The inserted sequence of approx 950 nucleotide pairs was positively identified by a hybridization-translation procedure. Hybridization of [3H]uridine-labeled poly(A)-containing RNA from an AFP-secreting cell line to excess pA5 DNA immobilized on nitrocellulose fibers was used to show the selectivity of this probe for detecting expression of the AFP gene. (40 refs)

79-4790 The Reactions of the *EcoRI* and Other Restriction Endonucleases. (Eng) Halford, S. E. (Dept. Biochemistry, Univ. Bristol, Bristol BS8 1TD, England); Johnson, N. P.; Grinstead, J. *Biochem J* 179(2): 353-365; 1979.

The reaction of *EcoRI* restriction endonuclease with both the plasmid pMB9 and DNA from bacteriophage λ was studied. With both circular and linear DNA molecules, the only reaction catalyzed by the enzyme was the hydrolysis of the phosphodiester bond within one strand of the recognition site on the DNA duplex. Both strands of the duplex were cleaved only after two independent reactions, each involving a single-strand scission. The reactivity of the enzyme for single-strand scissions was the same for both the first and the second cleavage within its recognition site. In addition, the mechanism of action of the enzyme on supercoiled and linear DNA substrates was similar. Other restriction endonucleases were tested against plasmid pMB9. The *HindIII* restriction endonuclease cleaved DNA in the same manner as the *EcoRI* enzyme. However, in contrast with *EcoRI*, the *SalI* and the *BamHI* restriction endonucleases appeared to cleave both strands of the DNA duplex almost simultaneously. The function of symmetrical DNA sequences and the conformation of the DNA involved in these DNA-protein interactions are discussed in the light of these observations. The fact that the same reactions were observed on both supercoiled and linear DNA substrates implies that these interactions do not involve the unwinding of the duplex before catalysis. (38 refs)

79-4791 Kinetic Complexity of Nuclear Poly(A)-containing RNA in Normal and Regenerating Rat Liver. (Eng) Krieg, L. (Institut für Experimentelle Pathologie, Deutsches Krebsforschungszentrum Heidelberg, Im Neuenheimer Feld 280, D-6900 Heidelberg 1, W. Germany); Alonso, A.; Volm, M. *Eur J Biochem* 96(1): 77-85; 1979.

The effect of hepatectomy on the content and composition of heterogeneous nuclear RNA sequences containing poly(A) segments at the 3' end was determined with the use of molecular hybridization techniques. Poly(A)-containing RNA isolated from the liver nuclei of untreated, partially hepatectomized, or sham-operated male Sprague-Dawley rats was hybridized to complementary DNA (cDNA). In the homologous reactions, two major classes of poly(A)-containing RNA were seen. When compared with normal liver, the base-sequence complexity of the least abundant class was lower in nuclei from livers isolated 3 hr after partial hepatectomy and was higher in those isolated 12 hr after the operation. In the heterologous reactions, hybrid formation was reduced. There was an increase of some abundant poly(A)-containing sequences and a loss or dilution of rare sequences 3 hr after operation. The latter effect was not specific to the regeneration process, as it occurred after laparotomy as well. Twelve hours after partial

hepatectomy, however, about 10% new poly(A)-containing sequences were detected; they corresponded to about 5,000 molecules of 4,500 nucleotides in length that were unique to regenerating nuclei. (18 refs)

79-4792 Incorporation of Polyribonucleotides in Mitochondria Isolated from the Rat Liver. (Fre) Giguere, L. (Institut du cancer de Montreal, Centre hospitalier Notre-Dame, 1560 est, rue Sherbrooke, Montreal, Canada H2L 4M1); Morais, R. *Rev Can Biol* 37(3): 189-200; 1978.

The uptake of polyadenylic acid (poly A), transfer RNA (tRNA), and polyinosinic acid:polycytidylic acid (poly I:C) in liver cell mitochondria isolated from male Wistar rats was studied. The uptake of the polymers decreased rapidly as the pH rose from 6.0 to 6.8 and slowly as the pH rose from 6.8-7.5. The uptake increased linearly with time in the case of poly A and tRNA, and it was considerably higher at 23 C than at 4 C. The uptake increased linearly with concentration over the entire range studied (0-25 $\mu\text{g/ml}$) for poly A and tRNA and in the concentration range 0-2 $\mu\text{g/ml}$ for poly I:C, but incorporation decreased markedly for the latter above 2 $\mu\text{g/ml}$. (36 refs)

79-4793 Defective Regulation of 3-Hydroxy-3-methylglutaryl Coenzyme A Reductase in a Somatic Cell Mutant. (Eng) Sinensky, M. (Eleanor Roosevelt Inst. for Cancer Res., Univ. Colorado Medical Center, Denver, CO 80262); Duwe, G.; Pinkerton, F. *J Biol Chem* 254(11): 4482-4486; 1979.

A somatic CHO-K1 cell mutant, selected to be resistant to the killing effects of 25-hydroxycholesterol in the absence of cholesterol, was shown to be defective in the inhibition of 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase activity by 25-hydroxycholesterol, cholesterol, and lipoproteins and thus able to maintain the enzyme activity found in cells in the absence of exogenous sterol constitutively. The mutant phenotype was shown to be dominant with respect to the wild type. Actinomycin D and cycloheximide prevented the increase of HMG-CoA reductase activity that occurs in the CHO-K1 cell when cholesterol is removed from the medium. Degradation of the enzyme, measured during inhibition of protein synthesis by cycloheximide, occurred at the same rate in the mutant as in the wild type. Kinetic studies indicated that the *K_m* for two substrates, the activation energy, and a break in the Arrhenius plot are the same for HMG-CoA reductase determined in wild type and mutant cells. It is concluded that the mutant is defective in the regulation of synthesis of HMG-CoA reductase. Of the four processes which determine cellular cholesterol levels (biosynthesis, esterification, efflux, and uptake), only biosynthesis was altered, demonstrating that these processes are not coor-

dinately controlled as has been suggested previously. (24 refs)

- 79-4794** Comparative Studies of Tubulin in Extracts of Normal and Transformed Cells. (Eng) Wiche, G. (Inst. Biochemistry, Univ. Vienna, 1090 Vienna, Austria). *Protides Biol Fluid Proc Colloq* 26: 523-526; 1978.

Tubulins extracted from normal and transformed cells were tested for their functional similarities and their stability in crude preparations. Copolymerization experiments with tubulin of normal mouse BALB/c-3T3 fibroblasts and rat glial C-6 tumor cells or simian virus 40 (SV40)-transformed 3T3 fibroblasts showed that the various tubulins copolymerized in an additive fashion and, furthermore, incorporated into the same microtubule polymers. Thus, functionally, the tubulins extracted from normal and transformed cells were very similar. However, differences were observed regarding their stability. The colchicine-binding decay of tubulin was found to be about two times faster in extracts of normal 3T3 fibroblasts or Chinese hamster lung cells than in those of their SV40-transformed counterparts. (7 refs)

- 79-4795** The Role of Contractile and Cytoskeletal Proteins During Wound Healing and Tumor Cell Invasion. (Eng) Gabbiani, G. (Dept. Pathology, Univ. Geneva, Geneva, Switzerland); Chaponnier, C. *Protides Biol Fluid Proc Colloq* 26: 573-578; 1978.

The role of contractile and cytoskeletal proteins during wound healing and tumor cell invasion was investigated using rats and rabbits and human tumors and normal tissues. Open wound debris in rat or rabbit skin consisted of granulation tissue in which myofibroblasts and newly growing epithelial cells fixed anti-actin antibodies (AAA). These cells also fixed anti-smooth muscle myosin antibodies, but they did not differ from normal cells in the degree of fixation of anti-tubulin antibodies (ATA). Compared with normal skin, the healing wounds showed striking differences in epithelial and fibroblastic cells. The epithelial cells showed a more irregular configuration, displaying several microvillous or plaquelike cytoplasmic projections, and they contained well-defined cytoplasmic microfilaments, predominantly at the cell periphery. The spaces between single epithelial cells were wide and irregular, with few desmosomes, and there was an increase in the gap junctions. In the underlying granulation tissue, fibroblastic cells exhibited the characteristic cytoplasmic features of myofibroblasts. Staining for actin and myosin was always increased in skin, oral cavity, laryngeal, and mammary gland tumor cells, but there was no change in

staining for ATA. Basal cell carcinomas stained less brightly than squamous cell carcinomas, particularly with AAA sera. The tumor cells contained prominent microfilaments, especially the cells situated at the advancing edge of the neoplasms. (20 refs)

- 79-4796** Production of Factors Required for Cell Attachment and Spreading is a Constitutive Property in Mouse A9 Cells. (Eng) Dairkee, S. H. (Dept. Molecular Biology, Univ. California, Berkeley, CA 94720); Gilbert, M. W. *J Cell Physiol* 99(3): 319-326; 1979.

The release of spreading promoting factors (SPF) into the medium by the A9 line of mouse L cells was studied. A9 cells proliferated equally well on bacteriological (BD) and tissue culture dishes (TCD). At a density of $10^4/\text{cm}^2$, the cells did not attach to the BD substrate, but after a few cell doublings, many cells appeared to spread out on the substrate. Conditioned medium (CM) from such cells promoted the spreading of fresh cells on both BD and TCD surfaces. The SPF in the CM was nondialyzable and dilution reduced its ability to promote cell spreading. The production of SPF was cell density-dependent, cells at a higher density conditioning their medium more and, therefore, promoting spreading to a greater extent. SPF produced by monolayer and suspension cultures appeared to be identical. CHO cells, which do not attach to or spread on BD, also produced SPF, but they were unable to respond effectively to their own CM or to A9 CM. A9 cells, however, displayed the same degree of cell spreading in A9 CM and CHO CM. The A9 CM also caused the CHO cells to clump extensively in suspension. (17 refs)

- 79-4797** Structural Transitions and Functional Activity of Microsomes in Normal and Tumor-bearing Rats. (Rus) Kotrikadze, N. G. (State Univ., Tbilisi, USSR); Gabuniia, G. D.; Tsartsidze, M. A.; Lomsadze, B. A. *Soobshch Akad Nauk Gruz SSR* 92(3): 701-704; 1978.

Paramagnetic probes were used to evaluate the structural transitions and activity of the marker enzyme glucose-6-phosphatase in microsomes isolated from the liver of normal rats and rats with Walker carcinoma. Analysis of the rotation correlation of probes I and II in the microsomes from normal rats showed that structural transitions were recorded at 28-40-60 C for probe I and at 20-30 and 50-58 C for probe II. In microsomes isolated from nontumorous tissue of tumor-bearing rats, the structural transitions were recorded at 40-64 C for probe I and at 38-48 C for probe II. Correspondingly, structural transitions in microsomes isolated from the tumor tissue were recorded at 60-70 C and 10-20 or 40-60 C. The decrease in the rotation correlation time in the tumor tissue microsomes was associated with a decrease in marker enzyme activity. (11 refs)

79-4798 Cell Surface Charges and Invasion of Drug Resistant Sublines of Ehrlich Ascites Tumor Cells. (Eng) Gu, G. (Inst. Experimental Biology, Shanghai, China); Liu, L.; Li, M.; Hong, L. *Chin Med J [Engl]* 92(4): 247-252; 1979.

Ehrlich ascites tumor cells developed into drug-resistant (DR) sublines after several months of exposure to vinblastine (VB), actinomycin D, or 5-fluorouracil (FU), but they regained sensitivity after treatment with these antitumor agents was stopped for 4 mo or more. The electrophoretic mobilities of the three DR sublines were different from that of the original line. The cell-surface charge densities of cells that had regained drug sensitivity following VB or FU treatment were different from those of the respective DR sublines. (15 refs)

79-4799 The Proton Stoichiometry of Electron Transport in Ehrlich Ascites Tumor Mitochondria. (Eng) Villalobo, A. (Dept. Physiological Chemistry, Johns Hopkins Univ., Sch. Medicine, Baltimore, MD 21205); Lehninger, A. L. *J Biol Chem* 254(11): 4352-4358; 1979.

Initial rate measurements of the stoichiometric relationships between H^+ ejection, K^+ and Ca^{2+} uptake, and electron transport were carried out on mitochondria from Ehrlich ascites tumor cells grown in mice. With succinate as substrate and *N*-ethylmaleimide to prevent interfering H^+ reuptake via the phosphate carrier, close to 8 H^+ were ejected per oxygen atom reduced (H^+ / O ejection ratio = 8.0); with the NAD-linked substrates pyruvate or pyruvate + malate, the H^+ / O ejection ratio was close to 12. The average H^+ / site ratio (H^+ ejected/ $2e^-$ /energy-conserving site) was thus close to 4. The simultaneous uptake of charge-compensating cations, either K^+ (in the presence of valinomycin) or Ca^{2+} , was also measured, yielding average K^+ / site uptake ratios of very close to 4 and Ca^{2+} / site ratios close to 2. It was also demonstrated that each calcium ion enters the respiring tumor mitochondria carrying two positive electric charges. These stoichiometric data observed in mitochondria from Ehrlich ascites tumor cells thus are in complete agreement with similar data on normal rat liver and rat heart mitochondria and suggest that the H^+ / site ratio of mitochondrial electron transport

may be 4 generally. It was also observed that the rate of ΔH^+ back-decay in anaerobic tumor mitochondria following oxygen pulses is some 6- to 8-fold greater than in rat liver mitochondria tested at equal amounts of mitochondrial protein. (44 refs)

79-4800 The Acylation of *sn*-Glycerol 3-Phosphate in Mammalian Organs and Ehrlich Ascites Tumor Cells. (Eng) Haldar, D. (Dept. Biological Sciences, St. John's Univ., Jamaica, NY 11439); Tso, W. W.; Pullman, M. E. *J Biol Chem* 254(11): 4502-4509; 1979.

The properties of acyl-CoA:*sn*-glycerol-3-phosphate acyltransferase in mitochondrial and microsomal fractions from liver and other organs of rat, mouse, guinea pig, rabbit, and beef are described. In liver of all species, the specific activity of the mitochondrial and microsomal enzyme was similar, whereas in other organs the microsomal enzyme was at least 10 times more active. In all types of mitochondria the enzyme showed a strong preference for palmityl-CoA, was insensitive to *N*-ethylmaleimide, and produced monoacylglycerophosphate as a major product. Microsomal preparations, by contrast, catalyzed the acylation of glycerophosphate with both palmityl-CoA and oleyl-CoA, were almost completely inhibited by *N*-ethylmaleimide, and produced diacylglycerophosphate as the chief reaction product. These criteria, as well as striking differences in response to acetone, were also used to characterize mitochondrial- and microsomal-associated activities in Ehrlich ascites tumor cells. The properties of the enzyme in the mitochondrial fraction of Ehrlich cells resembled those of its microsomal counterpart. Subcellular distribution studies using marker enzymes indicated that glycerophosphate acyltransferase activity in these cells is confined to the microsomes. Taken together, the results raise the possibility that the absence of the acyl-CoA-specific mitochondrial enzyme may have a bearing on the unusual positioning of saturated fatty acids found in certain phospholipids in these tumor cells. Additional experiments indicated that, regardless of whether palmityl-CoA or oleyl-CoA is used as acyl donor, 1-acyl-*sn*-glycerol 3-phosphate is the major if not sole product of *sn*-glycerol 3-phosphate acylation in rat liver mitochondria and microsomes as well as in Ehrlich tumor cell microsomes. (47 refs)

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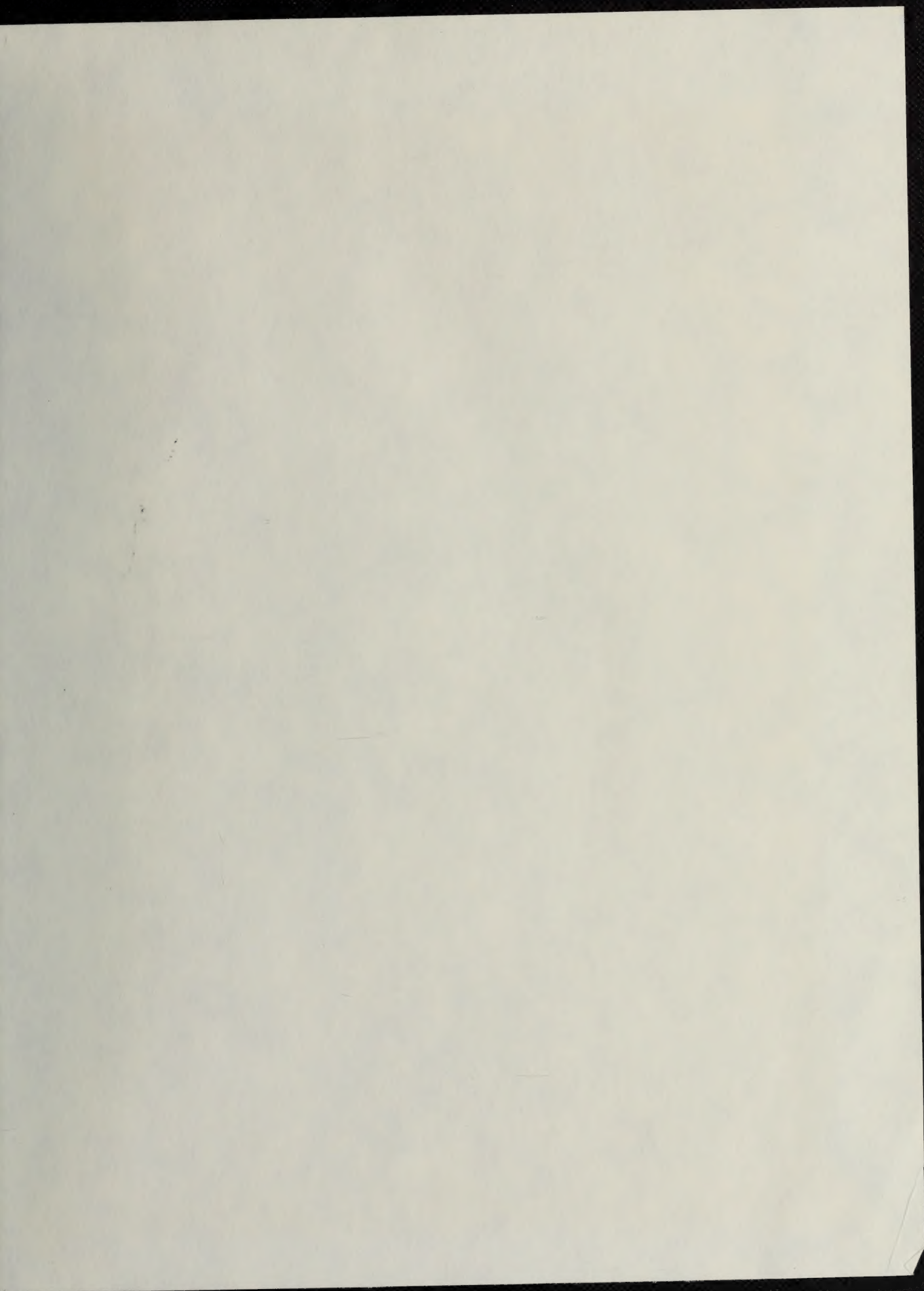


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